982/1

NEWARK PUBLIC LIBRARY

MAR 9 1982

U. S. GOVT. PUBS.
DEPOSITORY 376

JANUARY-FEBRUARY 1982

Abstracts 82-0001-82-0296

Smoking and Health Bulletin

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

HE 20.7211:

INTRODUCTION

All items appearing in the *Smoking and Health Bulletin* will become part of the Technical Information Center (TIC) of the Office on Smoking and Health and can be referred to by their accession numbers. At the end of each year a cumulated index will be published; therefore the user is advised to retain the individual issues as well as the cumulative index. The *Bulletin* is issued at irregular intervals as the amount of new material warrants. The first and last abstract numbers and the cutoff date are shown on each issue to permit consecutive filing.

The Office on Smoking and Health continues to offer retrieval of information on smoking through the Automated Search and Retrieval System. This search system, announced in the June-July 1976 issue of the *Bulletin,* permits researchers and others to obtain bibliographic information on smoking and health in print-out form. A Search Request form, which can be submitted to the Office, will be found in each issue of the *Bulletin.* The data base, totalling over 20,000 items, contains all literature collected, processed, and published in the *Bulletin* from 1970 to the present.

Bibliographic and reference services continue to be offered and can be obtained by writing to or calling the Office on Smoking and Health. Because of service staff limitations, copies of reference items can be provided in single copies only, and only in those cases where the material cannot be obtained through more immediate sources such as local public libraries, or school and university libraries. Visitors may use the collection during office hours: 8:30 a.m.–5:00 p.m. weekdays. Advance arrangements of any planned visits are suggested.

We welcome suggestions or comments from our readership regarding our publications and services.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Phone: 301-443-1690

ISSN: 0081-0363
January-February
Abstracts 82-0001-82-0296

Smoking and Health Bulletin

CONTENTS

AGRICULTURE 1
CHEMISTRY 4
PHARMACOLOGY AND TOXICOLOGY 8
MORTALITY AND MORBIDITY 15
NEOPLASTIC DISEASES 16
NON-NEOPLASTIC RESPIRATORY DISEASES 21
CARDIOVASCULAR DISEASES 27
PREGNANCY AND INFANT HEALTH 33
OTHER DISEASES AND CONDITIONS 36
BEHAVIORAL AND PSYCHOLOGICAL ASPECTS 38
SMOKING PREVENTION AND INTERVENTION 46
SMOKING CESSATION METHODS 51
TOBACCO PRODUCT ADDITIVES 54
TOBACCO MANUFACTURING AND PROCESSING 55
TOBACCO ECONOMICS 58
LEGISLATION 70
GENERAL 72
AUTHOR AND ORGANIZATIONAL INDEX i
SUBJECT INDEX vi

AGRICULTURE

82-0001. Barford, M. **A View From Overseas: Securing Low Tar in the Field.** *Flue Cured Tobacco Farmer* 18(3): 24, 26, 1981.

Domestic tobacco farmers will probably still be growing flue-cured Virginia tobacco 10 to 20 years from now, since such tobacco is essential to the cigarette blends of the United States, and to many blends elsewhere in the world. Flue curing is likely to continue despite its higher cost; and it is suggested that farms adopt various energy-saving methods, such as insulation of kilns, better organization of priming and tobacco handling, and air or sun curing. Research into leaf modification to breed tobacco with a good nicotine content but low in tar is recommended. Nicotine gives smoking a good deal of its satisfaction, while tar is suspected of posing the chief hazard to health from smoking. The nicotine-rich "Rustica" type of tobacco, grown in such areas as India and the Soviet Union, may be useful in future plant breeding efforts. Since some of the tar ingredients help give cigarettes their taste and aroma, more research will be needed to learn how to retain such ingredients in future tobacco varieties while reducing those constituents implicated in the smoking and health controversy. Manufacturers might prefer to use such low-tar tobacco for their products, rather than rely on expensive and elaborate premanufacturing treatments, filters, or porous paper to reduce yields.

82-0002. Collins, G. B.; Legg, P. D. **Genetic Manipulation of Tobacco Using in Vitro Methods.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 1-14.

The development of efficient in vitro methods for culturing tobacco cells has enabled geneticists and breeders to use cell and tissue culture techniques in experiments designed to genetically alter tobacco, improve quality, and reduce health risks associated with tobacco components. Anther and pollen culture methods have been used to produce haploid and double-haploid genetic lines which offer major advantages by reducing the time required to produce homozygous lines and in facilitating genetic analysis. Variant selection at the cellular level has produced a number of potentially useful tobacco mutants. Selection of recessive mutants is made feasible through the availability of haploid cell cultures. Aneuploids, including previously unattainable nullisomics, have been generated by culturing anthers of monosomic tobacco plants. The culture of immature hybrid embryos has made it possible to obtain otherwise unobtainable interspecific hybrids between tobacco and wild *Nicotiana* species. Resistance to diseases such as wildfire, black root rot, mosaic virus, and black shank are genetic factors already transferred into cultivated tobacco. Other traits which need to be transferred include specific biochemical constituent levels, and resistance to other diseases and insects. Before development and release of new tobacco cultivars, their constituents (total and individual alkaloids, total nitrogen, nitrate nitrogen, protein nitrogen, and total volatile nitrogenous bases) are compared with those of acceptable, currently grown cultivars. (Auth. Abs. Mod.)

82-0003. Collins, W. K. **MH Label Changes Require Careful Use of Sucker-Control Chemicals. But You Can Still Get Acceptable Control.** *Flue Cured Tobacco Farmer* 18(3): 20, 1981.

Effective with the 1981 tobacco crop, all labels on sucker-control products containing maleic hydrazide (MH) will prohibit more than one application unless a washoff occurs 6 to 12 hours after application. Also, 7 days must pass between application and harvest. In the past, as a result of multiple applications of MH, residues on cured leaf often reached levels unacceptable to certain important foreign buyers. The following seven-step program of sucker control is suggested for harvesting cured leaf without excessive MH: (1) apply a maximum of 80 pounds of nitrogen/acre, to avoid excess sucker growth; (2) apply a contact-type sucker control chemical when about half of the plants reach button stage; (3) top plants promptly; (4) apply the contact solution again 3 to 4 days after first application; (5) top remaining plants; (6) apply an MH product about 7 days after the last contact; and (7) allow at least 7 days after application before harvest. Since weak contact solutions only kill one of the two suckers in each leaf axil, encouraging the misuse of MH, application techniques used for contact solutions need to be improved. Care should be taken to avoid spraying the field too fast. Also, the longer the period between application of MH and harvest, the better the opportunity for MH residues to wash off the leaves. Sucker control will not be affected, since MH residues play no role in sucker control.

82-0004. Davis, D. L.; Atkinson, W. O.; Dorough, W. **Residues of Maleic Hydrazide in Burley Tobacco.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 124-140.

Growing concern over the health effects of the residues of maleic hydrazide (MH), a systemic compound used to control axillary buds on tobacco, was the basis for this research project to determine: (1) the residues on tobacco produced in Kentucky; and (2) a practical means of reducing MH residues while maintaining satisfactory sucker control. The establishment of 80 ppm as the accepted tolerance level had led to the issuance of a Rebuttable Presumption Against Registration by the Environmental Protection Agency, a process still in progress. Representative market samples from six areas were collected and compared, which showed higher MH residues in western Kentucky and higher averages for upper leaves in all areas. The rather large differences seen within areas were attributed to different application rates and/or environmental differences. Application of MH near harvest date increased residues, while allowing an additional 10 days between application and harvest reduced them; split applications at the same total rate tended to increase residues. Composite samples from the auction markets of 1975, 1976, and 1979 produced residues of 67, 71, and 88 μg/g, respectively. The 10-year average for 1963-1972 was 57 μg/g of tobacco leaf. The transfer of MH from the tobacco to its condensate was shown to vary from 0.6 to 2.3 μg/g of condensate, a transfer equal to 2 to 3 percent.

82-0005. Legg, P. D.; Collins, G. B. **Influence of Genetic Factors on Tobacco Leaf and Smoke Chemistry.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 15-31.

The controversy over smoking and health has stimulated research into ways of changing the genetic character of tobacco plants, in hopes of producing safer chemical constituents. The nature and levels of the constituents have been measured in a wide range of tobacco types, introductions, and *Nicotiana* species. Chemical characteristics appear to be under the same type of genetic control as are morphology and agronomic characters, although useful information on such genetic control is limited. The most extensive genetic information on chemical constituents has been collected for alkaloid quality and quantity. Care must be taken not to confuse traits due to other factors, such as adaptation, disease responses, and growth features, for genetic differences. The first step in using genetic alteration to change a chemical trait is to identify a source of germ plasm with the desired genetic factors. The task is simplified if the donor plants used as the germ plasm source are closely related to the commercial cultivars to be altered. The donors are often different from the cultivars in such traits as plant height, maturity, and photoperiodic response. These agronomic traits, rather than different genetic factors for chemical constituents, may sometimes account for phenotypic differences in chemical composition. Most Burley tobacco cultivars presently used are resistant to diseases such as tobacco mosaic and wildfire. Resistance factors can be transferred to susceptible Burley cultivars by the backcross method. The incorporation of resistance factors does not appear to significantly alter cultivar nitrogenous constituents and tar content. The use of disease-resistant cultivars helps to prevent farm production losses and to prevent significant changes in leaf chemistry from diseases. The chemical composition of tobacco leaves is also affected by environmental conditions during the growing season; by cultural practices such as land preparation, fertilization, topping time and procedures, maturity at harvest, and application of chemicals; and by air, flue, or homogenized leaf curing.

82-0006. Pandeya, R. S.; White, F. H. **Delgold—a New Flue-Cured Tobacco Variety.** *Canadian Tobacco Grower* 29(2): 78-80, February 1981.

Delgold, a new flue-cured tobacco variety released in 1980 (first commercial crop year, 1981), is described. Delgold, previously known to growers as Strain 76N2-E, produces faster growing, lighter seedlings than Virginia 115, and one to two more leaves than other varieties. The overall leaf dimensions also are larger than for Virginia 115, with increased tolerance to black root rot and weather fleck diseases compared to Virginia 115 and Nordel. It has not exhibited any tendency for brittleness, leaf drop, or problem ground sucker growth. In the past 2 years, Delgold has produced 300 to 400 kg/ha more of cured leaf yield of high quality, compared to Virginia 115 and Nordel. Delgold has produced about 10 percent higher cured leaf with 17 percent higher total leaf alkaloids than Virginia 115. The nicotine content is higher than Virginia 115 (3.38 percent vs. 2.28 percent), while its tar—nicotine ratio is lower.

82-0007. Sims, J. L. **Nitrogenous Compounds in Burley Tobacco as Affected by Certain Culture Practices.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 32-43.

Field experiments, conducted on Maury silt loam soil to determine the effect of nitrogen (N) fertilization and Burley tobacco genotype on selected leaf and smoke constituents, are described. Two commercial cultivars (Ky 14 and Burley 21) and three Burley breeding lines (low-alkaloid Burley 21, low-intermediate-alkaloid Burley 21, and high-intermediate-alkaloid Burley 21) were used. Additional experiments were conducted to determine the effect of varying levels of potassium (K) and magnesium (Mg) fertilization on the yield and chemical composition of Burley tobacco leaves and smoke. Leaf yields and concentrations of nitrate N, total N, and total volatile nitrogenous bases (TVNB)-N were directly related to the theoretical alkaloid levels for B21 and the low-alkaloid genotypes. In contrast, concentrations of protein N were related inversely to theoretical alkaloid concentrations. A significant N rate X genotype interaction existed for total alkaloids indicating alkaloid concentration was affected less by N fertilization of the low-alkaloid than commercial genotypes. Generally, adding N fertilizer at the rate of 200-lb N/acre produced small increases in average levels of total particulate matter (TPM), nicotine, tar (TPM-nicotine-water), and NO in cigarette smoke over amounts obtained from the 100-lb rate. Further additions of N to either 300- or 400-lb N/acre greatly increased values for NO, decreased TPM and tar, and had little effect on nicotine. The data suggest that the decreases in TPM and tar at the high rates of N fertilizer may be attributed to high levels of leaf nitrate. Potassium fertilization reduced protein-N, TVNB, amino-N, Ca, and Mg contents of leaf by 12 percent, 10 percent, 25 percent, 23 percent, and 32 percent, respectively, compared to the control, when applied at the rate of 400-lb K/acre. Additionally, the same rate of K decreased total particulate matter, nicotine, and cyanide delivered from all Burley cigarettes by 24 percent, 7 percent, and 50 percent, respectively, compared to the control. Aldehyde content of cigarette smoke was increased by K application while Mg fertilization had little effect on any leaf or smoke constituent measured. (Auth. Abs. Mod.)

82–0008. Trevathan, L. E.; Moore, L. D. **A Study of Calcium Fertilization on Weather Fleck and Chemical Composition of Flue-Cured Tobacco.** *Tobacco International* 183(19): 52-55, September 18, 1981.

The effects of calcium fertilization on the reaction of *Nicotiana tabacum* L. "North Carolina 88" to ambient levels of air pollution, primarily ozone, and the relationships between the calcium content and the nicotine, reducing sugar, and nitrogen content of leaves were investigated. Calcium ($CaSO_4$) was applied at 0, 112, 225, and 562 kg/ha. Leaves were harvested at 5, 6, 7, and 9 weeks after transplanting and were analyzed for calcium, nicotine, reducing sugars, and nitrogen. The amount of calcium applied was not related to the calcium or nicotine content of leaves. There was a direct correlation between the calcium content of leaves, leaf position, and weather fleck severity. The calcium content of leaves was negatively correlated with the nitrogen content. Lower leaves had the highest concentrations of calcium, the greatest amount of weather fleck, and the lowest concentrations of nitrogen. It appears that the manipulation of calcium fertilization under field conditions to increase the tolerance of flue-cured tobacco to weather fleck is of little practical value. It is concluded that the direct nutritional effects of applied nitrogen and phosphorus fertilization are the principal cultural factors which may be used to increase tobacco tolerance to oxidants. (Auth. Abs. Mod.)

82–0009. Wilkinson, R. E.; Kasperbauer, M. J.; Young, C. T. **Free Amino Acid Content of Burley Tobacco Leaves Developed Under Different Light and Temperature Conditions.** *Journal of Agricultural and Food Chemistry* 29(3): 658-660, May-June 1981.

Free amino acid contents of Burley tobacco (*Nicotiana tabacum* L.) leaves differed among genetically uniform plants that were grown under different temperature and light environments. The amino acids were listed in metabolically related groups. Those derived from oxalacetate, α-ketoglutarate, and D-3-phosphoglycerate accumulated in higher concentrations in leaves that developed under short, relative to long, photoperiods. There was a photoperiod by temperature interaction. When end-of-day red or far-red followed 8- or 16-hour photosynthetic periods, the highest concentrations of free amino acids accumulated in leaves of plants that received red light at the end of daily 8-hour photosynthetic periods. Far-red light following 8-hour photosynthetic periods resulted in lower free amino acid concentrations. The responses to brief irradiations with red or far-red light are interpreted to indicate that the phytochrome system was involved, contributing to differences in free amino acid concentrations. Thus, leaf free amino acid contents were influenced by the relative synthesis and depletion activities that existed under each particular environment. (Auth. Abs.)

82–0010. Woodlief, W. G.; Chaplin, J. F.; Campbell, C. R.; De Jong, D. W. **Effect of Variety and Harvest Treatments on Protein Yield of Close-Grown Tobacco.** *Tobacco International* 183(16): 40-43, August 7, 1981.

Extractable protein yield of NC PY-10, LAFC 53, Speight G-28, and SC 58 tobacco (*Nicotiana tabacum* L.), planted as "close-grown," was compared by utilizing four harvest treatments: (1) harvest during the button stage, (2) harvest of ratoon regrowth following cutting and discarding of the mature tobacco, (3) harvest of mature tobacco, and (4) harvest of three successive midgrowth crops by utilizing ratooning. During tobacco leaf growth and elongation, protein synthesis is greater than protein degradation, but when the leaf matures and senescence begins, protein degradation becomes greater than protein synthesis. Not all leaves on a tobacco plant reach maximum elongation or enter senescence simultaneously. It was necessary to consider the stage of plant growth at harvest. Results showed no significant differences among the tobacco cultivars tested. Treatment 4, with three successive midgrowth harvests, produced a significantly greater amount of extractable protein per hectare than the other harvest treatments tested, due to the large amount of fresh weight harvested and its higher protein content. Seasonal conditions and senescence appear to be crucial in affecting the amount of extractable protein produced. (Auth. Abs. Mod.)

82–0011. Yocum, J. O. **Guide for the Production of Pennsylvania Tobacco, Special Circular 264.** Pennsylvania State University, Cooperative Extension Service, University Park, Pennsylvania, July 1981, 12 pp.

Information is provided on the production practices developed from experience and research in growing tobacco in Pennsylvania. The facets of tobacco farming discussed are: (1) the tobacco varieties suitable to Pennsylvania, their planting requirements, and disease resistance characteristics; (2) seedbeds preventive measures, transplanting requirements, field culture, and the accompanying disease possibilities; and (3) tobacco curing, stripping, grading, and baling. Also fertilization, germination, and pesticide recommendations and data are given where appropriate.

See also, **82–0058**

CHEMISTRY

82–0012. Andersen, R. A.; Kasperbauer, M. J.; Vaughn, T. H.; Burton, H. R. **Effects of Plant Growth and Air Curing on Amounts of *trans*-Coniferyl and *trans*-Sinapyl Alcohols in Midveins of Burley Tobacco.** *Journal of Agricultural and Food Chemistry* 29(4): 841-845, July-August 1981.

A quantitative gas chromatographic method was developed for the estimation of *trans*-coniferyl and *trans*-sinapyl alcohols in leaf midveins of field-grown Kentucky 14 Burley tobacco. It was confirmed by gas chromatography—mass spectrometry that both alcohols were major phenylpropanoids in hot-water extracts of the leaf midvein samples. Concentrations of both alcohols increased as the maturity of plants increased. Greater quantities of alcohols were obtained after air curing than at harvest. Leaf position on the stalk also affected the amounts of the alcohols recovered, but the magnitude and direction of this effect depended on plant maturity and air curing parameters. Levels of *trans*-sinapyl alcohol among all the samples were only about one-third those of *trans*-coniferyl alcohol; the amounts found ranged from 7.3 to 70.5 mg of coniferyl alcohol and 0 to 24.6 mg of sinapyl alcohol per kg dry weight of sample. (Auth. Abs.)

82–0013. Bialer, M.; Sloneker, S. D.; Kostenbauder, H. B **Identification of an Inhibitor of Pulmonary Biotransformations in Tobacco Smoke Condensate.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 158-172.

In an effort to identify the substance in the particulate phase of tobacco smoke which inhibits the pulmonary metabolism of nicotine, research was conducted to isolate and define some of the characteristics of the substance(s) responsible (inhibitor). The inhibition of benzo(*a*)pyrene, the only single pure compound present in the system, was analyzed using an isolated perfused rabbit lung model. Based on study data, the following inhibitor characteristics were established: (1) soluble in ether and partially soluble in pentane and distilled water; (2) extractable from a cigarette smoke condensate pentane mixture with a solution of NaOH 1 N or HC1 1 N; (3) not extractable out of the same mixture with a solution of NaHCO, five percent; (4) optimal pH for extracting the inhibitor to ether from an aqueous medium was at a neutral pH of 7.4; (5) stable to acidic and basic pHs; (6) inhibitor activity was retained even after the active fraction was stored in the refrigerator for several weeks; and (7) extraction out of an aqueous medium with ether or basic pH did not reduce its inhibition significantly. After experimentation, it was concluded that the acidic moiety in the inhibitor molecule is a phenolic group, containing acidic (phenolic) and basic functions in its molecule.

82–0014. Chamberlain, W. J.; Snook, M. E.; Chortyk, O. T.; Baker, J. L. **Characterization of an Aryl Nitrile Fraction of Cigarette Smoke Condensate.** *Tobacco International* 183(4): 77-79, February 20, 1981.

Aryl nitriles, naphthoquinones, and fluorenones were isolated from a neutral subfraction of cigarette smoke condensate (CSC) and characterized by a combination of gel filtration chromatography and gas chromatography. Gel filtration chromatography of a benzene-eluted subfraction of CSC neutrals on Bio-Beads SX-12 produced two fractions containing alkyl phenols, aryl nitriles, quinones, and fluorenones. Subsequent separation of these gel fractions on Sephadex LH-20 removed the alkyl phenols and permitted the identification of benzonitrile and its methyl, dimethyl, and trimethyl derivatives, cinnamonitrile, α- and β-cyanonaphthalene, two methylcyanonaphthalenes, the monomethyl, dimethyl, and trimethyl naphthoquinones, fluorenone, and both methylfluorenones and dimethyl fluorenones. (Auth. Abs.)

82–0015. Davis, D. L.; Song, B. H.; Everette, G. **Culture and Curing Effects on Dark Tobacco Chemistry.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 44-56.

Dark tobacco is used chiefly in the manufacture of snuff, chewing tobacco, cigars, and heavy smoking tobacco. Data on its chemical composition have been relatively limited. To help remedy this, a large number of dark fire-cured tobacco samples collected from Western Kentucky over a 3-year period were analyzed for several chemical constituents with emphasis on the nitrogenous compounds. Total alkaloids averaged 3.80, 3.66, and 4.85 percent for each of the 3 years. Upper stalk tobacco had higher total alkaloid values, total leaf nitrogen, α-amino nitrogen, total volatile nitrogenous bases, and petroleum ether extractables. Nitrate nitrogen showed more variation than did total alkaloids or total nitrogen during the 2 years for which this analysis was completed. The average nitrate N was 0.34 and 0.73 percent for each of the 2 years. Calcium levels were greatest in the lower leaves with the opposite being found for phosphorus. Potassium appeared to be higher in the middle stalk tobacco. The ash percentage was higher in the lower leaves while alkalinity values were higher for the middle stalk leaves. Hydrocarbon waxes ranged from C_{26} to C_{35} with C_{31} being the predominate wax. Anteiso, normal, and iso paraffins were found in the cuticle wax with the latter two being most abundant. In a study of the effects of nitrogen fertilization on dark tobacco chemistry, it was found that increased rates of nitrogen increased calcium to a point. However, phosphorus or potassium were not influenced by rates of nitrogen. Increased nitrogen rates tended to increase the levels of nitrogenous constituents in the dark tobacco leaf. There did not appear to be an effect of nitrogen fertilization on petroleum ether extractables. When the same varieties were air-cured and fire-cured, total alkaloids and nitrate N appeared to be higher in the dark air-cured, whereas total N was slightly higher in the dark fire-cured. (Auth. Abs. Mod.)

82-0016. Djordjevic, M. **Uticaj Perforacije Cigaretnog Papira na Kvalitativan Sastav Kondenzata Duvanskog Dima. [Effect of Cigarette Paper Perforation on Qualitative Composition of Tobacco Smoke Condensate.]** *Arhiv za Poljoprivredne Nauke* 41(143): 413-423, 1980, Serbo-Croatian.

Smoke condensates from machine smoking of 20 domestic cigarettes wrapped in perforated or unperforated paper were collected on a Cambridge filter and analyzed. Simultaneous smoking of five cigarettes was done on a Filtron 302 machine under standard conditions. The smoke condensates from cigarettes wrapped in unperforated paper (condensate A) and in perforated paper (condensate B) contained 0.98 and 0.62 mg nicotine/cigarette, respectively. Dilution of smoke with air resulted in a decrease of the total acidic, basic, and neutral fractions and an increase in the phenolic fraction contents in the smoke condensate. Gas chromatographic analysis of the ether-soluble acidic fractions of the smoke condensates revealed that condensate A contained more of saturated n-monocarboxylic acids of higher molecular weight (mostly n-C_{20}) than the condensate B (mostly n-C_{12} and C_{16}). Composition of the acidic fraction obtained by alkaline hydrolysis of the neutral fraction of smoke condensate was found to be essentially the same in A and B condensates. Hence, dilution of smoke with air did not affect transfer of the neutral fraction into the mainstream smoke.

82-0017. Djordjevic, M. **Uporedno Ispitivanje Zastupljenosti Nikotina u Duvanu i Kondenzatu Dima Cigareta Razlicitih Kvalitetnih grupa. [Parallel Investigations of Nicotine Content in Tobacco and Smoke Condensate of Cigarettes of Different Quality.]** *Arhiv za Poljoprivredne Nauke* 41(143): 409-412, 1980, Serbo-Croatian.

Yugoslav-made cigarettes of four quality types were analyzed for nicotine percentage in tobacco blends, smoke condensate amount from one cigarette, and nicotine content in the condensate from one cigarette. Nicotine content of tobacco blends, determined spectrophotometrically in the distillate from alkaline tobacco suspension, varied insignificantly from one quality type to another. Smoke condensate was obtained by simultaneously smoking eight cigarettes on a Filtron 302 model of the Borgvaldt smoking machine under standard conditions. The amount of smoke condensate determined by the Cambridge-filter method increased from 15.17 to 21.91 mg/cigarette in parallel with the cigarette quality. Also, nicotine content in the condensate increased from 0.53 in the lowest quality cigarettes to 0.98 mg/cigarette in cigarettes of the highest quality. The nicotine percentage in the smoke condensate from the first quality cigarette varied with the moisture content of the cigarette. The highest nicotine content (5.15 percent) was found in the condensate from a cigarette with 13.9 percent moisture. Also, cigarettes with 13-14 percent moisture yielded the lowest amount of condensate per cigarette. The higher the nicotine content in the smoke, the lower the content of the allegedly carcinogenic polycyclic aromatic hydrocarbons.

82-0018. Fu, M. C.; Lillich, T. T. **Oxygen Uptake by Paracoccus Denitrificans as a Bioassay of Tobacco Smoke Condensate Toxicity.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 303-320.

Modification of tobacco smoke toxicity, either through genetic or environmental manipulation of the tobacco, can potentially reduce the correlation of smoking with cancer, and with pulmonary and heart diseases. Since success depends on determining the toxicity of samples from large numbers of tobaccos, a rapid reproducible short-term bioassay was developed, based on the effect of test samples on oxygen (O_2) uptake by *Paracoccus denitrificans* vessels using either succinate or reduced nicotinamide adenine dinucleotide (NADH) as electron donors. The oxygen uptake in this bacterial species is mediated by an electron transport system (ETS) that has many characteristics similar to the ETS of mammalian mitochondria. Toxicities were calculated from the amount of test sample necessary to obtain a 50 percent inhibition of O_2 uptake relative to control vesicles. Using this bioassay, it was possible to rank order different tobaccos according to the relative toxicities of their smoke condensate and condensate fractions. The bioassay also made it possible to identify fractions, derived from smoke condensates, which are known to contain tumor initiators, promotors, or both. The relative toxicity for the five different tobaccos tested were (in decreasing order): in whole cigarettes smoke condensate—Maryland, 2R1, Burley, Flue Cured, and Turkish; in water insoluble cigarette smoke condensate—Burley, 2R1, Turkish, Maryland, and Flue Cured. (Auth. Abs. Mod.)

82-0019. Gairola, C. **Short-Term Bioassays for the Evaluation of Cigarette Smoke Condensates.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 284-302.

In an ongoing research program to produce safer tobacco, three short-term bioassays were developed for the routine evaluation of cigarette smoke condensates, since traditional methods of chemical analysis can take as long as 2 years to complete. Of the three bioassays, only the Ames' Test (gene mutation in a prokaryote) was reasonably quantitative for routine comparison of condensate mutagenesis. Potency estimates for 500 experimental tobaccos were obtained by using a standardized protocol for testing smoke condensates from University of Kentucky Reference Cigarettes. The yeast D_7 test was not sensitive enough to give an adequate dose—response, although use of already initiated D_7 cells improved the response slightly. Measurement of DNA synthesis on mouseback epidermis provided a qualitative test but could not be used for evaluation of quantitative differences among condensates. Further investigations are being conducted to select suitable quantitative bioassays for routine evaluation of cigarette smoke and its condensates. (Auth. Abs. Mod.)

82–0020. Herzfeld, L. **Zigaretten: Warum Schadstoffindex? (Letter). [Cigarettes: Why Is a Noxious Substance Index Needed? (Letter).]** *Munchener Medizinische Wochenschrift* 123(23): 939-940, June 5, 1981, German.

Both the gas and particulate phase of cigarette smoke are inhaled simultaneously; hence both must be taken into consideration when educating smokers to minimize their health risks. An index of noxious substances would make it possible to adhere to this preventive medical goal by taking into account effects of the whole cigarette. The values of 30 mg, 2 mg, 6 percent, and 1,500 ppm for tar, nicotine, carbon monoxide (CO), and nitrogen oxides (NO_x), respectively, are recommended as the maximum values for developing a less harmful cigarette. Tar and nicotine contents and the index value should be included on cigarette labels; the index indicating CO and NO contents in the gas phase.

82–0021. Kuwata, K.; Uebori, M.; Yamazaki, Y. **Determination of Phenol in Polluted Air as *p*-Nitrobenzeneazophenol Derivative by Reversed Phase High Performance Liquid Chromatography.** *Analytical Chemistry* 52(6): 857-860, 1980.

A convenient analytical technique to determine traces of phenol in polluted air, such as urban air, industrial emissions, auto exhaust, and tobacco smoke, is described. The technique had to meet the following requirements: (1) sample analysis should be repeatable within a reasonable time; (2) phenol should be separated from other phenolic materials; (3) phenol vapor should be accurately detected at the ppb level; and (4) the method should be applicable in an aqueous medium in which phenol vapor is easily trapped. Phenol was collected by using a fritted bubbler with 10 mL of 0.06 percent sodium hydroxide solution and determined by reversed phase high performance liquid chromatography. The *p*-nitrobenzeneazo derivative of phenol could be thoroughly separated from other phenolic members. The detection limit of phenol was 0.05 ppb for 150 L of a gas sample. Phenol vapor in the low ppm or ppb levels could be determined with less than 3 percent relative standard deviation. It is suggested that the analytical method by liquid chromatography might be extended to phenolic compounds other than phenol in polluted airs if suitable conditions for the derivatization and identification of each member are examined.

82–0022. Matsumoto, T.; Yoshida, D.; Tomita, H. **Determination of Mutagens, Amino-α-Carbolines in Grilled Foods and Cigarette Smoke Condensate.** *Cancer Letters* 12(1-2): 105-110, March 1981.

The amounts of two mutagens, 2-amino-9*H*-pyrido(2,3-*b*)indole (AC) and 2-amino-3-methyl-9*H*-pyrido(2,3-*b*)indole (MeAC), in grilled foods and cigarette smoke condensate were determined by high pressure liquid chromatography and a spectrofluorometer. Two kinds of blended cigarettes were tested. The smoke condensate of one cigarette blend contained 79.7 ng of AC and 6.2 ng of MeAC per cigarette, while the other blend had 80.5 ng of AC and 7.9 ng of MeAC. The proportionate contribution of these mutagens to the carcinogenicity of tobacco smoke, however, has not been established. (Auth. Abs. Mod.)

82–0023. Matsuzaki, H.; Hashimoto, Y. **Purification and Characterization of Acid Phosphodiesterases of Cultured Tobacco Cells.** *Agricultural and Biological Chemistry* 45(6): 1317-1325, June 1981.

Two acid phosphodiesterases (phosphoric diester hydrolase, E.C. 3.1.4) were purified to apparent homogeneity from cultured cells of *Nicotiana tabacum* var. Bright Yellow. These enzymes appeared to differ in some physicochemical properties but exhibited similar broad substrate specificities toward cyclic and *p*-nitrophenyl nucleotides, and bis-*p*-nitrophenyl phosphate. Of these compounds, the most active substrates were bis-*p*-nitrophenylphosphate and 2′,3′-cyclic nucleotides. Both *p*-nitrophenyl 3′- and 5′-thymidylates were hydrolyzed at comparable rates, whereas the hydrolysis of 3′,5′-cyclic AMP occurred preferentially at the 5′-phosphodiester bond. A unique catalytic property of these acid phosphodiesterases was the ability to liberate inorganic phosphate from ATP, ADP, and *p*-nitrophenyl phosphate. Ribonucleic acid, its breakdown products, and inorganic phosphate were found to be effective as inhibitors. (Auth. Abs.)

82–0024. Mokhnachev, I. G.; Mironenko, N. Ye. **Selektivnaya Al'tratsiya Komponentov Tabachnogo Dyma. [Selective Filtration of Smoke Components.]** *Izvestiya Vysshikh Uchebnykh Zavedeniy Pishchevaya Tekhnologiya* (4): 82-86, 1980, Russian.

Retention of dry smoke condensate and nicotine on acetate or cellulose cigarette filters was studied by machine smoking of cigarettes under standard conditions, and determining spectrophotometrically the condensate and nicotine collected on the aerosol filter. Retention of nicotine by both filter types was found to be stronger than that of the condensate in all but one of the tobacco varieties studied. Retention of the condensate decreased and that of nicotine increased when smoke concentration increased, in the experiments with smoking only half of a cigarette with one filter and the second half with a new filter of the same type and length. Smoke from the lower part of a cigarette was more concentrated than from the upper part. Thus, composition of the filtered smoke is an important factor in the retention efficiency of a cigarette filter. A selective retention of nicotine was shown in the experiments with filters of different lengths. Selective retention of smoke components depended on their distribution between aerosol particles of different sizes and on their elution from the filter during successive inhalations. The effect of physicochemical interaction of nicotine with filter material was derived from the fact that more nicotine than condensate is retained by the filter in the subsequent phase of smoking.

82–0025. Parrish, M. E.; Good, B. W.; Hsu, F. S.; Hatch, F. W.; Ennis, D. M.; Douglas, D. R.; Shelton, J. H.; Watson, D. C.; Reilley, C. N. **Computer-Enhanced High-Resolution Gas Chromatography for the Discriminative Analysis of Tobacco Smoke.** *Analytical Chemistry* 53(6): 826-831, May 1981.

The isolation, separation, and identification of the volatile components of cigarette smoke have become increasingly important in tobacco research because of the heightened emphasis placed on the development of lower delivery products. A semiautomated microprocessor-controlled glass capillary gas chromatographic procedure is described, which provides for automatic processing of cigarette smoke with improved reproducibility compared to nonautomated systems. More importantly, the technique provides for the transmission of the digitized chromatographic data from the microprocessor to a host computer facility for postrun processing: profile averaging, profile subtraction, and graphic plotting. Examples are presented that both demonstrate the procedures used and illustrate the value of this approach to a wide range of applications, especially in the area of sensory evaluation. Results which show important parallels between pattern similarity and discrimination by an expert panel are presented. (Auth. Abs.)

82–0026. Prue, D. M.; Martin, J. E.; Hume, A. S.; Davis, N. S. **The Reliability of Thiocyanate Measurement of Smoking Exposure.** *Addictive Behaviors* 6(2): 99-105, 1981.

Procedures for the determination of thiocyanate (SCN) levels in saliva samples were evaluated in a series of three experiments. Laboratory analysis procedures as well as sampling and storage conditions were examined to identify sources of measurement error in the assessment of SCN. The results of the study indicated that spectrophotometric analysis of saliva samples can be a reliable method of determining SCN levels. Also, storage containers, length of storage, and storage temperatures can lead to critical differences in SCN level when saliva samples were not analyzed immediately after sampling. Guidelines for the assessment of saliva SCN are provided in a discussion of the results. (Auth. Abs.)

82–0027. Pryor, W. A. **Mechanisms and Detection of Pathology Caused by Free Radicals. Tobacco Smoke, Nitrogen Dioxide and Ozone.** In: McKinney, J. D. (Editor). *Environmental Health Chemistry: The Chemistry of Environmental Agents as Potential Human Hazards.* Ann Arbor, Michigan, Ann Arbor Science, 1979, pp. 445-466.

The two types of free radicals in cigarette smoke are those in the tar and in the gas phase of smoke. The radicals in tar are long-lived and can be extracted into organic solvents where they are stable. They are ultimately derived from tobacco pigments. One puff of smoke contains more than 10^{15} tar-associated radicals from either mainstream or sidestream smoke. The gas-phase radicals are short-lived, lasting at least 10 minutes in gas phase, but have only a 10-second half-life in solution. Concentrations are greater than 10^{15} radicals per puff in both mainstream and sidestream smoke. These gas-phase radicals are mainly oxygen-centered and diffuse far from the cigarette without an appreciable decrease in concentration. The spin-trap method was used to determine the nature of the radicals; both sidestream and mainstream smoke were found to contain about 10^{17} radicals per gram of smoke.

82–0028. Rosa, N.; Pandeya, R. S. **Estimation of Tobacco Chemical Constituents by Infrared Reflectance Spectroscopy.** *Lighter* 51(1): 17-21, Winter 1981.

The use of infrared reflectance spectroscopy to analyze up to five different tobacco constituents simultaneously is reported. Both InfraAlyzer and laboratory estimates were made and compared for total alkaloids, reducing sugars, and total nitrogen in cured tobacco lamina samples. These parameters, which are useful in the assessment of cured leaf quality of flue-cured tobacco, can have a wide variation in chemical constituents depending on leaf color, body, and plant position. Results showed a strong association of the constituents. There were also strong correlations between the laboratory and InfraAlyzer estimates for all three constituents. Although infrared reflectance spectroscopy can simultaneously analyze tobacco samples for such constituents, its accuracy and reliability may depend on laboratory values being used for calibration. The infrared technique can be used, however, in the preliminary screening of a large number of plants in a short time.

82–0029. Seeman, J. I.; Secor, H. V.; Chavdarian, C. G.; Sanders, E. B.; Bassfield, R. L.; Whidby, J. F. **Steric and Conformational Effects in Nicotine Chemistry.** *Journal of Organic Chemistry* 46: 3040-3048, July 1981.

The stereoselectivity of iodomethylation of nicotine and seven nicotine analogs having pyridine alkyl groups was determined by using ^{13}C nuclear magnetic resonance (NMR). Alkylation at the pyridine (N) and at the pyrrolidine (N') nitrogens was observed. Two modes of N'-iodomethylation occurred, cis and trans to the pyridine ring. The N'-iodomethylation occurred regioselectively cis to the pyridine ring for all compounds examined. The N/N' and N'_{cis}/N'_{trans} ratios for the nicotinoids were evaluated with regard to (1) the orientation of the N'-methyl group in the free base, (2) conformational properties of the pyridine ring with respect to the pyrrolidine ring, and (3) steric hindrance and buttressing effects on the pyridine nitrogen. Methyl groups at C_2 or C_4 of nicotine reduced the relative rate of N' alkylation. Methyl groups at C_4 and C_5 increased the relative rate of N alkylation, but reduced the rate when at C_2 and C_6. Methyl groups at C_5 and C_4 appeared to increase the rate of N' alkylation relative to nicotine. (Auth. Abs. Mod.)

82–0030. Wald, N.; Doll, R.; Copeland, G. **Trends in Tar, Nicotine, and Carbon Monoxide Yields of UK Cigarettes Manufactured Since 1934.** *British Medical Journal* 282(6266): 763-765, March 7, 1981.

Between 1934 and 1979 the average tar yield of cigarettes manufactured in the United Kingdom decreased by 49 percent, nicotine by 31 percent, and carbon monoxide by 11 percent, all estimated on a sales-weighted basis. The average tar yield decreased progressively after the Second World War, due to the introduction of filter cigarettes and to changes in the manufacture of plain cigarettes. The average nicotine yield increased initially, decreased by 43 percent from about 1950 to 1974, but increased again by 9 percent between 1974 and 1979. The average carbon monoxide yield started to decrease about 1961; while it decreased substantially in plain cigarettes, the rapid increase in sales of filter cigarettes at this time, at the expense of plain cigarettes, largely offset the reduction in carbon monoxide yield that would otherwise have occurred. As with nicotine, carbon monoxide yield showed a small rise in later years (4 percent between 1976 and 1979). The trends in tar yield may explain the reduction in lung cancer in the United Kingdom better than has been suspected, while the trends in nicotine and carbon monoxide yields are probably not sufficiently different to distinguish which of them might be the more likely cause of cardiovascular disease. (Auth. Abs.)

82–0031. Williamson, R. E.; Chaplin, J. F. Levels of Chemical Constituents in Cured Leaves of Four Burley Tobacco Cultivars According to Stalk Position. *Tobacco International* **183(15): 182-185, July 24, 1981.**

The levels of 15 chemical constituents and pH of tobacco (*Nicotiana tabacum* L.) were determined in cured leaves from eight stalk positions of Burley cultivars Burley 21, Ky 12, Ky 14, and a low-alkaloid breeding line, LA Burley 21, selected for a range in total alkaloid content. Reducing sugars, total alkaloids, total polyphenols, chlorides, alpha-amino N, total N, protein N, nitrate N, petroleum ether extract, waxes, holocellulose, ash, starch, water-soluble acids, alkalinity of water-soluble ash, and pH were determined. Differences due to cultivars and stalk position were significant for most constituents. Cultivar X stalk position interactions were nonsignificant for levels of all constituents except total polyphenols and alpha-amino N. Levels of chemical constituents for cultivar averaged over stalk positions were significant for reducing sugars, total alkaloids, total polyphenols, chlorides, alpha-amino N, total N, nitrate N, waxes, holocellulose, ash, starch, water-soluble acids, alkalinity of water-soluble ash, and pH. When averaged over cultivars, differences due to stalk position were significant for all constituents except starch. The results show that considerable variation exists between cultivars and stalk positions for levels of several chemical constituents in Burley tobacco. (Auth. Abs.)

PHARMACOLOGY AND TOXICOLOGY

82–0032. Andersson, K.; Fuxe, K.; Agnati, L. F. **Effects of Single Injections of Nicotine on the Ascending Dopamine Pathways in the Rat. Evidence for Increases of Dopamine Turnover in the Mesostriatal and Mesolimbic Dopamine Neurons.** *Acta Physiologica Scandinavica* 112(3): 345-347, July 1981.

The effects of nicotine administered systematically on the ascending dopamine pathways of the rat were analyzed in male rats weighing from 150 to 200 g. A dose of 0.3 or 1 mg/kg i.p. nicotine in tartrate form was given 1 hour before decapitation and analysis. The dopamine nerve terminal systems were studied in the marginal zone of the nuc. caudatus, the medial and central part of the nuc. caudatus, the anterior and posterior part of the nuc. accumbens, and the posterolateral and posteromedial part of the olfactory tubercle. There was a significant reduction in dopamine stores in the medial caudate and in the anterior part of the nuc. accumbens after a single injection of 1 mg/kg i.p. nicotine. There was a preferential increase in dopamine turnover within the medial caudate and within the posterolateral part of the olfactory tubercle. A dose of 0.3 mg/kg nicotine resulted in a selective reduction of dopamine stores in the anterior part of the nuc. accumbens. Results appear to indicate that nicotine administered in one single acute dose, given systematically, can change dopamine levels and turnover in both striatal and subcortical limbic dopamine nerve terminal systems. Nicotine also appeared to increase dopamine turnover in the mesostriatal and mesolimbic dopamine nerve terminal systems. It is suggested that nicotine dependence may be related to an enhancement of activity in certain types of mesolimbic dopamine neurons.

82–0033. Ashton, H.; Stepney, R.; Thompson, J. W. **Should Intake of Carbon Monoxide Be Used as a Guide to Intake of Other Smoke Constituents?** *British Medical Journal* 282(6257): 10-13, January 3, 1981.

The relationship between blood carboxyhemoglobin (COHb) and plasma nicotine concentrations was studied in a group of 12 smokers smoking cigarettes of three levels of standard delivery. While the intake of carbon monoxide from a single cigarette was unrelated to the intake of nicotine, presmoking "trough" concentrations of the two substances (reflecting longer term exposure) were highly correlated. Various other measures of nicotine exposure were at best only moderately correlated with blood nicotine concentrations. Thus trough COHb concentrations might be used to provide a reliable indication of the exposure to nicotine of individual smokers smoking the same type of cigarette, and of the relative exposure to nicotine of populations smoking cigarettes of different standard deliveries. (Auth. Abs.)

82–0034. Bachner, I.; Bachner, J.; Lenz, K. **Freisetzung und Resorption von Nikotin aus Zigarettentabak. Eine in-vitro-Studie. [Liberation and Absorption of Nicotine From Cigarette Tobacco. An In Vitro Study.]** *Scientia Pharmaceutica* 48(3): 241-247, 1980, German.

The extraction rate of nicotine from tobacco of a single Austrian cigarette of a mild variety was determined by leaching tobacco with synthetic gastric juice at a pH of 1.1 and 3.0 at room temperature for 2 to 120 minutes; the nicotine content of the extracts was then determined by high-pressure liquid chromatography. About 80 to 90 percent of the total nicotine of the cigarette was extracted relatively fast at both pH values. Nicotine absorption from synthetic gastric juice at a pH of 1.1 or 3.0 and from synthetic intestinal juice at pH 7 was determined by making it diffuse through an artificial dipoid membrane under conditions reproducing those in the human gastrointestinal tract. Only 2.9 or 3.3 percent of the nicotine content of the gastric juice diffused in 3 hours through the membrane into synthetic plasma. This absorption ratio corresponds to 0.15 mg of absorbed nicotine from a cigarette which averages 4.95 mg nicotine; that is a toxicologically insignificant amount. In contrast, nicotine is readily absorbed from a neutral intestinal juice into the small intestine from which it proceeds directly into the liver with a half-life of 7.8 minutes.

82–0035. Beleslin, D. B.; Krstic, S. K.; Stefanovic-Denic, K.; Strbac, M.; Micic, D. **Inhibition by Morphine and Morphine-Like Drugs of Nicotine-Induced Emesis in Cats.** *Brain Research Bulletin* 6(5): 451-453, 1981.

The injection of morphine, methadone, and pethidine into the cerebral ventricles of unanesthetized cats initially induced a transient emesis, but afterwards depressed or abolished the emetic action of nicotine that had been similarly injected earlier. Nicotine and morphine have been found in previous investigations to induce emesis when injected into the cerebral ventricles of cats. Since both drugs affect emesis similarly, the study was conducted to examine the effects of morphine and the morphine-like drugs, methadone and pethidine, upon nicotine-induced emesis in the cat. It was suggested that the emetic and anti-emetic effects of morphine, methadone, and pethidine might be due to agonist—antagonist activity. The area postrema of the fourth ventricle may have been the site where the nicotine-induced emesis was inhibited.

82–0036. Crawford, F. E.; Back, D. J.; Orme, M.L'E.; Breckenridge, A. M. **The Effect of Cigarette Smoking on Plasma Concentrations of Oral Contraceptive Steroids.** *British Journal of Clinical Pharmacology* 11(4): 438p-439p, 1981.

A group of 311 women attending a family planning center was studied to determine the effects of cigarette smoking on the plasma levels of oral contraceptive steroids. Each woman took one of three available oral contraceptives: Eugynon 30 (30 μg ethinylestradiol or EE_2, and 250 μg levonorgestrel or Ng); Microgynon (30 μg EE_2 and 150 μg Ng); or Ovranette (30 μg EE_2 and 150 μg Ng). A blood sample was taken and information recorded for: weight, age, time of previous pill, other medications, and smoking status. The samples were analyzed for plasma concentrations of EE_2 and Ng. Plasma thiocyanate levels were also measured to verify smoking habit information. There was a significant difference ($P < 0.001$) between plasma thiocyanate levels in smokers (9.2 ± 4.0 μg/ml) and nonsmokers (2.0 ± 1.4 μg/ml). The EE_2 and Ng plasma concentrations did not differ significantly in smokers and nonsmokers at any of the time intervals studied. There was a wide variation in individual plasma levels in both groups, with EE_2 smokers showing a range of 3 to 279 pg/ml; EE_2 nonsmokers, 3 to 450 pg/ml; Ng smokers, 0.44-0.8 ng/ml; and Ng nonsmokers, 0.25 to 5.38 ng/ml. Cigarette smoking did not appear to alter the rate of metabolism of contraceptive steroids.

82–0037. Dmitriev, M. T. **Gigienicheskaya Otsenka Tabachnogo Dyma. [Hygienic Evaluation of Tobacco Smoke.]** *Zdravookhranenie Belorussii* (10): 25-28, 1980, Russian.

Average toxicity data (concentrations, pollution index, fraction of total toxicity) for the most important smoke components are tabulated, and the toxic effect of each important smoke component on human organism is explained in detail. The high value of the summary pollution index (expressed as the maximum permissible concentration) of tobacco smoke is connected with extraordinary imperfection of tobacco combustion. Formation of different toxic components in the complex process of tobacco combustion is explained. Mathematical expressions are derived for hygienic evaluation of tobacco smoke. The expressions are applied to calculate ventilation standards for the premises where smoking is permitted and the maximum permissible rate of cigarette smoking (0.036 of a cigarette/day) without harmful effect on the human organism. Chemical load on organism of a smoker is compared with the load on the organism from automobile exhaust gases.

82–0038. Driscoll, P.; Baettig, K. **Selective Inhibition by Nicotine of Shock-Induced Fighting in the Rat.** *Pharmacology Biochemistry and Behavior* 14(2): 175-179, 1981.

Individual stress may be one factor increasing the likelihood of taking up the smoking habit. It is now believed that one reinforcement of smoking behavior is a nicotine-mediated reduction in the impact of aversive stimuli. This effect helps protect the coping ability of the smoker from disruptive reactions such as aggressive behavior and/or a reduction in efficiency. The present study investigated nicotine effects on rats as a possible animal model for human behavior. The frequency of shock-induced fighting, posturing and "no reaction" (running, jumping, or freezing), after acute SC injections of 0, 0.1, 0.2, and 0.4 mg/kg nicotine, was measured in 38 pairs of male and 12 pairs of female rats. Sensitivity to footshock was also measured, at the same nicotine doses, in males. Nicotine inhibited shock-induced fighting in 32 pairs of high-frequency fighting males in a dose—dependent fashion, with fighting being gradually replaced by posturing at the 0.1 and 0.2 mg/kg doses. There was a significant increase in the "no reaction" category (especially freezing behavior) only at 0.4 mg/kg, indicating that reduced activity may have been partly responsible for the reduction in fighting seen at that dose. None of the doses had a significant effect on sensitivity to footshock. Nicotine had no effect on shock-induced fighting in the six low-frequency fighting male pairs, and affected the female pairs only at the 0.4 mg/kg level, where fighting was also decreased due to an increase in the "no reaction" category. It was concluded that small doses of nicotine, under favorable conditions, were capable of inhibiting shock-induced fighting in rats without altering shock sensitivity or depressing activity. (Auth. Abs. Mod.)

82-0039. Feely, J.; Crooks, J.; Stevenson, I. H. **The Influence of Age, Smoking and Hyperthyroidism on Plasma Propranolol Steady State Concentration.** *British Journal of Clinical Pharmacology* 12(1): 73-78, 1981.

Plasma propranolol steady state concentration (Css) was determined during chronic dosage (160 mg per day) in 22 hyperthyroid patients aged 16 to 75 years (11 smokers, 11 nonsmokers) and again following treatment when euthyroid. Plasma propranolol Css in hyperthyroid patients was lower in smokers than in nonsmokers ($P<0.05$). The mean levels among smokers (age 45 ± 5 years) and nonsmokers (46 ± 5 years) were 33.6 ± 7.8 ng/ml and 61.9 ± 15.9 ng/ml, respectively. There was a significant relationship between age and plasma propranolol Css for smokers ($r=0.82$, $P<0.01$) and nonsmokers ($r=0.78$, $P<0.01$). When the patients achieved the euthyroid condition after treatment for hyperthyroidism, there was again a significant correlation, ($r=0.74$, $P<0.05$) between plasma propranolol Css and age in smokers, though not in nonsmokers. The mean plasma propranolol Css in the euthyroid condition was higher in nonsmokers (87.4 ± 19.6 ng/ml) than in smokers (58.1 ± 6.4 ng/ml) but did not reach statistical significance. Hyperthyroidism and smoking are known to increase the rate of drug metabolism, and it is suggested that these variables may give rise to or accentuate an age-related reduction in propranolol clearance. (Auth. Abs. Mod.)

82-0040. Jaffe, J. H.; Kanzler, M.; Friedman, L.; Stunkard, A. J.; Verebey, K. **Carbon Monoxide and Thiocyanate Levels in Low Tar/Nicotine Smokers.** *Addictive Behaviors* 6(4): 337-343, 1981.

A comparison of smokers and nonsmokers shows that carbon monoxide (CO) in expired breath correlates significantly with the number of cigarettes smoked, and thiocyanate (SCN) levels are higher in smokers; however, the CO and SCN readings do not appear to be significantly affected by the tar and nicotine levels of the brands. Smokers, male and female, were subdivided into four categories according to brand nicotine content: high ($\geq$ 1.0 mg/cig), light (0.9 to 0.6), low (0.5 to 0.3), and very low (0.2 to 0.1). The CO and SCN tests were conducted to determine if those smoking lower tar and nicotine cigarettes might compensate by smoking more, maintaining nicotine intake while increasing levels of toxic gases such as CO or hydrocyanic acid. The CO tests used 200 smokers and 28 nonsmokers. Although CO levels were lower among those who smoked very low tar and nicotine cigarettes, the difference did not reach statistical significance. The thiocyanate saliva test (150 smokers and 52 nonsmokers) found that higher SCN levels of smokers did not vary significantly among brands, although a trend toward higher SCN levels was found among males who smoked very low yield brands. The prediction that switching to lower yield brands would substantially increase CO or SCN levels was not supported, with apparently little additional risk associated with switching to lower yield cigarettes.

82-0041. Kagedal, B.; Martensson, J.; Sorbo, B.; Tibbling, L. **Serum Levels of Thiocyanate and Thyroid Hormones in Smoking and Non-Smoking Subjects.** *Research Communications in Substance Abuse* 2(3): 267-275, 1981.

It is known that tobacco smoke contains significant amounts of hydrogen cyanide, which is converted to thiocyanate in the human body. Thiocyanate, free thyroxine index, free triiodothyronine index, and thyrotropin were determined in smoking and nonsmoking subjects. Although the serum thiocyanate levels were definitely raised in cigarette smokers, no effect on the thyroid hormone levels was observed. The subjects studied were on an adequate iodine intake, as verified by iodide analyses in urine from a selected number of subjects. It was concluded that the amounts of thiocyanate formed in cigarette smokers exert no significant thyrostatic effect on subjects who are not iodine deficient. (Auth. Abs. Mod.)

82-0042. Langone, J. J.; Vunakis, H. V. **Radioimmunoassay of Pharmacologically Active Compounds: Applications to Nicotine and Its Metabolites.** In: Albertini, A., Da Prada, M., Peskar, B. A. (Editors). *Radioimmunoassay of Drugs and Hormones in Cardiovascular Medicine.* Symposia of the Giovanni Lorenzini Foundation, Volume 3, 1979, pp. 55-70.

Sensitive and specific radioimmunoassays were developed for nicotine, and its metabolite cotinine. These assays have been used to analyze the nicotine and cotinine content of serum, urine, amniotic fluid, cord blood, and spinal fluid in individuals who smoke. The nicotine content of cigarettes smoked, and cotinine levels in the blood stream did not correlate well, which may indicate differences in smoking habits, metabolic capabilities, or other factors. The half-life of nicotine is about 30 min in serum, while cotinine has a half-life of 20 to 30 hours. Cotinine was found to remain in the blood for nearly 2 weeks after a subject has quit smoking. Up to 120 ng/ml of cotinine and 40 ng/ml nicotine were detected in the amniotic fluid of pregnant women in the 14th to 20th weeks of gestation. Significant levels of cotinine were also present in spinal fluid of 11 smokers tested, and in tobacco harvest workers there was a tenfold increase of cotinine in those with direct contact with the tobacco. Since cotinine occurs in only trace amounts in tobacco, dermal nicotine absorption is probably responsible for green tobacco sickness.

82-0043. Mascres, C.; Franchebois, P. **Observations Histochimiques de la Muqueuse Buccale Chez le Patient Cirrhotique non Fumeur. [Histochemical Observations of Oral Mucosa in the Nonsmoking Cirrhotic Patient.]** *Pathologie Biologie* 29(4): 203-210, April 1981, French.

The effect of chronic alcoholism and smoking on oral mucosa was studied by comparing biopsies from a sample of 12 cirrhotic nonsmoking patients with biopsies from 10 cirrhotic patients who smoked and 10 healthy nonsmoking controls. The smoking patients used to consume 1.5 to 4 packs of cigarettes/day. Stains and enzymatic reactions were used in histological examinations of the oral mucosa specimens. Although there were more similarities than differences between the two cirrhotic groups, the following differences were observed: (1) much stronger activity of acid phosphatase in the epithelium of nonsmokers; (2) the presence of the sulfhydryl and disulfide groups in the surface keratin of nonsmokers and the absence of these groups in smokers; (3) a less pronounced nuclear activity (DNA) in cirrhotic smokers than in nonsmoking patients; (4) more reactive basal cell mitochondria in nonsmoking than in smoking patients; and (5) enzymatic changes in the vascular walls made evident by a decrease of thiamine pyrophosphatase and alkaline phosphatase activities in cirrhotic nonsmokers and an increase in these activities in the smokers.

82-0044. Morosco, G. J.; Nightingale, T. E.; Frasinel, C.; Goeringer, G. C. **Pancreatic Elastase Activation as a Possible Indicator of the Relative Hazard of Different Cigarettes.** *Journal of Toxicology and Environmental Health* 8: 89-94, 1981.

Dogs were exposed on an acute basis to smoke from high-nicotine and from nicotine-free cigarettes. Anesthetized animals were prepared by laparotomy, duodenotomy, and pancreatic duct cannulation. Pancreatic fluid was collected and preexposure elastase levels were determined. Samples were then taken during exposure to smoke from cigarettes of both types. There was no measurable elastase activity in the baseline samples. Similarly, no activity was found in fluids collected before and during exposure to smoke from nicotine-free cigarettes. Enzyme activity was detected, however, in samples collected during exposure to smoke from high-nicotine cigarettes once blood levels of nicotine reached 50-70 ng/ml. The results suggest that cigarette smoke may provoke activation of elastase and indicate the need for further study of any association of cigarette smoking with pancreatic disease. (Auth. Abs.)

82-0045. Nightingale, T. E.; Frasinel, C.; Swidersky, P.; Galarraga, J. **Cigarette Smoke Inhalation and Blood Dose Markers in Beagle Dogs.** *Toxicology and Applied Pharmacology* 60(3): 451-457, September 30, 1981.

The time course of three tobacco-related blood dose markers was determined in beagle dogs during and after two-, four-, and six-cigarette acute smoke exposures. Venous blood samples were taken from a jugular catheter at selected intervals and analyzed for nicotine, cotinine, and carboxyhemoglobin (COHb). Smoke was generated from reference cigarettes (1.91 mg of nicotine) by a machine and delivered to the trachea of tracheostomized dogs. Cigarettes were chain-smoked in pairs with a 5-minute break between pairs when four or six cigarettes were smoked. Blood nicotine, cotinine, and COHb levels all rose during smoke inhalation. Peak values of COHb were dosimetrically related to the number of cigarettes, while peak nicotine and cotinine values were not related consistently. The exposure protocol used in this study produced blood levels of all three measured parameters which were in the range of published values for human smokers, so that it is possible to approximate acute human cigarette smoke inhalation in the beagle dog model. (Auth. Abs.)

82-0046. Ochs, H. R.; Greenblatt, D. J.; Otten, H. **Disposition of Oxazepam in Relation to Age, Sex, and Cigarette Smoking.** *Klinische Wochenschrift* 59(33): 899-903, September 1981.

The kinetics of single 30-mg oral doses of oxazepam were determined in 22 male and 9 female volunteers aged 20 to 86 years. Oxazepam plasma concentrations were measured in multiple plasma samples drawn during 36 hours after each dose. Mean kinetic variables in males and females, respectively, were: elimination half-life, 7.5 and 8.5 hours; volume of distribution, 0.96 and 1.17 l/kg; clearance, 1.48 and 1.70 ml/min/kg. Sex differences were not significant, nor were any of the kinetic variables significantly related to age. However, oxazepam clearance increased significantly with heavier cigarette smoking ($r=0.48$, $p<0.01$). Mean clearance in smokers (1.98 ml/min/kg) was significantly higher than in nonsmokers (1.23 ml/min/kg, $p<0.01$). Thus, smoking is a more important determinant of oxazepam clearance than age or sex. (Auth. Abs.)

82-0047. Pleho, A. **Changes in Manganese and Copper Ions Toxicity Under the Influence of Nicotine—Their Effects on the Permeability of the Small Intestine.** *Folia Medica (Sarajevo)* 14(1): 123-129, 1979.

The change of permeability of the wall of jejunum and ileum for Na salicyl induced by nicotine was investigated in white rats treated with manganese (Mn) and with copper (Cu). The investigation was conducted on three differently treated groups of animals with Mn and Cu: (1) the content of one of the metals in clinically healthy animals was increased by administration of the metal in doses of 15 mg/kg Mn and 3 mg/kg Cu body weight for 6 days; (2) after previous treatment with nicotine in increasing subconvulsive doses, there followed application of Mn or Cu in single doses; and (3) after decreasing the blood volume for 1 ml/100 gr body weight, there followed application of the metals in doses of 15 mg/kg for Mn, and 3 mg/kg for Cu. The change of permeability induced by nicotine occurred with both metals, only in reverse direction. In intoxication with Mn, nicotine led to increased permeability, while in intoxication with Cu the permeability was decreased. The decrease of blood volume and replacement of lost iron afterwards augmented the basic effect of the metal on clinically healthy animals. (Auth. Abs.)

82-0048. Rasmussen, R. E.; Boyd, C. H.; Dansie, D. R.; Kouri, R. E.; Henry, C. J. **DNA Replication and Unscheduled DNA Synthesis in Lungs of Mice Exposed to Cigarette Smoke.** *Cancer Research* 41: 2583-2588, July 1981.

An investigation of the effect of cigarette smoke upon laboratory mice found that chronic smoke exposure caused persistent changes in the replication and repair of genetic material, though the role of the changes in oncogenesis was not established. Mice of the hybrid strain BC3F1/Cum (C57BL/Cum x C3H/AnfCum) were chronically exposed to measured amounts of machine-generated whole Kentucky reference 2A1 cigarette smoke. DNA replication and unscheduled DNA synthesis (UDS) were measured in lung tissue in vitro using a short-term organ culture method. Within 1 week of beginning smoke exposure, DNA replicative activity, as indicated by incorporation of (^{3}H) thymidine into total lung DNA, was increased more than twofold over sham-exposed controls and remained elevated as long as smoke exposure was continued. Treatment of lung tissues in vitro with either the lung carcinogen 4-nitroquinoline-1-oxide or methylmethane sulfonate stimulated UDS, measured as incorporation of (^{3}H)thymidine into lung DNA in the presence of hydroxyurea, presumably as the result of DNA repair activity. Until the 10th to 12th week of smoke exposure, at which time the accumulated deposition of total particulate material in the lung was approximately 40 mg, the level of UDS stimulated by the alkylating chemicals declined to approximately 50 percent of that seen in lung tissue from sham-exposed control mice. If the mice were removed from smoke exposure, DNA replicative activity returned to normal levels within 1 week, but the UDS response to DNA damage remained depressed up to 5 months after ending smoke exposure. The results showed that both transient and apparently permanent changes were produced in mouse lung as the result of exposure to cigarette smoke. The role of these changes in lung neoplasia is under investigation. (Auth. Abs. Mod.)

82-0049. Reaves, T. A., Jr.; Liu, H. M.; Qasim, M.; Hayward, J. N. **Vasopressin Release by Nicotine in the Cat.** *Peptides* 2(1): 13-17, 1981.

A study to determine the sites of action of nicotine for the neurohypophysial release of arginine vasopressin (AVP) is described. In the chamber-isolated, unanesthetized cat, an IV infusion of nicotine (25-50 μg/kg/min for 10 min) produced a 54-fold rise in plasma AVP (65±12.5 μU/ml) and a behavioral sequence of restlessness, ear twitching, salivation, chewing, and retching. Chloralose anesthesia and acute surgical preparation increased plasma AVP (9.4±3.8 μU/ml) four times the control level in the unanesthetized state (2.4±0.1 μU/ml). In the anesthetized cat, an IV infusion of nicotine produced a 140-fold rise in plasma AVP (802.7±289.0 μU/ml) and a biphasic response in mean arterial blood pressure (MABP) which rose to 32 percent above control in the first 5 min and fell to 25 percent below control during the last 5 min. Bilateral surgical section of the carotid sinus and vagus nerves increased plasma AVP (47.6±25.8 μU/ml) to five times the control level in the anesthetized state (9.4±3.8 μU/ml). In the anesthetized, baroreceptor-denervated cat, an IV infusion of nicotine produced a three-fold rise in plasma AVP (141.5±31.3 μU/ml) and a biphasic MABP response. Hypophysectomy abolished the nicotine-induced rise in AVP but did not modify the biphasic MABP response. These data suggest that in the cat nicotine releases AVP from the neurohypophysis by multiple sites of action within the receptive fields of the carotid sinus and vagus nerves and at unknown loci within the central nervous system. (Auth. Abs.)

82-0050. Romano, C.; Goldstein, A.; Jewell, N. P. **Characterization of the Receptor Mediating the Nicotine Discriminative Stimulus.** *Psychopharmacology* 74(4): 310-315, September 1981.

Results from T-maze testing of male albino rat behavior suggested that the discriminative stimulus or cue receptor for nicotine was pharmacologically similar to the nicotine cholinergic receptor in autonomic ganglia. It was already known that nicotine presumably provides a recognizable cue for self-administration by smokers. During training days, the rats received subcutaneous (SC) administrations of nicotine tartrate (N) at 0.4 mg base/kg SC in 1.0 ml/kg saline or saline at 1.0 ml/kg SC. After 5 minutes, the rat was placed in the center arm of an electrified T-maze, with nonelectrified goal boxes at the extremes of the right and left arms. Only one of the goal boxes was available on a given day, determined by the drug condition. For a given rat, for example, only the right arm's goal box would be accessible after N administration. The rat's initial direction of turning was noted as correct or incorrect. Drugs which could act as potential agonists or antagonists were also tested. For rats trained on 0.4 mg N/kg, the effective dose needed for 50 percent correct test scores (ED_{50}) was 0.11 mg N/kg. The enantinomer of natural nicotine (+)nicotine was much less potent, and both position isomers of nicotine were inactive. Anabasine, which is active at nicotinic cholinergic receptors, provided the nicotine cue. Cytisine, a potent nicotinic agonist in vitro, was ineffective after SC administration, shown to be due to its inability to enter the brain in adequate amounts. High doses of cytisine by the intracerebroventricular route partially provided the cue. The cue was blocked by low doses of mecamylamine and pempidine and by high doses of hexamethonium, acting as antagonists. (Auth. Abs. Mod.)

82–0051. Rondahl, L. **Synthetic Analogues of Nicotine. X. Synthesis and Biological Testing of Some N-(arylmethyl)azacycloalkanes.** *Acta Pharmaceutica Suecica* 17(6): 347-351, 1980.

Four synthetic analogs of nicotine (aziridine and azetidine analogs), numbered 4 to 7, were prepared and tested for biological nicotine-like activity on guinea-pig *vas deferens* , frog *rectus* , and rabbit *jejunum* preparations. Substances 4 and 5, N-benzylaziridine and N-benzylazetidine, displayed high activity on the guinea-pig preparation, but were inactive on the frog or rabbit tissues. Derivatives 6 and 7, N-(3-pyridylmethyl)aziridine and N-(3-pyridylmethyl)azetidine, exhibited low but significant activities on the guinea-pig, and small activities on the frog preparation. No reaction on rabbit tissue is mentioned. The high activity of analogs 4 and 5 was surprising. The pyridine ring has been theorized as necessary for nicotine-like activity. Synthesis of the compounds is carefully described.

82–0052. Russell, M.A.H.; Jarvis, M. J.; Devitt, G.; Feyerabend, C. **Nicotine Intake by Snuff Users.** *British Medical Journal* 283(6295): 814-817, September 26, 1981.

Blood nicotine and cotinine concentrations were measured in 27 volunteers before and after taking snuff. Within 10 min after snuffing, blood nicotine concentrations were comparable to those obtained after the 10 min or so that it takes to smoke a cigarette. Nicotine intake from snuffing was related to the experience of the snuffer. In daily and occasional snuffers, increases in plasma nicotine concentrations averaged 77.7 and 12.3 nmol/l (12.6 and 2.0 ng/ml) respectively. The increase shown by the daily snuffers was comparable to the average increase of 62.3 nmol/l (10.1 ng/ml) obtained from a single cigarette by a group of heavy smokers. The peak nicotine concentrations in the daily snuffers were also similar to the peak values in 136 heavy smokers, 222.6 and 226.3 nmol/l (36.1 and 36.7 ng/ml), respectively. Unusual multiple-dose snuffing produced massive increases in plasma nicotine to concentrations that have never been recorded in smokers. The similarity of the concentrations produced by regular daily snuffing and regular daily smoking suggests that the plasma nicotine concentration has some controlling influence over the self-regulation of these two quite different forms of tobacco use. The rapid absorption of nicotine from snuff confirms its potential as an acceptable and relatively harmless substitute for smoking, since it is free of the tar, carbon monoxide, and nitrogen oxides found in tobacco smoke. Because snuff is not inhaled into the lungs, there is less risk of lung cancer, bronchitis, or emphysema; however, snuff may be associated with oral cancer. The possible effects of taking snuff on coronary heart disease are not clear. (Aut. Abs. Mod.)

82–0053. Sabharwal, P. S.; Lockard, J. M. **Biological Effects of Maleic Hydrazide.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 141-157.

Maleic hydrazide (MH) is used in tobacco to inhibit plant growth by chromosome breakage and inhibition of mitosis. Tests were conducted to investigate the possibility of similar effects in mammalian chromosomes. Maleic hydrazide and two commercial formulations of MH that are sprayed on tobacco, MH_{30} and Royal MH_{30}, were tested in three short-term bioassays. Toxicities of all three chemicals were very low in cultured rat lung cells. However, the toxicities of MH_{30} and Royal MH_{30} were greater than that of the active ingredient, MH, suggesting that the inert ingredients of the commercial formulations are not biologically inactive. MH, MH_{30}, and Royal MH_{30}, all made by Uniroyal, did not induce micronuclei in mouse bone marrow cells, but MH from another commercial source induced a dose-related increase in micronuclei, indicating that it had clastogenic effects. MH from the same source as that which induced micronuclei and Royal MH_{30} from Uniroyal caused slight increases in sister chromatid exchanges in human lymphocytes when applied in high concentrations. Further investigations into the possible chromosome-damaging effects of MH and its commercial products are recommended. (Auth. Abs. Mod.)

82–0054. Sidorski, S.; Mysliwiec, M.; Bielawiec, M. **Fibrynoliza i Krzepniecie Krwi a Palenie Papierosow. [Fibrinolysis, Blood Clotting and Cigarette Smoking.]** *Polski Tygodnik Lekarski* 36(2): 57-60, January 12, 1981, Polish.

Hematocrit, clotting time of calcified plasma, blood fibrinogen level, and euglobulin fibrinolysis were determined in 111 male and female cigarette smokers and 36 male and female nonsmokers (controls), aged 20 to 62 years. Blood samples were taken from the smokers in the morning before smoking and 10 minutes after they had smoked five nonfiltered cigarettes within a 1-hour period. Significant differences were found between the smokers and the controls with respect to clotting time (t) and fibrinolytic activity index (1,000/t): t was shorter in smokers, particularly men, than in controls; and 1,000/t index was lower in male smokers. Also significant were differences in the 1,000/t index and fibrinogen levels in the smokers before and after smoking five cigarettes: the 1,000/t index increased; hence fibrinolysis was more active, as well as fibrinogen level after smoking, particularly in women. The results suggest that male cigarette smokers have a tendency of hypercoagulability and inhibition of fibrinolytic activity in blood, particularly after smoking a number of cigarettes within a short period. Increased fibrinolytic activity in female smokers after smoking is considered as a compensatory defensive mechanism.

82–0055. Sullivan, J. M.; Connors, N. A.; Rana, M. W. **The Effects of Nicotine on the Rat Adrenal Medulla.** *IRCS Medical Science: Nervous System* 8: 805-806, 1980.

In this study of the anatomical effects of nicotine on rat adrenal medulla, 36 Sprague-Dawley rats (average weight, 60 grams) were divided into four groups. Group I received one injection (0.5 mg/kg nicotine/day); group II, two injections (1.0 mg/kg nicotine/day); group III, three injections (1.5 mg/kg nicotine/day); and group IV, 10 ml of normal saline injection/day as a control. After 60 days, the animals were sacrificed and the tissues imbedded, serially sectioned, and stained for examination. The control rats weighed an average of 10 percent more than the experimental rats. Also, in the adrenal medulla of the experimental rats more epinephrine-secreting cells were present; and hyperplastic medullary tissue (short cords and acina of columnar, ovoid, or polyhedral shaped chromaffin cells, with vascular hyperplasia) was observed, indicating that nicotine caused a delaying of differentiation of medullary ganglionic cells.

82–0056. Sullivan, J. M.; Rana, M. W. **Effects of Nicotine on Norepinephrine Metabolism in Female Rat Adrenal Medulla.** *IRCS Medical Science: Physiology* 9: 612, 1981.

A study to quantitatively analyze norepinephrine (NE) and epinephrine (EPI) within the female rat adrenal medulla due to the action of intraperitoneal injections of nicotine is reported. Sprague-Dawley female rats ($n = 12$) were equally divided into an untreated group, and a treatment group which received 1.0 mg/kg nicotine, dissolved in normal saline solution, in one injection/day for 60 days. The untreated group received 1.0 ml of normal saline on the same treatment schedule. The average NE levels in the adrenal glands of treated animals were found to be 18.1 ± 2.8 μg, compared to 8.9 ± 0.47 μg in the untreated group, an elevation considered significant ($p < 0.01$). The average levels of EPI were 33.2 ± 3.3 μg in the adrenal glands of treated animals and 25.0 ± 1.13μg in untreated animals, also significant ($p < 0.05$). Study results confirm that chronic administration of nicotine, in a dose approximating that received by moderate cigarette smokers, causes biochemical changes in the chromaffin cells of the adrenal medulla of the female rat.

82–0057. Sullivan, J. M.; Rana, M. W. **Effects of Nicotine on Norepinephrine Metabolism in Female Rat Lung.** *IRCS Medical Science: Drug Metabolism and Toxicology* 9: 772-773, 1981.

In a study to determine the levels of norepinephrine (NE) within female rat lung after intraperitoneal injection of nicotine, 24 young female Sprague-Dawley rats were divided into experimental and control groups. Experimental rats received 1.0 mg/kg nicotine/day, dissolved in normal saline solution. Each control received 1.0 ml of normal saline injection/day. The first effects observed from the nicotine injections were increased breathing and sympathetic stimulation. There was also an increase in NE within the postganglionic sympathetic nervous system fibers of the treated rats, mostly in nerves located in the tunica adventitia-tunica media junction of arteries. The average levels of NE in the lungs of treated rats were 98 ± 7.5 μg, while untreated rats measured 86 ± 10.4 μg. The slight change of NE from the chronic administration of nicotine, in a dose which approximated that obtained by moderate cigarette smokers, was not statistically significant.

82–0058. Sullivan, S. L. (Editor). **Proceedings of the Fifth Workshop Conference.** Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, 365 pp.

Topics covered by the papers presented at the Conference include the uses and characteristics of tobacco such as tobacco genetics, leaf chemistry, nitrogenous compounds, culture and curing effects, disease control, and the use of maleic hydrazide. Many of the remaining papers are concerned with the effects of tobacco smoke constituents and cover such topics as the metabolic handling of nitrosoureas and nitrosocarbamates, biological effects of maleic hydrazide, the effect of smoke constituents on cardiovascular and respiratory systems, chronic exposure in primates, bioassay methods and results, and carcinogenicity. The role of tobacco and health in history is mentioned, as well as research for a less hazardous cigarette. Each paper includes a bibliography, and most contain an abstract of the study reported. There is extensive use of graphic and tabular data.

82–0059. Villiger, B.; Broekelmann, T.; Kelley, D.; Heymach, G. J., III; McDonald, J. A. **Bronchoalveolar Fibronectin in Smokers and Nonsmokers.** *American Review of Respiratory Disease* 124(5): 652-654, November 1981.

Bronchoalveolar lavage (BAL) fluid was analyzed for concentrations of fibronectin (FN). It was shown that smokers have significantly higher concentrations of FN (11.3 ± 3.9 μg/mg albumin) than nonsmokers (7.3 ± 2.9 μg/mg albumin). A segmental BAL was performed on 16 smokers (9 men and 7 women) and 13 nonsmokers (6 men and 7 women). The alveolar macrophages of smokers were found to secrete more FN in vitro than those of nonsmokers. This may indicate a higher metabolic turnover in the lower respiratory tract, and also a stimulatory effect of smoking.

See also, **82–0076**

MORTALITY AND MORBIDITY

82-0060. Anderson, A. E., Jr.; Foraker, A. G. **Smoking, Mortality, and Sex in a Community Hospital Necropsy Population.** *Southern Medical Journal* 74(9): 1097-1100, September 1981.

Analysis of 344 necropsies at a community hospital disclosed a male to female ratio of 1.9:1.0 and a smoking incidence over twice that of the general population. Overall longevity for men was less than for women. Intragroup comparisons showed an inverse relationship between smoking and longevity. Smokers in general and subsets of male and female smokers thus died earlier than respective nonsmokers. There were five diseases that accounted for 39 percent of the deaths of smokers in the sample: bronchogenic carcinoma, peptic ulcer, aortic aneurysm, acute myocardial infarction, and centrilobular emphysema. Trends for nonsmoking men and women were not different. However, smoking women surprisingly died earlier than smoking men at comparable pack-year exposure levels. Conversely, nonsmokers were most likely to achieve old age with freedom from diseases usually associated with smoking. It was concluded that smoking predisposes to hospitalization, with premature death. Men suffer the most, probably because of traditionally heavier usage. For reasons unclear, however, women who do smoke may exhibit excessive vulnerability in some situations. (Auth. Abs. Mod.)

82-0061. Gibinski, K.; Wistuba, J.; Lelek, E.; Klisinski, J.; Orzechowska, R.; Rys, M. **Poglebiona Analiza Ekonomiczno-Zdrowotnych Skutkow Palenia Tytoniu w Regionie Wielkoprzemyslowym. [Comprehensive Analysis of Economic and Health Consequences of Smoking in a Heavily Industrialized Region.]** *Polskie Archiwum Medycyny Wewnetrznej* 65(3): 197-201, 1981, Polish.

A 2-year survey of 10,712 workers, 58 percent of them smokers, representative of the working population of the Silesian Industrial District of Poland, revealed a 3.13 percent excess of hospitalization days among the smokers over nonsmokers, 14.4 percent more doctor visits by smokers, a 36 percent higher mortality rate related to smoking, and a 16 percent higher disability incidence among smokers. Approximate costs of these health effects of smoking and costs of production loss and losses due to fires caused by smokers were evaluated and compared with profits from cigarette sales (i.e., net gain of the Polish Tobacco Monopoly). The balance of losses and profits was found to be consistently negative for the national economy despite large yearly fluctuations in loss estimates. This finding appears to be independent of the social and economic systems, prices, and monetary policies. Further studies over the next several years are believed necessary to approximate more accurately the loss:profit ratio.

82-0062. Kestenbaum, H. **Tobacco and Health: An Introduction.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 235-239.

Twentieth century scientists were the first to study seriously the association between smoking and certain diseases. A British study in the 1950s, which examined the smoking habits and characteristics of over 34,000 physicians, found that smokers were 14 times more likely to develop cancer than nonsmokers, and that there was a dose—response relationship between smoking and lung cancer. Studies conducted at about the same time in the United States and Sweden reached similar conclusions. In 1962, the Royal College of Physicians of London released a report linking cigarette smoking with lung cancer and bronchitis and suggesting smoking as a probable contributor to the development of coronary heart disease. In 1964, the U.S. Surgeon General's report of more than 6,000 studies in the field concluded that cigarette smoking was a health hazard, associated with increased death rates, lung cancer in men, chronic bronchitis and emphysema, and higher death rates from coronary artery diseases in men. A 1979 update reviewed 30,000 studies and emphasized many of the earlier findings, although some scientists continue to disagree. Three areas of concern for the 1980s are: (1) the claim of some researchers that cigarettes can be made less harmful, which is disputed by those who insist that smoking even a less-hazardous cigarette can have adverse effects, especially if the smoker compensates by inhaling more deeply or more often; (2) the debate over whether "passive smoking" can produce lung impairment; and (3) the special occupational hazards of workers who smoke, such as those in the asbestos industry. Four main areas already addressed by legislative, judicial, or administrative actions in over 30 countries are: (1) prohibitions on cigarette advertising, (2) health warnings on cigarette packs, (3) restrictions on public smoking, and (4) public funds for antismoking education.

NEOPLASTIC DISEASES

82-0063. Anderson, W. H.; Bright, M.; Snider, H. L. **Relation of Smoking to Cardiopulmonary Disease.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 173-188.

The quantitative aspects of smoking, as determined in this study of 781 males and 154 females through questionnaires, puff characteristics, and tar and nicotine exposure of the airways (analysis of cigarette butts), produced a high degree of repeatability in the measurable phases. Data showed that patients with lung cancer have a different puff pattern than other groups and quantitatively more smoke exposure, although factors other than cigarette smoking determine which of the smoking-related diseases a smoker may acquire. Coal miners, who have only two-thirds the rate of lung cancer as that of the general population, smoke less on all 18 quantitative parameters measured, while females, who have less arteriosclerotic heart disease, chronic obstructive pulmonary disease, and lung cancer than males, have quantitatively less exposure to cigarette smoke than males because of the manner in which they smoke.

82-0064. Buckley, J. D.; Doll, R.; Harris, R.W.C.; Vessey, M. P.; Williams, P. T. **Case—Control Study of the Husbands of Women With Dysplasia or Carcinoma of the Cervix Uteri.** *Lancet* 2(8254): 1010-1014, November 7, 1981.

Smoking as a risk factor for cervical dysplasia and carcinoma was found to be independent of sexually related factors. In a case—control study to determine the association between cancer of the cervix and sexual activity of the women's husbands, 31 women with cervical abnormality and their husbands were matched with 62 control couples. It was found that sexual promiscuity by the husbands increased the risk of cancer in their wives. Smoking effects on the incidence of cervical abnormality were independent of the sexual risk factors.

82-0065. Davies, J. M.; Stevens, R. G.; Moolgavkar, S. H. **Estimation of Relative Risk From Vital Data: Smoking and Cancers of the Lung and Bladder. (Letters).** *Journal of the National Cancer Institute* 66(4): 611-613, April 1981.

The statistical data relating male and female lung and bladder cancer death rates to smoking, which was reported in an earlier paper (*Journal of the National Cancer Institute* 63: 1351-1357, 1979) are questioned, with possible explanations explored. In defense of their article, the authors state that since the objective had been to quantify the impact of cigarette consumption on total population mortality, the use of the average consumption for all ages was valid. The estimate of attributable risk of smoking for women is acknowledged to be too low, however, due to an error in computation; but the overall fit of the model is still considered valid for males. See also 80-0762.

82-0066. Dobre, V. **Der Widerwille gegen das Rauchen-ein Subjektiv-Paradoxales Symptom bei Zigarettenrauchern mit Zentralem Lungenkrebs. [Aversion Against Smoking—A Paradoxical Subjective Clinical Symptom of Pulmonary Cancer With Central Localization in Cigarette Smokers.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 156(3): 267-271, 1981, German.

A survey of the smoking habits of 79 male patients with centrally localized lung cancer was undertaken to evaluate the previously observed symptom of disgust with cigarette smoking by such patients and possible use of this symptom for early diagnosis of lung cancer. The questionnaires revealed that 68 patients had smoked, on the average, a total of over 200,000 cigarettes; 71 had smoked more than 20 cigarettes/day; and 8 patients averaged 15 to 20 cigarettes/day. Seventy patients (88.6 percent) acknowledged having quit or reduced smoking within the past 2 years, 52 of them within 2 to 5 months before the first medical consultation. Aversion to smoking was given by most of these patients as the reason for changing the smoking habit. It is concluded that the aversion to smoking is a fairly specific symptom of lung cancer, which may be used as an early indicator of a central bronchial carcinoma, particularly in men over 40 who smoke.

82-0067. Eastman, A.; Mossman, B. T.; Bresnick, E. **Formation and Removal of Benzo(*a*)pyrene Adducts of DNA in Hamster Tracheal Epithelial Cells.** *Cancer Research* 41(7): 2605-2610, July 1981.

Carcinogens can damage DNA, leading to possible mutation and transformation. Cells, however, have various processes for excising or circumventing such lesions. A cloned cell line derived from normal hamster tracheal epithelium was tested for its response to benzo(*a*)pyrene (BP), a carcinogen found in cigarette smoke. The hamster cells metabolized BP to reactive forms, as assayed by alkylation of DNA. Alkylation with radiotracer amounts of BP was maximum at 8 hours, at which time 70 percent of the applied hydrocarbon had been converted to water-soluble forms. At longer incubation times, the rate of removal of BP-DNA adducts exceeded the rate of formation. When BP-containing medium was replaced with fresh medium at 2 or 4 hours, a subsequent biphasic removal of adducts occurred, a rapid removal for the first 4-hour postincubation and then a slower repair. About 50 percent of the DNA-bound hydrocarbon remained in DNA after 48 hours. Cells were able to divide in the presence of these lesions, undergoing five doublings (5 days), while only 60 percent of the adducts were removed from the DNA. Integrity of DNA during this period was monitored by the alkaline elution technique. A toxic dose of BP was required to cause any increase in the rate of elution. Minimal single-strand breakage was observed from 2 to 8 hours during BP treatment, but at 15 hours DNA appeared normal. Comparison was made with a nontoxic dose of methyl methanesulfonate which caused very rapid elution of DNA after only 1 hour treatment. At least 15 deoxyribonucleoside-bound BP adducts were separated by high-pressure liquid chromatography. Four adducts, probably deoxyadenosine-BP, were removed almost completely in 24 hours, while the others appeared to be poorly removed. It was suggested that lesions which remain in DNA, instead of being repaired or removed, are more likely to have subsequent mutagenic effects on the cells. (Auth. Abs. Mod.)

82-0068. Gaafar, H. A.; Al-Mansour, A. H. **The Effect of Cigarette Smoke on the Vocal Cord Mucosa of the Rabbit.** *Journal of Laryngology and Otology* 95(7): 721-729, July 1981.

The effect of long-term cigarette smoke inhalation on the ultra-microscopic structure of rabbit vocal cord mucosa was examined. A group of adult male rabbits was exposed to cigarette smoke twice daily for 10 minutes, in batches of three or four, for a period of 3 months. Control animals were not exposed to cigarette smoke. The smoke-exposed rabbits showed variable degrees of hyperplasia, with disturbed stratification, in the epithelium covering the true vocal cords. The hyperplastic cells might have led to dysplasia with atypical changes and eventually to cancer. Damage was also found in the nerves and muscles in the tunica propria, especially in the neurotubules, neurofibrils, and muscle fibrils. If such changes are also caused in human vocal cords, they might help to explain the hoarseness and vocal fatigue associated with smoking. The results of the study strengthen the view that prolonged and heavy smoking is associated with an increased frequency of laryngeal carcinoma.

82-0069. Greenland, S. **Multivariate Estimation of Exposure-Specific Incidence From Case—Control Studies.** *Journal of Chronic Diseases* 34(9-10): 445-453, 1981.

A population based case—control study of smoking and bladder cancer serves to illustrate the use of multivariate methods which circumvent the drawbacks of the ordinary Bayes' theorem method used to estimate exposure-specific incidence from case—control studies. The methods described are the "smooth first," whereby one first smoothes the case—control data using a multivariate model and then applies the ordinary method to the smoothed data; "smooth last," whereby one derives exposure-specific estimates by the ordinary method and then smoothes the estimates using a multivariate model; and "pseudo-rate," fitting and adjusting a prospective model to the pseudo-rates computed as exposure-specific case—control ratios. The smoking vs. bladder cancer data involved males in age groups ranging from 50 to 79, including smokers and nonsmokers. The smooth-first method is used to remove a smoking-cancer anomaly wherein smoking appears to halve the cancer rate among 60- to 64-year-olds, and to at least double the rate among all other age groups. The smooth-last method is used for a slight age anomaly, wherein the increase in cancer incidence between the 55- to 59-year-old group and the 60- to 64-year-old group is almost negligible, whereas it would be expected to conform to the much steeper increase observed between the other age groups and in other bladder cancer incidence data. The pseudo-rate method is also generally discussed. When the exposure variables are all categorical, the pseudo-rate method using a first-order logistic model will give the same results as the smooth-first method using the log-linear model with all two-way interactions and all interactions of all orders between the exposure variables. The exposure variables are continuous.

82-0070. Infante, F.; Voirol, M.; Raymond, L.; Hollenweger, V.; Conti, M. C.; Loizeau, E. **Alcohol, Tobacco and Nutriments Consumption in Liver Cancer and Cirrhosis Patients in Geneva.** *Inserm* 95: 53-58, 1980.

In researching the etiology of liver cancer and cirrhosis, it was found that men with cirrhosis smoked significantly more than their controls, and cirrhotic women smoked five times more than their controls. The Geneva, Switzerland, study examined the nutrition, and alcohol and tobacco consumption of 195 patients (51 females and 144 males) with cirrhosis, liver cancer, or both. Alcohol consumption was very high among patients compared to the controls, but there was no significant difference in nutritional intake. It is noted that female patients were an average of 5 years younger than male patients at admission to the hospital. Though regular consumption of tobacco in males was only slightly higher than in controls, the total life consumption was far higher.

82-0071. Jacobsen, M. **Dust Exposure, Lung Diseases, and Coalminers' Mortality.** Doctoral Dissertation, University of Edinburgh, Great Britain, Institute of Occupational Medicine, 1976, 325 pp.

Relationships between dust exposure, lung diseases, and coal miners' mortality were studied using data relating to 31,000 miners who constituted more than 90 percent of the working populations in 24 collieries throughout the British coalfields. With the exception of lung cancer deaths, the highest mortality occurred among men who had symptoms of persistent cough and phlegm, and who complained of breathlessness. This was so both for cigarette smokers and nonsmokers. Cigarette smoking was associated with increasing prevalence of bronchitic symptoms (persistent cough and phlegm) and breathlessness among more than 11,000 of the coal miners. Mortality among cigarette smokers was 57 percent higher than among nonsmokers. The cigarette smokers had a nearly eightfold excess of lung cancer deaths; mortality attributed to bronchitis, to respiratory diseases in general, and to cardiovascular diseases also reflected the excess hazard—twice that of nonsmokers. (Auth. Abs. Mod.)

82-0072. Kandaswami, C.; O'Brien, P. J. **Pulmonary Metabolism of Benzo(*a*)pyrene: Effect of Asbestos.** *Biochemical Pharmacology* 30(7): 811-814, April 1981.

Cigarette smoke remains the main etiologic risk factor in pulmonary carcinoma, but exogenous agents such as environmental chemicals also appear to be involved. The risk of bronchogenic carcinoma is known to increase from occupational exposure to asbestos. Studies show that exposure to asbestos and to polycyclic aromatic hydrocarbons of the type present in cigarette smoke can have synergistic or cocarcinogenic effects. Asbestos workers who smoke have a higher risk of carcinoma than nonsmokers or smokers in the general population. Since asbestos is retained in the lungs, the carcinogen may adsorb to the asbestos particulate, thus retarding pulmonary clearance of the carcinogen and increasing its exposure level. The membrane-bound system of aryl hydrocarbon hydroxylase mediates the metabolism of carcinogenic hydrocarbons, such as benzo(*a*)pyrene. A study was conducted to determine if asbestos could affect aryl hydrocarbon hydroxylase activity of rat lung microsomes. Results indicated that asbestos treatment led to a marked diminution of aryl hydrocarbon hydroxylase activity, suggesting that asbestos could prevent benzo(*a*)pyrene from being metabolized in lung tissue.

82-0073. Korsgaard, R. **Chromosome Analysis of Malignant Human Effusions in Vivo.** *Scandinavian Journal of Respiratory Diseases* Supplementum No. 105, 100 pp., 1979.

Observations of abnormal chromosome content in neoplasmic cells have led to the concept of permanent DNA damage as an initiator of malignant tumors. In this study, chromosome and DNA analysis is explored as a possible diagnostic tool for carcinoma. Effluents from 267 patients suspected of having cancer were tested via standard cytological techniques and experimental cytogenetic methods; 200 were ultimately classified as malignant. The subjects ranged from 26 to 93 years in age; 123 male (108 smokers) and 77 female (49 smokers). The majority of the malignancies were of the pleural cavity. A significant overrepresentation of smokers occurred in the pulmonary squamous-cell group, but not in the group with adenocarcinoma. Even so, cigarette smoking was the common carcinogenic factor; of the nonpleural carcinomas, the smoker to nonsmoker ratio was 64 to 46. Chromosome analysis proved particularly useful in cases of effusions due to dissemination of adenocarcinomas and mesotheliomas, where cytology alone sometimes failed to give a definite diagnosis. In some cases, the cytogenetic method gave a positive diagnosis months or years before cytology confirmed the presence of neoplasm.

82-0074. Lee, P. N.; Castelli, W. P. **Filter Cigarettes and Heart Disease. (Letters).** *Lancet* 2(8247): 642, September 19, 1981.

An earlier article (*Lancet* 2(8238): 109-113, July 18, 1981) which found no sign that filter cigarettes of the 1960s and early 1970s gave any protection against coronary heart disease (CHD) is compared to a recent review (*Journal of Epidemiology and Community Health* 35(1): 16-22, March 1981) which concluded that filter cigarette users did have lower mortality than plain cigarette users for lung cancer and slightly lower for CHD. The review was based on study results from thousands of deaths, whereas the Framingham conclusions used in the earlier article were based on only 60 CHD deaths in smokers. It is suggested that more emphasis should have been placed on the limitations of the Framingham Study data and sample size. In response, Castelli, one of the authors of the *Lancet* article, notes that although the later review was able to estimate small effects with great confidence, the data were collected under less stringent control than in the Framingham Study; and the different conclusions reached were also affected by other factors. For example, subjects who switched to a filter cigarette may have been health conscious in other ways. It is noted that increasing the size of samples or findings does not necessarily make poor results significant. Even the review's best result, a 25 percent fall in lung cancer rates in filter smokers, still does not make for a safe cigarette. Emphasis might be better placed on quitting smoking. See also 81-1493 and 81-1074.

82-0075. Lemoine, J. M. **Tabagisme et Bronchoscopie. [Tobacco Smoking and Bronchoscopy.]** *Broncho-Pneumologie* 30(4): 247-263, 1980, French.

In a prospective study of 1,587 outpatients in Paris hospitals, bronchoscopic examination was carried out and the smoking habits of the patients were surveyed. Total cigarette consumption of the male patients (79 percent of 1,587) was inverse of that of female patients: 65 percent of all men vs. 12 percent of all women had consumed 150,000 or more cigarettes over a 20-year period. The incidence of bronchogenic carcinoma in male and female smokers of 150,000 to 300,000 cigarettes was 26.5 percent (male) and 12.5 percent (female). The incidence of chronic bronchitis for the same category of smokers was 25 percent for both men and women but increased more rapidly in women as total cigarette consumption rose. An endoscopic view of the bronchi in both male and female smokers of a minimum of 150,000 cigarettes was normal in only about 4 percent. It is estimated that 93.5 percent of all bronchogenic carcinomas in men may be detected before symptoms appear, if the preferential target population is limited to men of all ages who had consumed at least 150,000 cigarettes.

82-0076. Lubawy, W. C.; Isaac, R. S. **Immediate and Delayed Effects of Tobacco Smoke Components on Pulmonary ^{14}C Benzopyrene Metabolism.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 189-202.

Benzo(*a*)pyrene (BaP) is thought to be responsible for the high incidence of lung cancer in smokers; and since metabolic activation is required for BaP-induced carcinogenesis, the factors which affect its metabolism in pulmonary tissue need to be identified. In this study, isolated perfused rabbit lung was examined for the effects of whole smoke, the delayed effects of whole smoke condensate, and the gas phase of smoke on lung tissue. Male New Zealand rabbits, pretreated with either corn oil, 3-methylcholanthrene (3MC) or tobacco smoke condensate (TSC), were used. Results showed that the initial effect of whole smoke is inhibition of BaP metabolism, followed by the induction of aryl hydrocarbon hydroxylase (AHH) which caused a delayed increase of BaP metabolism. The gas phase alone of smoke produced little or no effect, so the materials responsible are probably in the particulate matter of smoke. The corn oil pretreated rabbits produced relatively few metabolites; the 3MC and TSC pretreatments showed increased BaP metabolism.

82–0077. Matanoski, G. M.; Elliott, E. A. **Bladder Cancer Epidemiology.** *Epidemiologic Reviews* 3: 203-229, 1981.

Epidemiological studies have demonstrated a two- to fourfold excess risk of bladder cancer in males specifically associated with cigarette use. From 39 to 56 percent of all bladder cancers in men are attributable to smoking, although the role of cigarettes in female bladder cancer is not clear. Smoking-associated mortality rate increases due to lung cancer in women have not been reflected in bladder cancer rates. Data suggest a long latency period of exposure before cancer appears; cessation of smoking had to occur up to 15 years before to reduce its effect as a risk factor. An increased risk with increased dose has also been demonstrated. In this review of epidemiological studies of bladder cancer, the other risk factors discussed include sugar substitutes, coffee, occupations, viruses, drug use, and other diseases.

82–0078. Miller, E. C.; Miller, J. A. **Mechanisms of Chemical Carcinogenesis.** *Cancer* 47(Supplement, 5): 1055-1064, March 1, 1981.

Chemicals appear to be of major importance in the induction of human cancers. Generally recognized carcinogenic chemicals from cigarette smoke may promote tumor formation in the lungs, urinary tract, and other sites, while exposure to betal nut and tobacco quid may promote tumors in the buccal mucosa. The most important of the known causes of cancer appear to be the carcinogens to which persons are exposed as a consequence of social habits, such as use of tobacco products. Chemical carcinogens include a wide range of structures. Their common feature is that their ultimate forms are electrophilic reactants; in most cases, these reactants arise through metabolism in vivo. Carcinogenesis by chemicals is a multistage process. The first stage, initiation, occurs rapidly and appears to be irreversible. The available data indicate that initiation generally results from one or more mutations of cellular DNA. Covalent reactions of electrophilic derivatives of carcinogens with DNA are the major cause of these mutations. The second stage, promotion, occurs over a longer period of time, and is a complex process, for which the early stages are largely reversible. The critical events appear to be epigenetic. Complete carcinogens have both initiating and promoting activities, but the ratios of these two activities for various chemicals may differ greatly. This knowledge of the mechanisms of carcinogens by chemicals provides a useful basis for approaches to the prevention of human cancer. (Auth. Abs. Mod.)

82–0079. Shaw, H. M.; Milton, G. W. **Smoking and the Development of Metastases From Malignant Melanoma.** *International Journal of Cancer* 28(2): 153-156, August 15, 1981.

Factors which may influence the carcinogenic effects of cigarette smoke include the duration of the individual's smoking habit, the amount of tobacco consumed daily, and whether the individual is a current or ex-smoker. Investigations on 2,583 malignant melanoma patients indicated that in both men and women, although there did not appear to be a causal relationship between cigarette smoking and melanoma, smoking did facilitate the spread of metastases. Significantly more ex-smokers or smokers than lifelong nonsmokers first presented for melanoma treatment with established metastases. Also, in patients with localized disease, metastases appeared within 10 years more often in ex-smokers than in lifelong nonsmokers. (Auth. Abs. Mod.)

82–0080. Stenback, F.; Peto, R.; Shubik, P. **Initiation and Promotion at Different Ages and Doses in 2200 Mice. III. Linear Extrapolation From High Doses May Underestimate Low-Dose Tumour Risks.** *British Journal of Cancer* 44(1): 24-34, July 1981.

The carcinogenic effects of 7,12-dimethylbenz(*a*)anthracene (DMBA), a component found in cigarette smoke, were investigated by treating the shaved backs of mice with initiating doses at 10, 30, 100, or 300 μg. Some hypotheses about cancer mechanisms predict a linear dose—response relationship, where there is a proportionality between dose and number of tumors. Others predict an upward curvature in the relationship (e.g., doubling the dose would more than double the response). A downward curvature would involve the change in dose being greater than the change in response. Among mice that experienced no initial carcinogenic promotion, only DMBA doses sufficient to cause ulceration with subsequent promotion due to wound healing caused a rapid crop of tumors, so the dose—response curve exhibited strong upward curvature. Among promoted animals, the response of the skin to initiation appeared to have been nearly saturated by all DMBA doses tested, so that a 30-fold decrease in dose produced only a 3-fold decrease in effect. The dose—response relationship thus exhibited strong downward curvature. Among promoted animals, estimation of the risks associated with very low doses of carcinogen by linear extrapolation through the origin from the effects of larger doses (which is often assumed to be conservative) would underestimate the true risks by 10-fold or more. Results emphasize that whereas linear interpolation from the results of high doses may be reasonable for data on the effects of continuous treatment with nontoxic dose levels of carcinogen, it may be misleading when extrapolating, as here, from the effects of single large doses. (Auth. Abs. Mod.)

82–0081. Sydnor, K. L. **Tumorigenicity of Tobacco Smoke Condensates.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 321-340.

The effects of the water-soluble fraction of tobacco smoke condensate in a strain of rats highly susceptible to established chemical carcinogens were examined. The aqueous extract was found to have cocarcinogenic properties which enhance the tumorigenic potency of low doses of benzo(*a*)pyrene (BaP) which is equipotent to 2.0-μg of BaP when administered to 30-day-old rats. Fractionation of tobacco smoke condensate provides a method for separating fractions of cigarette smoke condensate to identify toxic as well as carcinogenic fractions. The majority of the tumor-inducing property of the water-insoluble condensate is in Izard Fraction I and II. To date, Izard Fraction III has been shown to contain compounds which inhibit the carcinogenic response to BaP. Although considerably more research is needed to identify the principal constituents of smoke condensate which contribute to the carcinogenic response, progress has been made in identifying fractions which make minute contributions. (Auth. Abs. Mod.)

82–0082. Voirol, M.; Infante, F.; Raymond, L.; Hollenweger, V.; Conti, M. C.; Loizeau, E. **Alcohol, Food and Tobacco Consumption in Pancreatic Diseases.** *Inserm* 95: 59-62, 1980.

A nutritional study was begun in 1977 in Geneva, Switzerland, to investigate nutrition, alcohol, and tobacco consumption as factors in pancreatic cancer. To date, more than 800 digestive patients and 700 controls have been interviewed on their dietary habits. Only confirmed cases, diagnosed through biopsy or necropsy, are included in the study. The results show a higher (double) life alcohol and tobacco intake in the pancreatic patients over the controls. A more specific nutrient factor may also be related; the female cancer patients showed a lower protein, lipid, and glucide intake than controls.

82–0083. Wehner, A. P.; Dagle, G. E.; Milliman, E. M.; Phelps, D. W.; Carr, D. B.; Decker, J. R.; Filipy, R. E. **Inhalation Bioassay of Cigarette Smoke in Rats.** *Toxicology and Applied Pharmacology* 61(1): 1-17, October 1981.

Three groups of Fischer 344 rats were exposed to three types of cigarettes—code 13 (high tar, low nicotine), code 27 (low tar, medium nicotine), and code 32 (high tar, high nicotine) in smoking machines for up to 24 months. Two control groups, one sham treated, the other untreated, were also used. The rats were analyzed for weight gain, survival rate, and pulmonary interstitial reaction. The sham-exposed animals had significantly lower body weights than the untreated controls. The smoke-exposed animals had significantly lower weights than the sham-exposed controls; the weights were lower for the code 27 and code 32 animals than for the code 13 animals during the second year of exposure. The survival of the code 13 animals was similar to that for the sham-exposed and untreated control group; survival times of the code 27 and code 32 animals were shorter. Smoke-induced histopathologic lesions consisted primarily of pulmonary smoke granulomas; the smoke granulomas were less severe in the code 27 exposure group than in the groups exposed to smoke from code 13 or code 32 cigarettes. Additional changes included pulmonary alveolar epithelial hyperplasia, and squamous metaplasia and basal cell hyperplasia of laryngeal and tracheal epithelium. One primary epidermoid carcinoma was found in the lung of a code 27 rat. The rats tolerated the chronic exposures relatively well, and certain of the smoke-induced lesions allowed differentiation between the different types of cigarettes. (Auth. Abs. Mod.)

82–0084. Weiss, W. **Lung Cancer and Occupational Lung Disease.** *Clinics in Chest Medicine* 2(2): 289-300, May 1981.

In the development of lung cancer, smoking might be the primary factor in up to 88 percent of cases, according to this report of the relationship of smoking to cancer and occupational lung diseases. Studies have shown a definite smoking relationship with asbestos exposure, where the rate of cancer is increased dramatically only in those workers who also smoke. The smoking relationship with higher incidence in some other exposure studies cannot be verified, since smoking habit information was not used as a study factor, as in many studies on occupational exposure to chemicals. The five histologic types of lung cancer (squamous cell, adenocarcinoma, small cell, large cell, and mixed squamous cell and adenocarcinoma) are important etiologically and biologically in characterizing the tumor and in prognosis and therapy. The generally poor results of treatment are explained by an examination of tumor development, where clinical recognition is confined to approximately the last quarter of the tumor life cycle. The added effect of smoking appears to mask symptoms and delay identification of the cancer, and it appears that screening at-risk populations is also fruitless because of the biologic nature and natural development of lung neoplasms. Diagnostic procedures are reviewed and available therapy (including surgery) is discussed. It is concluded that since smoking has been identified as a prime factor in the development of lung neoplasms, it must be taken into consideration when evaluating any other causes of the disease.

82–0085. Witschi, H. P.; Hakkinen, P. J.; Kehrer, J. P. **Modification of Lung Tumor Development in A/J Mice.** *Toxicology* 21(1): 37-45, 1981.

Strain A mice were injected with urethan, 3-methylcholanthrene, or dimethylnitrosamine, three chemical carcinogens occurring in cigarette smoke. The mice were also given repeated injections of butylated hydroxytoluene (BHT). This treatment significantly increased multiplicity of lung tumors induced by all three carcinogens. Two other antioxidants, butylated hydroxyanisole or α-tocopherol (vitamin E), did not enhance tumor formation, nor did methylcyclopentadienyl manganese tricarbonyl, an agent capable of producing cell proliferation in lung. Lungs were more susceptible to the carcinogenic action of urethan 2 weeks following BHT-induced injury, but not during the phase of acute cell proliferation in lung. It is concluded that the effects of BHT on lung tumor development in mice are not related to its properties as an antioxidant or to its capability to produce extensive cell proliferation in lung. (Auth. Abs. Mod.)

82–0086. Woolner, L. B.; Fontana, R. S.; Sanderson, D. R.; Miller, W. E.; Muhm, J. R.; Taylor, W. F.; Uhlenhopp, M. A. **Mayo Lung Project. Evaluation of Lung Cancer Screening Through December 1979.** *Mayo Clinic Proceedings* 56(9): 544-555, September 1981.

Preliminary results of the Mayo Lung Project incidence study showed that squamous carcinoma was almost equaled in frequency by small cell carcinoma (30 percent and 26 percent, respectively). The Mayo Lung Project was conducted to determine whether screening of "high-risk" populations by means of periodic cytology examinations and thoracic roentgenograms would result in lower mortality from bronchogenic carcinoma than that found in a control population. The high-risk population screened consisted of 11,001 men, 45 years of age or older, who were chronic excessive smokers and were without known lung cancer. Prevalence screening, which began in November 1971 and was completed in July 1976, revealed 91 new or "prevalence" lung cancers. Of the 9,211 patients who had negative screening tests, "incidence" rescreening began in March 1972 and will continue until July 1982. The incidence patients were divided into a close surveillance group, due for rescreening every 4 months (n=4,618), and a control group, those advised to have a chest roentgenogram and sputum test at least once a year (n=4,593). As of December 31, 1979, there were 109 new incidence cases of lung cancer found in the close surveillance group and 78 in the controls. The findings of almost equal incidence of squamous and small cell carcinoma were considered surprising, since it had been thought that squamous cell bronchogenic carcinoma would predominate in a high-risk population of cigarette smoking males. An evaluation of the relative value of the two screening tests in the early detection of lung cancers showed that chest roentgenography far surpassed sputum cytology as a means of detection. Sputum cytology is highly effective in early detection of squamous cancer—50 percent of all early stage squamous cancers are first detected by this means. It is of little use, however, in the early detection of adenocarcinoma or large cell types. In small cell cancer, neither screening modality is of value. (Auth. Abs. Mod.)

NON—NEOPLASTIC RESPIRATORY DISEASES

82–0087. Bonini, S.; Toccaceli, F.; Dato, A.; Brostoff, J. **The Circadian Assessment of Peak Expiratory Flow as an Additional Tool for Adequate Treatment (and Prevention) of Bronchial Asthma.** In: Smolensky, M. H.; Reinberg, A.; McGovern, J. P. (Editors). *Recent Advances in the Chronobiology of Allergy and Immunology. Advances in the Biosciences, Volume 28.* New York, Pergamon Press, 1980, pp. 33-40.

Peak Expiratory Flow (PEF) was measured at 4-hour intervals, for at least 3 consecutive days in 79 asthmatic inpatients, 11 hay fever outpatients, and 30 healthy subjects of whom 15 were smokers. The circadian variation of PEF was expressed by an index, referred to as the Circadian Bronchial Lability Index (CBLI), where CBLI equals PEF daily maximum minus PEF daily minimum, divided by PEF daily maximum. It was found that healthy smokers had higher CBLI values than healthy controls, 11.5±8.3 vs. 8.4±4.0, respectively. Preventive aspects of respiratory health suggest special attention should be given to those people (such as healthy smokers) without clinical asthma and normal pulmonary function tests, but who do show abnormal bronchial lability. (Auth. Abs. Mod.)

82–0088. Carta, P.; Cocco, P. L.; Aru, G.; Barbieri, M. T.; Cocco, E.; Meloni, M.; Sanna-Randaccio, F.; Casula, D. **Smoking and Dust Effects Upon Lung Function of Healthy Workers.** *European Journal of Respiratory Diseases* 62(Supplement 113): 26-27, 1981.

The effect of dust exposure from metalliferous mines upon lung function was investigated in 207 healthy, nonsymptomatic workers, 135 face-miners and 72 white collar employees unexposed to dust. Both groups, comparable by age, were divided into never and current smokers. Lung function measurements included spirometry (VC, FEV_1, RV), effort-independent flows (MMEF, $MEF_{50,75}$), and closing volume measurements by the bolus method (CC percent TLC). The dependence of each lung function test upon height, age, smoking (pack-years), and a "dust index" was examined by multiple regression analysis. Smoking and dust measurements influenced more the loss of the MMEF and the maximal flows than that of the FEV_1, with the smoking effect being more pronounced. Similar results were found for the CC percent TLC measurement. Results suggest that the health hazards of dust exposure and smoking have a summatory effect upon lung function, with both having an early effect upon small airways. In spite of their high variability, measurements for examining the small airways seem to be among the more sensitive methods for investigating lung function.

82–0089. Connellan, S. J.; Carson, R.; Holland, F.; Joyce, H.; Pride, N. B. **Role of Bronchial Hyperreactivity and Atopy in**

Enhancing Decline of Lung Function in Male Smokers. *European Journal of Respiratory Diseases* 62(Supplement 113): 130-131, 1981.

Male smokers with a declining forced expiratory volume (FEV_1) of at least 25 percent over 20 years were found to be more reactive to inhaled histamines. This supports the "Dutch Hypothesis" of atopic predisposition to development of severe chronic air flow obstruction in smokers. A family history of allergy, and prick skin test results to four common allergens in these smokers also supported the hypothesis. However, this theory does not explain the predominance of men and the social gradient of bronchitis.

82–0090. Da Silva, A.M.T.; Hamosh, P. **Airways Response to Inhaled Tobacco Smoke: Time Course, Dose Dependence and Effect of Volume History.** *Respiration* 41(2): 96-105, 1981.

The effect of smoking three brands of cigarettes with different composition on airways resistance (Raw), thoracic gas volume (TGV), and the maximal expiratory flow volume (MEFV) curve was examined. Raw increased significantly with all three brands after one puff. The maximum effected was reached after three puffs. Instantaneous flow at 50 percent of vital capacity (FEF_{50}) decreased significantly with cigarettes high in nicotine content, but not so after smoking a low-nicotine (0.31 mg) cigarette. Instantaneous flow at 75 percent of vital capacity out (FEF_{75}) increased significantly 30 min after the low-nicotine cigarette was smoked. A deep inspiration prior to Raw determination reduced by approximately one-third the bronchoconstrictor effect of cigarette smoke. All effects were reversible within 30 min, except the delayed effect of the low-nicotine cigarette on the FEF_{75}. It was concluded that the probable site of action of tobacco smoke is in the large and central airways. The bronchoconstrictor effect rapidly reaches a plateau. A delayed bronchodilation of the small airways observed after smoking the low-nicotine cigarette might represent a response usually masked by other long-acting components in smoke. It was suggested that the airway response to tobacco smoke is complex and probably a result of several components present in smoke which still have to be identified. (Auth. Abs.)

82–0091. Dahms, T. E.; Bolin, J. F.; Slavin, R. G. **Passive Smoking: Effects on Bronchial Asthma.** *Chest* 80(5): 530-534, November 1981.

A relationship between passive smoke exposure and pulmonary changes in asthmatics (reduced function of small airways of the lung) was established in a study of 10 patients with bronchial asthma and 10 control subjects exposed to sidestream cigarette smoke for 1 hour. All subjects showed the same increase in carboxyhemoglobin as a result of the exposure: 0.40 percent. The asthmatic group demonstrated a significant linear decrease in pulmonary function during this exposure. After 1 hour of smoke, the forced expiratory volume after 1 second (FEV_1) decreased 21.4 percent, the mean forced expiratory flow (FEF25-75 percent) during the middle half of the forced vital capacity (FVC) decreased 19.2 percent, and FVC decreased 20.0 percent in the asthmatic patients. These alterations were readily reversible when given inhalations of metaproterenol following the exposure. The control subjects showed no change in pulmonary function when similarly exposed. It was concluded that smoke particulates might be the primary stimulus for the response of asthmatic subjects, who probably have increased sensitivity in bronchiolar receptors, and may also have a greater number of particles deposited on the hypersensitive airways. The gaseous phase of cigarette smoke may also have been a causative agent, as well as emotional responses that may have increased with exposure, affecting pulmonary function. (Auth. Abs. Mod.)

82–0092. DeLucia, A. J.; Bryant, L. R. **Chronic Tobacco Smoke Exposure in Primates.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 259-283.

Male stumptail macaques were exposed to cigarette smoke for 2 to 6 years, with dosages equivalent to human consumption of one pack/day (low-dose) approximated by measurement of inhaled smoke volumes and blood carboxyhemoglobin levels after smoke exposure. Pulmonary function testing, hematological studies, and alveolar macrophage (PAM) biochemical assays were employed to assess health effects of chronic smoke exposure in the animals. Multiple breath washout measurements, as well as assessment of airway resistance and pulmonary compliance, did not differ significantly in smokers and nonsmokers. Repeated measurements of hemoglobin, hematocrit, and red blood cell counts in these animals discerned a mild 5-10 percent polycythemia in smoke-exposed animals, similar to findings in healthy humans and a variety of animal species exposed to cigarette smoke or carbon monoxide. Decreased levels of 2,3-diphosphoglycerate in the blood of smokers suggest possible impairment of O_2 delivery to tissues, since this organic phosphate is an important modulator of O_2 hemoglobin affinity and normally increases with hypoxemia. Alveolar macrophage recovery by bronchopulmonary lavage produced higher cell yields from smoke-exposed animals. Levels of glutathione, a dominant cellular reducing substance, and maximal enzyme activities of the pentose phosphate pathway showed increases suggestive of PAM activation and possible improved scavenging of reactive oxidant species encountered in smoke. (Auth. Abs.)

82–0093. Diamond, L.; Sabo, J. **Inhalation Toxicology of Nitrogen Oxides: Relevance to Tobacco and Health.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 203-211.

The toxic pulmonary effects of nitrogen oxides in the gas phase of cigarette smoke, particularly nitric oxide and nitrogen dioxide are examined. When smoked under standard conditions, U.S. commercial cigarettes yield 5.0 to 800 mcg nitrogen oxide per cigarette. While fresh mainstream smoke does not contain significant quantities of nitrogen dioxide, upon aging the smoke's nitric oxide is oxidized to nitrogen dioxide. About 10 percent of the nitric oxide retained in the smoker's lungs may be converted to nitrogen dioxide. Nitrogen dioxide is a strong oxidizing agent and may damage unsaturated lipids in biological membranes, and can also react with water to form equal molar amounts of potentially harmful nitrous acid and nitric acid. Nitrous acid can ionize to release the nitrite ion, which can react with secondary and tertiary amines to form N-nitrosamines, some of which are carcinogenic. The nitrite ion is also able to react with hemoglobin to form methemoglobin, reducing the oxygen-carrying capacity of the blood. Nitrogen dioxide is more acutely toxic than nitric oxide on a mole for mole basis. The toxic effects of nitrogen dioxide are dose-dependent, with concentration being more important than duration of exposure. There is substantial variation in species susceptibility. Nitrogen dioxide damages the terminal and respiratory bronchioles and the proximal portions of the alveolar ducts. Pulmonary pathologies associated with nitrogen dioxide include increased infectivity, increased airway resistance and airway responsiveness, emphysema, and carcinoma. It is not yet known if synergistic effects occur between nitrogen oxides and the particulate phase of whole tobacco smoke.

82–0094. Ellis, D. A.; Thornley, P. E.; Wightman, A. J.; Walker, M.; Chalmers, J.; Crofton, J. W. **Present Outlook in Bronchiectasis: Clinical and Social Study and Review of Factors Influencing Prognosis.** *Thorax* 36(9): 659-664, September 1981.

In a study of 116 patients (35 smokers) with proven bronchiectasis of at least 5 years duration, which had been treated either surgically or medically, the proportion of smokers was similar in each category (mild, moderate, or severe). There were more ex-smokers in the severe group. Survivors (94) tended to improve under either treatment (only 11 percent became worse), but smokers showed a sharper decline in FEV_2. At the time of the review, only 20 patients were still smoking; but smoking was associated with a significant trend toward a deterioration in the severity of their condition (6 were worse while 3 had improved). Of the survivors, 77 percent had a good work record, 50 percent reported no social problems, and only 7 percent were severely disabled. It is concluded that while the worst effects of bronchiectasis can be mitigated by modern medical and surgical treatment, a minority of patients will still have severe physical and social problems. It is strongly recommended that patients with bronchiectasis be advised not to smoke.

82–0095. Gilman, M. J.; Sylvester, J. T.; Kennedy, T. P.; Menkes, H. A.; Traystman, R. J. **Vascular Effects of Cigarette Smoke in Isolated Pig Lungs.** *American Review of Respiratory Disease* 124(5): 549-553, November 1981.

Since the effects of cigarette smoke on human pulmonary vessels have not been established, pulmonary artery pressure was measured in pig lungs before and after exposure to cigarette smoke to determine the local effects of cigarette smoke on the pulmonary vasculature. During each of 4 exposures, smoke was administered into the trachea for 3 to 4 min at a rate of 20 to 25 puffs/min (puff volume of 35 to 50 ml) with a smoking machine. During hypoxia when baseline vasomotor tone was high, cigarette smoke caused an acute transient vasodilation; while during control, when baseline tone was low, no significant effect was observed. Cigarette smoke also caused a depression of the pulmonary pressor response to hypoxia, which developed gradually during the experiment. Further experiments were conducted to determine whether the pulmonary vascular effects of smoking were mediated by synthesis and release of prostaglandins. Indomethacin, at perfusate concentrations of 20 and 100 μg/ml, did not significantly alter the acute vasodilating effect of smoking, suggesting that prostaglandins synthesized by cyclooxygenase were not the mediators of this response. Indomethacin did, however, prevent the gradual depression of the pulmonary vasoconstrictor response to hypoxia. If the observed responses also occur in humans, they could be deleterious, and would underscore the need for a greater understanding of the pulmonary vascular effects of cigarette smoke. (Auth. Abs. Mod.)

82–0096. Godard, P.; Clot, J.; Jonquet, O.; Bousquet, J.; Michel, F. B. **Lymphocyte Subpopulations in Bronchoalveolar Lavages of Patients With Sarcoidosis and Hypersensitivity Pneumonitis.** *Chest* 80(4): 447-452, October 1981.

Sarcoidosis and hypersensitivity pneumonitis (HP), two granulomatous pulmonary diseases, were compared in a bronchoalveolar lavage study of 16 patients with sarcoidosis, 6 with HP, and 20 controls (10 healthy nonsmokers and 10 smokers with chronic bronchitis). Lymphocyte subpopulations evaluated by four membrane workers were: (1) the total cell count (in millions recovered per 100 ml lavage) was 168.5±5.1 for HP patients, 24.3±3.5 for those with sarcoidosis, 27.4±4.1 percent for nonsmokers, and 60.5±3.2 for smokers; (2) the proportion of lymphocytes was higher in patients with HP (68.9±4.8 percent) than in those with sarcoidosis (43±5.1 percent), in healthy nonsmokers (18.6±1.3 percent), or in the smokers (5.3±2.1 percent); (3) there were only a small number of B cells and of Fc-receptor bearing cells; and (4) the lymphocytes identified by these techniques were mainly T cells in HP and sarcoidosis, 76.8±3.6 percent and 68.4±3.5 percent, respectively. For nonsmokers and smokers the respective figures were 28.1±3.0 percent and 34.5±4.6 percent. The proportion of "active" E-rosette-forming cells was 9.8±1.5 percent for HP, 28.1±2.5 percent for sarcoidosis, 3.9±1.3 percent for nonsmokers, and 2.3±1.4 percent for smokers. (Auth. Abs. Mod.)

82–0097. Gruchow, H. W.; Hoffmann, R. G.; Marx, J. J., Jr.; Emanuel, D. A.; Rimm, A. A. **Precipitating Antibodies to**

Farmer's Lung Antigens in a Wisconsin Farming Population. *American Review of Respiratory Disease* 124(4): 411-415, October 1981.

The prevalence of Farmer's Lung Disease (FLD) and of antibodies to FLD antigens were measured among 1,444 adults on 632 farms in north central Wisconsin. The disease is a hypersensitivity pneumonitis usually occurring among agricultural workers exposed to moldy hay. Questionnaires elicited information about symptoms as well as personal characteristics, such as smoking habits. Blood samples were obtained for antibody determinations. FLD prevalence was 4.2 percent per 1,000 workers. Only 15 percent of the study population were current regular smokers, and over half (57 percent) reported never having smoked cigarettes. There was a higher prevalence of FLD among the never smokers than among the former or current smokers, although the differences were not considered significant because of the small number of cases. Several actinomycete molds are associated with FLD. Tests showed the prevalence of antibodies to *Micropolyspora faeni* was eight times higher among never smokers and six times higher among former smokers than among current smokers. Current smokers also had a lower prevalence of antibodies to *Aspergillus fumigatus*, a fungus associated with hypersensitivity pneumonitis. Three theories were suggested to account for the differences in prevalence: (1) cigarette smoking may offer a protective effect against FLD; (2) there may have been a self-selection for never smoking among those who eventually develop FLD; and (3) smokers may have had prior morbidity, which removed them from eligibility for the study.

82-0098. Heffner, J. E.; Sahn, S. A. **Salicylate-Induced Pulmonary Edema.** *Annals of Internal Medicine* 95(4): 405-409, October 1981.

Patients with salicylate intoxication were found to be at increased risk for salicylate-induced pulmonary edema if they were older, ingested salicylates chronically, and had a history of smoking. To assess the course and prognosis of salicylate-induced pulmonary edema, the records of 36 consecutive patients 16 years of age and older, admitted with serum salicylate levels greater than 30 mg/dL, were reviewed. Pulmonary edema developed in 8 patients, all of whom smoked, and pulmonary infiltrates were never seen in 28 patients, 8 of whom smoked. The fact that all 8 patients who developed pulmonary edema were smokers suggests that preexisting pulmonary disease may promote salicylate pulmonary edema. Smoking may damage the respiratory epithelium, lowering resistance to fluid conductance in the alveolus and promoting pulmonary edema in smokers who become salicylate toxic. (Auth. Abs. Mod.)

82-0099. Kawakami, Y.; Irie, T.; Kishi, F.; Asanuma, Y.; Shida, A.; Yoshikawa, T.; Kamishima, K.; Hasegawa, H.; Murao, M. **Familial Aggregation of Abnormal Ventilatory Control and Pulmonary Function in Chronic Obstructive Pulmonary Disease.** *European Journal of Respiratory Diseases* 62(1): 56-64, February 1981.

Chronic obstructive pulmonary disease (COPD) tends to aggregate in some families. A study of pulmonary function and ventilatory control was performed on three groups of subjects, including measurements of smoking indices. Groups A and B were the sons of patients with COPD. Group A consisted of 14 sons of the patients with normal blood gases, and group B comprised 12 sons of the patients with hypoxemia and hypercapnia. Group C consisted of 26 controls. The smoking index was arrived at by multiplying cigarettes smoked/day by years of smoking. The smoking index in group A was 300±237 and in group B 330±272, both significantly higher than group C at 130±127 ($p<0.05$). The effects of smoking were therefore examined separately from other pulmonary factors. An abnormal value was counted as one point for each pulmonary function parameter tested; and the total formed an abnormality score, which was then plotted against the smoking index for each subject with a smoking index less than 400, the highest value in group C. Although the smoking indices were found to be similar in normal controls and sons, the abnormality score of pulmonary function was significantly higher in sons than in normals. Since it is possible that in sons of COPD patients, other factors were associated with pulmonary function abnormalities, smoking may have had little to do with the pulmonary function abnormalities seen in the groups.

82-0100. Krasniqi, A.; Hadergjonaj, Sh.; Mandura, I.; Haxhiu, M. A. **The Serum Concentration of AAT in Normal Subjects in Relation to Sex, Age, and Smoking Habits.** *Jugoslavica Physiologica et Pharmacologica Acta* 15(3): 327-329, September 1979.

Analyses of serum samples of 482 normal subjects aged 18 to 60 years revealed no significant differences in serum concentrations of alpha-1-antitrypsis (AAT) in heavy smokers ($n=59$) when compared to nonsmokers ($n=86$). A deficiency of AAT has been associated with chronic obstructive pulmonary disease. An investigation was made of the normal values of AAT and the effect of sex, age, and smoking on the serum AAT concentration. Results show the average concentration of AAT in males as 403±92 ($n=449$) and in females as 436±81 ($n=33$). The serum concentration of AAT found in nonsmokers averaged 393±74 and in heavy smokers was 391±75. No age-related differences were found.

82-0101. Kujala, P. **Smoking, Respiratory Symptoms and Ventilatory Capacity in Young Men.** *European Journal of Respiratory Diseases* 62(Supplement 114): 1-55, 1981.

Finnish conscripts ($n=1{,}092$) near the end of their military service were examined to determine the relationship between smoking and ventilatory capacity in young men and to study the associations between smoking and chronic bronchitis, respiratory symptoms, and physical working capacity. The men (aged 17 to 29 years) were classified as nonsmokers (28 percent), ex-smokers (9 percent), and smokers (63 percent). A questionnaire on respiratory symptoms was completed, and lung function tests and 12-minute running tests were performed. Results showed that even in young men, smoking is associated with a reduction in ventilatory capacity, particularly in tall men. It was also found to increase respiratory symptoms and impair physical working capacity, as measured in the 12-minute running test.

82–0102. Laviolette, M.; Chang, J.; Newcombe, D. S. **Human Alveolar Macrophages: A Lesion in Arachidonic Acid Metabolism in Cigarette Smokers.** *American Review of Respiratory Diseases* 124(4): 397-401, October 1981.

Pulmonary alveolar macrophages (PAM) synthesize and release arachidonic acid metabolites that help regulate smooth muscle responses, cellular immune responses, and neutral protease production. A study to determine the effects of cigarette smoking on the functions or capacity of PAM is described. PAM recovered from young cigarette smokers and from age- and sex-matched subjects who had never smoked were used to investigate arachidonic acid metabolism. The uptake of radiolabeled arachidonic acid by PAM obtained from smokers and nonsmokers was not significantly different. The phagocytic indexes of the smoker and nonsmoker macrophages were 17.4±3.9 percent and 18.5±5.0 percent, respectively. Both smoker and nonsmoker macrophages produced prostaglandin, E_2 (PGE_2), thromboxane B_2 (TXB_2), and prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$). A significant decrease in PGE_2 and TXB_2 synthesis but not in $PGF_{2\alpha}$ synthesis by the smoker PAM compared with the nonsmoker PAM was observed using two different assays to measure prostaglandin production. Nonsmoker macrophages produced 644±128, 239±53, and 29±12 ng/1.5 x 10^6 cells of PGE_2, TXB_2, and $PGF_{2\alpha}$, respectively, whereas smoker macrophages synthesized 168±20, 41±3, and 17±1 ng/1.5 x 10^6 cells, as measured by radioimmunoassay. Similar differences were observed using isotonic assays. A cigarette smoke-induced lesion in phospholipid hydrolysis or the mechanism regulating phospholipid hydrolysis seems most consistent with these findings. The demonstrated in vitro defect in cigarette smoker macrophage arachidonic acid metabolism may play an important part in the pathogenesis of cigarette-smoke-induced diseases. (Auth. Abs. Mod.)

82–0103. Lewis, D. J.; Jakins, P. R. **Effect of Tobacco Smoke Exposure on Rat Tracheal Submucosal Glands: An Ultrastructural Study.** *Thorax* 36(8): 622-624, August 1981.

Tobacco smoke exposure is known to produce hypertrophy of, and hypersecretion by, the submucosal glands in rats. The ultrastructural appearance of rat tracheal submucosal glands after exposure to tobacco smoke for up to 2 years was examined. Within the mucous cells many of the rough endoplasmic reticulum cisternae were grossly dilated with an accumulation of amorphous, electronlucent material. The Golgi zones were prominent, and the secretion granules often contained dense cores and appeared to have coalesced. Histochemically, increased amounts of sulphated mucus were present in exposed rats. Serous, ciliated, and myoepithelial cells were unaffected by smoke exposure. The ultrastructural changes observed in the mucous cells of the present study occurred in those organelles which are believed to be involved in mucus synthesis, and which have also been considered possible sites at which exogenous compounds could affect mucus production. (Auth. Abs. Mod.)

82–0104. Meyer, W.; Burkhardt, A.; Klenke, B.; Vogts, H.; Wilke, A.; von Wichert, P. **Lung Phospholipid Metabolism After Smoke Exposure in Rabbits.** *Progress in Respiration Research* 15: 141-147, 1981.

The normal structure and function of lung alveoli depends in part on an intact surface-tension-reducing surfactant. A study of the influence of cigarette smoke on the biosynthesis of phosphatidylcholine in rabbit lung to establish smoking's influence on surfactant metabolism is described. The two metabolic alterations investigated were the ability of lung slices to incorporate fatty acids and choline into phosphatidylcholine, and changes in the amount and composition of phosphatidylcholine in lung tissue. Rabbits of both sexes with weights between 2.8 and 3.5 kg were used. The cigarettes, containing 14 mg condensate and 0.8 mg nicotine, were administered by a smoking machine at two exposures of 0.5 hours/day. At 1, 2, and 4 weeks, the rabbits were sacrificed and their lungs investigated. Effects of cigarette smoke observed were: a constant decrease in percent phosphatidylcholine of total phospholipids and a corresponding increase in the other noncholine-containing phospholipids; and the quality of phosphatidylcholine declined as a surface-active material due to a significant decrease in palmitate content, although the decrease was compensated for by a corresponding increase in total unsaturated fatty acids, especially palmitoleate and linoleate. Palmitate is needed for remodeling steps of *de novo* synthesized phosphatidylcholine to form the disaturated species. Impaired phosphatidylcholine biosynthesis may help explain the decrease of lung surfactant activity often observed in chronic smokers.

82–0105. Patrick, J. M. **Smoking, Coal, Asbestos, and the Lungs. (Letter).** *British Medical Journal* 283(6292): 675, September 5, 1981.

An earlier article analyzing the contributions of smoking and of occupational dust exposure to airways disease (*British Medical Journal* 283(6289): 457-458, August 15, 1981) is seen as presenting two lessons: (1) society and individuals should weigh the benefits of any industrial process against the costs of its associated mortality and morbidity; and (2) the smoking habit places a further burden on the lungs of those individuals whose lung function has already been adversely affected by exposure to dust. In Britain, occupational dust exposure is being reduced by controls, while smoking levels remain high. The opposite is presently true in tropical Africa. It may be possible to collect valuable data about chronic lung disease epidemiology from populations with a different mix of predisposing factors before smoking reaches higher levels and redresses the balance. See also 81-1762.

82–0106: Prevost, R. J.; Smolensky, M. H.; Reinberg, A.; Raymer, W. J.; McGovern, J. P. **Circadian Rhythm of Respiratory Distress in Asthmatic, Bronchitic and Emphysemic Patients.** In: Smolensky, M. H.; Reinberg, A.; McGovern, J. P. (Editors). *Recent Advances in the Chronobiology of Allergy and Immunology. Advances in the Biosciences. Volume 28.* Pergamon Press, 1980, pp. 237-250.

During a 112-day Houston (Texas) Area Oxidant Study, respiratory distress (defined as either episodes of coughing, coughing up phlegm, wheezing, whistling in the chest and/or shortness of breath) was found to be circadian rhythmic. Highest incidence occurred between 0600 and 1200 in the day-active asthmatic, bronchitic, or emphysemic participants. The described temporal susceptibility pattern for respiratory distress did not vary between participants of different ages, sexes, educational status, income level, or place of residence (with respect to level of air quality). Nor did the temporal pattern vary between asthmatics, bronchitics, or emphysemics. Furthermore, smoking did not alter the pattern. On the average, females suffered 1.4 as many distress episodes as did males. Participants greater than 50 years of age suffered twice as many respiratory complaints as those younger than 18 years of age. Moreover, smokers, on the average, suffered twice as often as nonsmokers. Of pertinence, also, was the finding that participants residing in areas of relatively elevated levels of air pollutants averaged more than 1-1/3 as many episodes as those residing in areas of relatively better air quality. (Auth. Abs.)

82–0107. Pritchard, J. N.; Black, A.; Foord, N.; Walsh, M. **A Comparison of the Regional Deposition of Monodisperse Polystyrene Aerosols in the Respiratory Tracts of Healthy Male Smokers and Non-Smokers.** *Journal of Aerosol Science* 12(3): 214, 1981.

Groups of 16 healthy male smokers and nonsmokers were compared for regional deposition of monodisperse polystyrene aerosols in the respiratory tract. All volunteers showed normal results in a variety of lung function tests. The normal range of deposition was defined by using the results of experiments where at least five nonsmokers had participated. To compare smokers and nonsmokers, each individual was compared with the mean for a particular experiment and a list was made of the number of standard deviations from the mean in each experiment. The probability of the subject's being that far from the mean was then calculated. Analysis indicated that two nonsmokers and five smokers differed significantly from the nonsmoking mean at the 5 percent level. Finding 2 nonsmokers out of a group of 16 differing in this way was not significant at the 5 percent level, but the occurrence of 5 smokers from 16 by chance was less than 0.2 percent. No correlation was found between subjects excluded at the 5 percent level with smoking history or lung function test data. Differences may have been due to small airway obstruction, suggesting that this method may, in the future, be adapted for the early diagnosis of respiratory disorders.

82–0108. Sardi, F.; Kovacs, S. **Changes in the Lung Function With Age, Duration of Dust Exposure, Smoking, and Silicosis in Miners.** *European Journal of Respiratory Diseases* 62(Supplement 113): 63-64, 1981.

Coal miners in southwest Hungary were examined to determine the effect on lung function of smoking and duration of dust exposure. Spirometric measurements were made in nonsilicotic (n = 1,278) and silicotic (n = 890) miners. Body plethysmography was carried out on nonsilicotic subjects (n = 476), silicotic (n = 679), nonsmokers (n = 309), and smokers (n = 846), as well as controls (n = 129). Flow-volume curves were analyzed, and single-breath diffusing capacity was measured. Smokers were found to have a higher intrathoracic gas volume (IGV) than nonsmokers. The higher IGV may indicate that smoking promotes the development of emphysema in miners, although emphysema can occur to a substantial degree in nonsmoking coal miners. The increase of residual volume with age was greater in smokers than in nonsmokers, and smokers also experienced a greater decrease of specific conductance with age. Prolonged dust inhalation appears to impair lung function, even if silicosis is not present and even in those who never smoked.

82–0109. Weiss, von Th.; Dorow, P.; Felix, R. **Inhalationsszintigraphische Befunde bei Small Airways Disease. [Radioaerosol Lung Imaging in Small Airways Disease.]** *Fortschritte auf dem Gebiete der Rontgenstrahlen und der Nuklearmedizin* 134(6): 629-633, June 1981, German.

Possible use of the aerosol lung imaging technique for early diagnosis of small airways disease was explored in 35 asymptomatic smokers, average age 36.3 years, with peripheral airways obstruction evidenced by abnormal small-airways parameters. The radioaerosol lung patterns of these patients were analyzed comparatively with those of 50 patients with chronic obstructive lung disease (COLD), average age 52.2 years, and 20 healthy nonsmokers, average age 41.3 years. The analysis consisted of qualitative inspection of polaroid images and determination of the aerosol distribution index (ADI) that describes density variations of different aerosol particles over the five pulmonary regions of interest. Visual comparison of images revealed a highly inhomogeneous aerosol distribution in COLD patients and a similar, but covering only a part of lung area, distribution pattern in patients with small airways disease. This characteristic aerosol pattern was observed in 71 percent of the patients with small airways disease. The ADI of these patients had an intermediate value between the low values of the healthy nonsmokers and the high values of the COLD patients.

See also, **82–0059, 82–0071, 82–0157**

CARDIOVASCULAR DISEASES

82–0110. Beaglehole, R.; Hay, D. R.; Foster, F. H.; Sharpe, D. N. **Trends in Coronary Heart Disease Mortality and Associated Risk Factors in New Zealand.** *New Zealand Medical Journal* 93(685): 371-375, June 10, 1981.

Between 1968 and 1978 there was a decline in coronary heart disease (CHD) mortality among New Zealand European men (17 percent) and women (14 percent), occurring in all age groups and in Maoris. One factor associated with the decrease is a leveling off of cigarette smoking. A survey of smoking habits based on the 1976 census found that 38 percent of men and 31 percent of women smoked cigarettes regularly. Among those aged 15 and over, 22 percent of men and 11 percent of women stated that they had given up smoking. Data from the Department of Statistics indicated that the number of cigarettes released for sale decreased from 2,890 per annum per person aged over 15 years to 2,720 per person in 1979, a decline of 5.9 percent. Tobacco sales (excluding tailor-made cigarettes) fell from 1.45 kg per annum per person aged over 15 years to 0.26 kg in 1979. In 1963, 37 percent of male doctors reported smoking regularly compared to 20 percent of male and 17 percent of female doctors in 1976. Socioeconomic differences also affected the frequency of smoking: in 1976, 48.6 percent of production and related workers, transport equipment operators, and laborers smoked compared to 27 percent of professional, technical, and related workers.

82–0111. Buda, A. J.; Macdonald, I. L.; Anderson, M. J.; Strauss, H. D.; David, T. E.; Berman, N. D. **Long-Term Results Following Coronary Bypass Operation.** *Journal of Thoracic and Cardiovascular Surgery* 82(3): 383-390, September 1981.

A study, conducted on a group of 102 patients who underwent aorta-coronary bypass grafting between 1969 and 1971, is described. There was a minimum followup of 7 years. Smoking appeared to be the largest single risk factor related to the success of the operation and survival of the patient. The other measured risk factors were hypertension, diabetes, hyperlipidemia, and positive family history. Preoperative predictors of clinical status at 5 years indicated that of the 35 asymptomatic patients, a total of 18 (or 51 percent) were smokers. Of the 48 symptomatic patients, no less than 36 were smokers (75 percent). Preoperative predictors of survival at 5 years revealed that of the 68 survivors, 43 were smokers (63 percent), while of the 25 nonsurvivors, 14 were smokers (56 percent). Smoking thus appears to decrease the chances for successful coronary bypass operations.

82–0112. Burch, P.R.J.; Seltzer, C. C.; Friedman, G. D.; Petitti, D. B.; Bawol, R. D. **Mortality in Cigarette Smokers and Quitters. (Letters).** *New England Journal of Medicine* 305(15): 889-890, October 8, 1981.

An earlier article (*New England Journal of Medicine* 304(23): 1407-1410, June 4, 1981) which had concluded that quitting smoking reduces mortality from coronary heart disease (CHD) is criticized. The original article had apparently adjusted death rates in quitters and persistent smokers by the direct method for age, sex, and race, while using a multivariate analysis to correct for other variables. Burch faults the article for: determining "relative risk" for the entire study population rather than for self-selected subgroups such as quitters and persistent smokers; not showing that the combination of variables chosen fully account for the CHD risk in each group separately (except for the variable of smoking); and not establishing whether the risk was causal or constitutional in nature. Seltzer adds that no significant association was found between CHD and variables reported in previous medical literature: duration of smoking, inhalation during smoking, dyspnea, education level, uric acid level, glucose level, race, vital capacity, and leukocyte count. Further, the multivariate relative-risk estimate combined both races and sexes into a single group, and the effects of other factors associated with CHD risk, such as physical activity, family history, and more sophisticated psychosocial factors, were not considered. Three of the original article's authors offer a reply, first by responding that self-selection does not necessarily invalidate a finding, but must be examined critically in each instance. It would be difficult to find a combination of variables that could fully account for CHD risk or to account for every factor in the causal web of a disease. Secondly, it is stated that multivariate analysis is often needed to handle many variables. It is suggested that it is better to derive the relative importance of risk factors from the data rather than to impose subjective bias. Sample size limitation led to only a single multivariate analysis across all race, sex, and age groups. See also 81-1497.

82–0113. Dawber, T. R. **The Framingham Study. The Epidemiology of Atherosclerotic Disease.** Cambridge, Massachusetts, Harvard University Press, 1980, 267 pp.

The Framingham Study represents a 24-year epidemiological investigation of the risk factors of atherosclerosis, following the life habits and health of 6,000 men and women. The results provide the basis for current recommendations for decreasing blood pressure, lowering cholesterol, reducing weight, monitoring fat intake in diabetics, increasing physical activity, and discontinuing cigarette smoking, in the prevention of atherosclerosis. The history of the study, from development of the hypotheses to the selection of the population sample, to the examination of the methodological problems encountered in longitudinal research, is presented.

82-0114. Dimsdale, J. E.; Hutter, A. M., Jr.; Hackett, T. P.; Block, P. C. **Predicting Extensive Coronary Artery Disease.** *Journal of Chronic Diseases* 34(11): 513-517, 1981.

In 171 men (aged 18 to 70) who underwent cardiac catheterization at Massachusetts General Hospital, smoking was identified, using stepwise multiple regression, as a prime risk factor in the prediction of the extent of coronary vessel disease. The only factors of greater significance were family history and age. Less important were cholesterol, angina severity, depression, Q waves, Peel Score, and anger. Multiple regression analysis predicts a dependent variable (Jeopardy Score) as a function of a combination of independent variables (risk factors), minimizing redundancy of information. Smoking history was determined in terms of pack-years. A curious finding was that smoking history was inversely related to multiple vessel disease; that is, people with many diseased vessels were light smokers. Although this is most likely a chance finding, it is speculated that heavy smokers have more lethal disease in the early phases of atherosclerosis and thus are underrepresented in the multiple vessel disease group. It is concluded that since family history is the most important risk factor for multiple vessel coronary disease, it is important to establish the pathophysiology of this relationship.

82-0115. Duncan, A.; DePratti, V. J.; George, R. R. **Elevated β-Thromboglobulin Levels Associated With Smoking and Oral Contraceptive Agents in Normal Healthy Women.** *Thrombosis Research* 21(4/5): 425-430, 1981.

A predisposition to thromboembolism in women has been associated with the taking of oral contraceptives (OC) and with smoking. Plasma measurement of β-thromboglobulin (β-TG) has been suggested as a sensitive indicator of thromboembolic disease states. A study, conducted among a group of women aged 18 to 46 years, included: (1) normal controls ($n=29$) who had never smoked and had not used OCs for at least 1 yr; (2) a group of 31 who did not smoke, but had been using OCs for at least 6 months; (3) 29 who did not use OCs but smoked; and (4) 22 smokers who were also OC users. Elevated levels of β-TG were found in OC users and smokers, but not in those who were both smokers and OC users. The results from the OC plus smoking group were no different than those of the controls, an outcome the study was unable to explain. The other findings, from the smokers only and the OC users only, seem to indicate an association with intravascular platelet release and an increased risk of thrombosis.

82-0116. Erikssen, J.; Forfang, K.; Jervell, J. **Coronary Risk Factors and Physical Fitness in Healthy Middle-Aged Men.** *Acta Medica Scandinavica* Supplement 645: 57-64, 1981.

A group of 1,832 men, between 40 and 59 years of age, underwent a near maximal bicycle exercise test, to examine possible associations between various levels of physical fitness and several selected coronary heart disease risk factors, including smoking. Physical fitness was found to decline with age. The arbitrary units used to measure smoking habits were: (1) ≥ 10 cigarettes a day, (2) 1 to 9 cigarettes a day, and (3) current nonsmokers. There was a highly significant inverse relationship between cigarette consumption and physical fitness. Cigarette consumption was also found to be lowest among the youngest individuals. The most physically fit individuals in all age groups invariably smoked the least, as well as having the highest spirographic values and lowest lipid levels, resting heart rate, and blood pressures. They also had lower hemoglobin concentration, possibly due to less smoking, as smokers appear to have a relative polycytemia.

82-0117. Freestone, S.; Ramsay, L. E. **Effect of Coffee and Cigarette Smoking in Untreated and Diuretic-Treated Hypertensive Patients.** *British Journal of Clinical Pharmacology* 11(4): 428P-429P, 1981.

The effects on pulse and blood pressure (BP) of coffee drinking, cigarette smoking, and a combination of the two habits were examined. The study involved 16 hypertensive patients, 8 with mild, untreated hypertension and 8 being treated with thiazide diuretic. All were habitual users of both caffeine and cigarettes. Each subject was studied on 4 separate days to compare the effect of: (1) a placebo (orange juice); (2) 500 ml of coffee containing about 200 mg caffeine, with no cigarettes; (3) smoking two king size tipped cigarettes, with orange juice but no caffeine; and (4) coffee plus smoking. The four stimuli were each taken over 15 minutes, and recumbent pulse and BP were measured for 120 minutes. The pattern of pulse and BP responses was similar in untreated and thiazide-treated patients. Smoking increased systolic and diastolic BP at 5 and 15 minutes and increased the pulse rate until 120 minutes. Coffee and cigarettes together increased systolic BP, diastolic BP, and pulse very significantly at all times from 5 to 120 minutes. The effect of coffee and cigarettes on pulse rate was no greater than that of smoking only, but the effect on BP was greater than from either smoking or coffee alone. The combined effect of coffee and cigarette smoking, lasting for at least 2 hours, was considered sufficient to change normotension to mild hypertension, or to cause a shift from good to poor blood pressure control.

82-0118. Greenhalgh, R. M.; Laing, S. P.; Cole, P. V.; Taylor, G. W. **Smoking and Arterial Reconstruction.** *British Journal of Surgery* 68(9): 605-607, September 1981.

Fasting levels of serum triglyceride, serum cholesterol, lipoprotein, uric acid, fibrinogen, and carboxyhemoglobin (COHb) were measured in 64 patients with stenosing arterial disease before reconstructive surgery, and compared with those for normal, age- and sex-matched controls. All except fibrinogen were significantly higher in the patients with arterial disease than in the controls. The outcome of arterial reconstruction, assessed both clinically and by Doppler pressure measurements, was compared in terms of these risk factors. The reconstructions of 12 patients failed between 3 months and 5 years, leaving 52 patients with patent reconstructions at the end of the followup period. There was no difference between the two groups in terms of any of the risk factors, except for COHb. The COHb level (associated with inhalation of cigarette smoke) was significantly higher in the reconstruction failure group than in the reconstruction success group. It is concluded that patients should stop smoking cigarettes before reconstructive arterial surgery is undertaken. (Auth. Abs.)

82–0119. Hansen, J. F. **Chest Pain, Risk Factors and Coronary Artery Disease.** *Danish Medical Bulletin* 27(3): 148-152, July 1980.

The value of certain risk factors in the diagnosis for Coronary Artery Disease (CAD) is investigated. Selective coronary arteriography was performed on 185 male and 49 female patients between 20 and 70 years of age. Smokers constituted 79 percent of the group. Prior to the arteriography a detailed history was taken, including chest pain (location, duration, and provoking factors), se-cholesterol, se-triglycerides, and hyperlipidemia, smoking, family history of CAD, presence of angina pectoris, and glucose intolerance. Although smoking, family history, and hyperlipidemia were highly significant in the incidence of CAD, this information had limited diagnostic value. However, the combination of angina pectoris and se-cholesterol led to a diagnostic efficiency of 91 percent.

82–0120. Hartz, A. J.; Barboriak, P. N.; Anderson, A. J.; Hoffmann, R. G.; Barboriak, J. J. **Smoking, Coronary Artery Occlusion, and Nonfatal Myocardial Infarction.** *Journal of the American Medical Association* 246(8): 851-853, August 21, 1981.

The association of smoking with a history of myocardial infarction (MI) was studied in 3,997 men who had coronary arteriography. The patients were subdivided into groups based on coronary occlusion (minimal, moderate, or severe) and plasma cholesterol level (low, moderate, or high). For men older than 50 years, smoking was significantly associated with MI in each occlusion group. For men younger than 50 years, the association was significant for men with moderate or severe occlusion. In the presence of higher cholesterol levels there was a stronger association of smoking with MI, but a weaker association of smoking with coronary occlusion. These results suggest that the association of smoking with MI does not depend primarily on the atherogenic effect of smoking. The association seems to be enhanced by high levels of coronary occlusion and cholesterol. (Auth. Abs.)

82–0121. Hopkins, P. N.; Williams, R. R. **A Survey of 246 Suggested Coronary Risk Factors.** *Atherosclerosis* 40(1): 1-52, August-September 1981.

A survey of 246 risk factors suggested as being associated with coronary heart disease (CHD), among them smoking and its effects, is reviewed which may provide clues to the etiology of CHD and suggest possible preventive measures. The CHD risk factors are listed, with probable mechanisms of action and associated risks, when available from human studies. Cigarette smoking is described as an initiator, promoter, potentiator, and precipitator of CHD associated with increased platelet adherence and thrombotic tendency, increased endothelial permeability, arrhythmias, and lower levels of high density lipoprotein. Cigarette smoking apparently is a significant risk factor when serum cholesterol concentrations are above 180 mg/dl. The importance of multiple risk factors in CHD is exemplified by the synergistic effect between cigarette smoking and the use of oral contraceptives. Either moderate smoking or oral contraceptive use about doubles the risk of a myocardial infarction in younger women, and the two factors combined increase the risk 12-fold. Methods for CHD prevention are also reviewed, and the importance of early prevention programs is stressed. A bibliography of all studies cited is included.

82–0122. Houben, H.; Thien, Th.; Van 'T Laar, A. **Haemodynamic Effects of Cigarette Smoking During Chronic Selective and Non-Selective β-Adrenoceptor Blockade in Patients With Hypertension.** *British Journal of Clinical Pharmacology* 12(1): 67-72, 1981.

An investigation of the hemodynamic changes brought about by cigarette smoking in 16 patients with essential hypertension, before and during treatment with selective and nonselective β-adrenoceptor blocking drugs, is reported. It was suggested that smoking could evoke pressor responses during nonselective β-adrenoceptor blockage. It is known that nicotine ingested during smoking can promote vasoconstriction, tachycardia, and elevated blood pressure, and can activate the parasympathetic nervous system. Nicotine is also known to affect the sympathetic nervous system, inducing raised concentrations of plasma noradrenaline and adrenaline. Cigarette smoking induced equally elevated blood pressure during all experimental sessions. When the subjects were smoking, heart rate increased by 16 beats/min; but when the smokers were also given a β-adrenoceptor blocker, the increase was significantly less. During smoking, the plasma adrenaline concentration rose significantly from 0.37 nmol/l to 0.77 nmol/l. Plasma noradrenaline concentration did not change. Results indicate that in hypertensive patients the increase in blood pressure during smoking remained unchanged during chronic treatment with nonselective and selective β-adrenoceptor blockers. However, both drugs significantly modified the increase in heart rate caused by cigarette smoking. It was concluded that β-adrenoceptor blocking drugs were not necessarily preferable for treating hypertension in habitual smokers.

82–0123. Kannel, W. B. **Cigarettes, Coronary Occlusions, and Myocardial Infarction.** *Journal of the American Medical Association* 246(8): 871-872, August 21, 1981.

Substantial evidence exists that cigarette smoking is a risk factor in coronary heart disease. The smoke components most harmful to the cardiovascular system are: nicotine, which promotes catecholamine release, increases myocardial irritability and heart rate, causes vascular constriction and a transient rise in pressure, and causes platelets to become more adherent; and carbon monoxide, which decreases the amount of oxygen available to the myocardium. Epidemiologic data indicate that the risk of myocardial infarction increases progressively with the number of cigarettes smoked daily, and that the effect is more closely related to the number smoked daily than to the duration of the habit. Smoking may also promote atherosclerosis by inducing chronic hypoxia of the endothelium and affecting platelet function. Cigarette smoking appears to have little or no relationship to angina pectoris. Predisposing factors and a poor cardiovascular risk profile seem to be necessary prerequisites for the adverse effects of smoking, and those at low cardiovascular risk do not appear to experience the same effects of smoking as high risk individuals. Cigarette smoking is considered the most preventable contributor to cardiovascular morbidity and mortality.

82-0124. Kapuscinska, B.; Bochenek, W.; Chwistecki, K. **Badania Wplywu Palenia Tytoniu na Stezenie Cholesterolu Calkowitego i Cholesterolu Frakcji Lipoproteidowych Surowicy Krwi. [Research of the Effect of Smoking on Total Cholesterol and Cholesterol of Lipoprotein Fractions of Blood Serum.]** *Polski Tygodnik Lekarski* 35(42): 1599-1601, October 20, 1980, Polish.

Experiments were carried out with two groups of male subjects. Members of group 1, 18 smokers of at least 15 cigarettes/day, averaging 44 years of age, stopped smoking at the beginning of the experiment because of coronary disorders. The 12 healthy nonsmokers in group 2 who averaged 25 years of age, volunteered to smoke about 20 cigarettes/day for 3 weeks. In both groups, blood specimens were analyzed before the experiment and 1 and 3 weeks after the beginning of the experiment. In group 1, total cholesterol levels decreased slightly and cholesterol levels of the serum high-density lipoprotein (HDL_{ch}) fraction increased insignificantly after 1 week of abstinence from smoking. At the same time, cholesterol levels of the serum low-density lipoprotein (LDL_{ch}) fraction and the LDL_{ch}/HDL_{ch} ratio decreased significantly by 16.6 mg percent ($p<0.005$) and 0.6 ($p<0.02$), respectively, from 172 mg percent and 3.1 levels before the experiment. It is concluded that smoking cessation may reduce the risk of coronary artery disease by changing the blood serum lipid level. In group 2, smoking did not produce significant changes in the serum lipid contents. This indicated that ceasing smoking rather than starting influences lipoprotein content of blood serum.

82-0125. Maher, F. A.; Bing, O.H.L.; Huber, G. L.; Arenella, L.; Abelmann, W. H. **Effect of Acute Tobacco Smoke Inhalation on the Mechanical Responses of Isolated Rat Cardiac Muscle.** *Environmental Research* 26(1): 87-94, October 1981.

To evaluate the effects of acute inhalation of tobacco smoke upon the intrinsic properties of cardiac muscle, rats were exposed to cigarette smoke from 2R1 Kentucky reference cigarettes in special smoking chambers for periods of 10 minutes/hour for 5 hours. Animals were sacrificed, and the mechanical properties of isolated left ventricular papillary and columnar carneae preparations were studied. Mean COHb concentration of animals exposed to tobacco smoke was 13.2±0.9 volume percent. No significant difference was found in rats exposed to tobacco smoke when compared to shams in: (1) cardiac chamber weight or chamber wet-to-dry weight ratios; (2) cardiac muscle performance in oxygenated Krebs-Henseleit solution (95 percent O_2, 5 percent CO_2); (3) muscle performance during 60 minutes of hypoxia (95 percent N_2, 5 percent CO_2), followed by 30 minutes of reoxygenation; (4) muscle performance during graded hypoxia with and without glycolytic blockade (iodoacetic acid 10^{-4} M); or (5) sensitivity of myocardial performance to isoproterenol. Thus, after acute exposure to tobacco smoke, no effects on the intrinsic mechanical properties of rat cardiac muscle were demonstrable. (Auth. Abs.)

82-0126. Manthey, J.; Stoeppler, M.; Morgenstern, W.; Nussel, E.; Opherk, D.; Weintraut, A.; Wesch, H.; Kubler, W. **Magnesium and Trace Metals: Risk Factors for Coronary Heart Disease? Associations Between Blood Levels and Angiographic Findings.** *Circulation* 64(4): 722-729, October 1981.

A deficiency or an excessive intake of metals with cardiovascular effects is suspected in the pathogenesis of coronary heart disease (CHD) and sudden death. In patients undergoing coronary arteriography, serum levels of six essential metals (magnesium, chromium, copper, manganese, selenium, and zinc) and whole blood concentrations of two elements without known essential function (cadmium and lead) were measured using atomic absorption spectrophotometry, including the flameless technique, or neutron activation analysis. Examination of associations between metal concentrations and clinical characteristics revealed markedly elevated blood cadmium levels in cigarette smokers ($n=28$), at 2.49±1.72 µg/l, compared to nonsmokers ($n=18$), 0.43±0.22 µg/l, with $p<0.001$. A correlation was noted between cadmium levels and the number of cigarettes smoked per day ($r=0.725$, $p<0.001$). It is concluded that cigarette smoking may be associated with increased serum cadmium levels, which may help explain the contribution of smoking to the risk of sudden death in patients with CHD. (Auth. Abs. Mod.)

82-0127. Norris, R. M.; Agnew, T. M.; Brandt, P.W.T.; Graham, K. J.; Hill, D. G.; Kerr, A. R.; Lowe, J. B.; Roche, A.H.G.; Whitlock, R.M.L.; Barratt-Boyes, B. G. **Coronary Surgery After Recurrent Myocardial Infarction: Progress of a Trial Comparing Surgical With Nonsurgical Management for Asymptomatic Patients With Advanced Coronary Disease.** *Circulation* 63(4): 785-792, April 1981.

Smoking histories were recorded during a surgical versus nonsurgical management trial, for a group of men aged 60 years or younger who had recovered from a recurrent myocardial infarction. Those patients who smoked were counseled to quit. Most of the patients stopped smoking or modified their smoking habit. Before the survey and counseling, 42 percent of the patients were nonsmokers or had stopped smoking. After entry to the trial, the figure climbed to 77 percent. Of the 23 percent of infarct patients who continued to smoke, the percentages for before and after entry into the management trial were: (1) less than 10 cigarettes/day, from 6 percent to 9 percent; (2) 10 to 19 cigarettes/day, from 15 percent to 9 percent; (3) 20 to 29 cigarettes/day, from 22 percent to 3 percent; and (4) 30 or more cigarettes/day, from 15 percent to 2 percent. The high rates of cessation or reduction of smoking among the patients may have been of some significance to the results of the treatment.

82–0128. Ossip, D. J.; Andrasik, F.; Epstein, L. H. **The Effect of Nicotine Extraction Filters on Pulse Rate and Vasomotor Activity: A Methodological Note.** *Addictive Behaviors* 6(2): 149-152, 1981.

Cardiovascular and peripheral vascular changes, such as heart rate increase and vasoconstriction, showed varying responses when three study subjects smoked high- and low-nicotine cigarettes. However, the use of different cigarette brands to manipulate nicotine is confounded in part by differences in taste and such components of manufacture as filter, paper, or length differences. Improved control of nicotine delivery using the same cigarette, which would reduce extraneous intercigarette effects, has recently been made possible by commercially available nicotine extraction filters. The present study is a within-subject analysis of the cardiovascular changes related to smoking cigarettes using two levels of nicotine extraction filters. (Auth. Abs. Mod.)

82–0129. Rabkin, S. W.; Boyko, E.; Streja, D. A. **Relationship of Weight Loss and Cigarette Smoking to Changes in High-Density Lipoprotein Cholesterol.** *American Journal of Clinical Nutrition* 34(9): 1764-1768, September 1981.

Both cigarette smoking and low serum levels of high-density lipoprotein cholesterol (HDL-C) are associated with atherosclerotic diseases. An investigation was conducted to examine the effect of weight loss on HDL-C levels in an overweight group of smokers and nonsmokers. Serum HDL-C was measured among women ($n = 56$) and men ($n = 9$) before and after a weight reduction program. None of the three male smokers and eight female smokers in the study quit smoking during the program. Despite a significant weight loss of 4.5 kg (5.8 percent of initial body weight among all subjects), there was no significant increase in HDL-C. The subset of women who were current smokers, however, showed a significant correlation ($p < 0.01$) between change in HDL-C and change in weight ($r = -0.876$) and percentage change in weight ($r = -0.881$). There were decreased HDL-C levels in women who smoked, compared to nonsmokers. The association was somewhat surprising in view of the negative association between smoking and body weight that might be expected to result in an increase in HDL-C, instead of the decrease observed. The smaller number of male smokers precluded meaningful analysis of smoking status and HDL-C changes.

82–0130. Seltzer, C. C. **Cigarette Smoking and Coronary Heart Disease: A Questionable Connection.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 267-277.

The data concerning the causal relationship between smoking and coronary heart disease (CHD), the leading cause of death and disability in the western world, is reevaluated to show that current scientific data do not support this causal hypothesis. The Surgeon General's 1964 report did not find a causal connection between cigarettes and CHD, and more recent evidence in support of the relationship is neither definitive, consistent, nor conclusive. For instance, it has not been proven by any scientific mechanisms that some component of cigarette smoke adversely affects coronary vessels; the Seven Countries study by Keys found no association between cigarette smoking and CHD in Finland, the Netherlands, Yugoslavia, Italy, Greece, or Japan. The relatively weak associations found are often attributable to some other underlying characteristic. Studies also indicate that cessation of smoking does not lower the risk for CHD, and that there is no relationship between passive smoking and CHD among nonsmokers.

82–0131. Simons, L.; Whish, P.; Marr, B.; Jones, A.; Simons, J. **Coronary Risk Factors in a Rural Community Which Includes Aborigines: Inverell Heart Disease Prevention Programme.** *Australian and New Zealand Journal of Medicine* 11(4): 386-390, August 1981.

A screening program and referral service for coronary risk factors in New South Wales found that the prevalence of smoking decreased with age among Aboriginal and non-Aboriginal subjects. The program and service were initiated in July 1978. Other risk factors surveyed were hyperlipidemia, hypertension, and obesity. During a 12-month period, a total of 2,612 non-Aboriginal and 224 Aboriginal subjects over 20 years of age were examined. Among all non-Aboriginal subjects the prevalence of cigarette smoking decreased with age, in contrast to the prevalence of all remaining risk factors, which increased with age. The prevalence of cigarette smoking also declined with age among all Aboriginal subjects, though smoking levels still remained higher than among the non-Aboriginal subjects. The number of current cigarette smokers did not appear to differ significantly between rural Inverell and Sydney. Subjects with multiple risk factors, considered "high risk" ($n = 430$) and termed the "Referral Group," were referred to local practitioners. Subjects with a single risk factor were selected at random ($n = 200$) as a "Control Group." Neither group had Aboriginal subjects. Rescreening at the end of the 12-month period found that 29 percent of the Referral Group were using fewer or no cigarettes, compared to 14 percent of the Control Group. Reduction in smoking appeared to represent a passive effect of the program, strengthened by community and media awareness.

82–0132. Sinzinger, H.; Kaliman, J.; Widhalm, K.; Pachinger, O.; Probst, P. **Value of Platelet Sensitivity to Antiaggregatory Prostaglandins (PGI_2, PGE_1, PGD_2) in 50 Patients With Myocardial Infarction at Young Age.** *Prostaglandins and Medicine* 7(2): 125-132, August 1981.

Based upon earlier findings that not only the synthesis of antiaggregatory prostaglandins by the vessel wall is altered in atherosclerotic vascular tissue of patients, but also the sensitivity of their platelets to these prostaglandins is changed, a well-defined group of 50 patients with myocardial infarction before the age of 40 years was examined. In all patients the coronary heart disease was verified by angiography. The sensitivity of the platelets was examined in relation to the smoking habit; the type and presence of hyperlipoproteinemia; one, two, and three vessel disease; and familial history. Except for hyperlipoproteinemia and smoking, all the patients were free of any additional risk factor. It was demonstrated that the platelet sensitivity to the antiaggregatory prostaglandins is significantly diminished in hyperlipoproteinemics in comparison to a control group. (Auth. Abs.)

82–0133. Sorlie, P. D.; Garcia-Palmieri, M. R.; Castillo-Staab, M. I.; Costas, R., Jr.; Oalmann, M. C.; Havlik, R. **The Relation of Antemortem Factors to Atherosclerosis at Autopsy. The Puerto Rico Heart Health Program.** *American Journal of Pathology* 103(3): 345-352, June 1981.

A group of Puerto Rican men (n=9,824) aged 35 to 79 took part in a prospective study of cardiovascular risk factors, including smoking. From 1965 to 1977 there were 970 deaths, of which 139 had a protocol autopsy using the procedures of the International Atherosclerosis Project. Correlation coefficients were determined for aortic atherosclerosis, and the strongest correlation found was with cigarette smoking. The average correlation coefficient was 0.32 for 120 subjects, 0.37 for the 31 rural subjects, and 0.31 for the 89 subjects from urban areas. The relationship with cigarettes was almost nonexistent for atherosclerosis in the coronary arteries, -0.16 among the 139 subjects (-0.05 for the 36 from rural areas and -0.22 for the 103 from urban areas). Correlations were also run for those deaths not of cardiovascular origin. The association of atherosclerosis in coronary arteries with smoking measured 0.06, while in the aorta it was 0.32. In this study, previous smoking status appeared to be associated with lesions only in the aorta.

82–0134. Sullivan, J. M.; Connors, N. A.; Rana, M. W.; Lawson, C. **Morphological Effects of Nicotine on Cardiac Arteries of the Rat.** *IRCS Medical Science: Cardiovascular System* 9: 340-341, 1981.

Nicotine's acute cardiovascular effects resemble those of sympathetic stimulation. It appears to cause sympathomimetic effects by releasing norepinephrine (NE) from adrenergic nerve terminals. A population of Sprague-Dawley rats (17 male, 17 female) were divided into two experimental groups and one control group. Group I received 0.5 mg/kg nicotine in one injection/day; group II received 1.0 mg/kg nicotine in two injections/day; and group III, the controls, received 1.0 ml of normal saline injection/day. The first effects of nicotine injection were increased breathing and sympathetic stimulation. The medial muscle mass in the hearts of the female experimental groups was greater than in the controls and increased with increasing dosages. The effects in males were inconsistent. In females in both experimental groups there was an increase in NE within the postganglionic sympathetic nervous system fibers along the cardiac arteries. Males in group I displayed a slight increase in NE, while males in group II did not show an increase in NE when compared to group I. The increase in medial muscle mass and NE in the females as a result of nicotine injection supports the contention that nicotine's effects are similar to those of adrenergic nerve stimulation.

82–0135. Thomas, M. **Smoking and Vascular Surgery.** *British Journal of Surgery* 68(9): 601-604, September 1981.

The first long-term study to determine whether smoking is a risk factor in the development of vascular disease was begun in Framingham, Massachusetts, in 1949, in which the subjects (n=5,209) were examined for a followup period of 16 years. Smoking was shown to be a risk factor, being more important for intermittent claudication than for stroke. Other studies have also found smoking to be a high-risk factor for intermittent claudication. Smoking, although not the cause or initiating factor of atherosclerosis, since atheroma existed before the advent of tobacco, probably accelerates the progression of disease already present. Smoking patients with vascular disease appear to have a better prognosis if they reduce or stop smoking, thus requiring less arterial surgery and fewer amputations, and experiencing fewer deaths. The effect of smoking on atherosclerosis is uncertain, but nicotine has not yet been implicated, although increased carboxyhemoglobin levels in smokers appear to cause greater risk of atheroma. Smoking also seems to adversely affect the results of arterial surgery, though the precise mechanism has not yet been elucidated. It is recommended that patients with intermittent claudication should not be offered elective arterial reconstruction unless they have stopped smoking.

82–0136. Vogelzang, N. J.; Bosl, G. J.; Johnson, K.; Kennedy, B. J. **Raynaud's Phenomenon: A Common Toxicity After Combination Chemotherapy for Testicular Cancer.** *Annals of Internal Medicine* 95(3): 288-292, September 1981.

The possible relationship between smoking and Raynaud's phenomenon, an illness characterized by intermittent pallor or cyanosis of extremities such as the fingers or toes, or less often, the ears and nose, is discussed. Patients (n=60) who had been treated for germ cell testicular cancer with vinblastine and bleomycin (and sometimes cisplatin) were evaluated for the presence of Raynaud's phenomenon. Methods used for evaluation were questionnaires, physician interviews and examinations, and chart reviews. The illness was detected in 22 of the patients (37 percent), there was equivocal evidence in 6 (10 percent), and no evidence in the remaining 32 (53 percent). The patients did not differ with respect to median age or tumor histology; however, patients with Raynaud's phenomenon were more likely to be cigarette smokers (17 of 22) than were disease-free patients (14 of 32). The difference was considered significant, with a chi-square of 5.99 ($p=0.02$). Bleomycin, vinblastine, and possibly cisplatin may have been responsible for the digital arterial damage in the patients. Cigarette smoking is associated with peripheral vascular disease, however, and may have promoted the drug-induced arterial damage.

See also, **82–0054, 82–0063, 82–0074, 82–0186**

PREGNANCY AND INFANT HEALTH

82–0137. Bosley, A.R.J.; Sibert, J. R.; Newcombe, R. G. **Effects of Maternal Smoking on Fetal Growth and Nutrition.** *Archives of Disease in Childhood* 56(9): 727-729, September 1981.

Standard anthropometric measurements made on 320 term neonates, 126 born to mothers who smoked, found that maternal smoking reduced fetal growth. Maternal height and triceps skinfold thickness were also measured. Maternal triceps skinfold thickness was significantly smaller in smoking mothers, and a correlation existed between maternal and infant triceps skinfold thickness. Measurements of infant growth, birth weight, occipito-frontal circumference, and crown-to-heel length were significantly smaller in infants of smoking mothers and remained significantly smaller when corrections were made for maternal triceps skinfold thickness, height, and social class. It was suggested that the reductions in head circumference may indicate long-term effects of maternal smoking on children's learning ability. While the data did not exclude a nutritional mechanism for the effect of maternal smoking on the fetus, the major growth-retarding effects remained after corrections for this, indicating that the mechanisms were multifactorial. Another possible explanation is that the direct toxic effects of nicotine, cyanide, carbon monoxide, or other toxins retard fetal growth. In addition, fetal oxygenation and growth could be adversely affected by carboxyhemoglobin in the cord blood of infants of mothers who smoke. (Auth. Abs. Mod.)

82–0138. Dalton, E. R.; Hughes, C. A.; Cogswell, J. J. **Cigarette Smoking in Pregnancy: A Health Education Problem.** *Public Health, London* 95: 207-214, 1981.

The smoking habits of 285 pregnant mothers were investigated in a maternity department in South East London. Thirty-two percent of the mothers who were smokers when they became pregnant claimed no medical adviser had attempted to dissuade them from smoking. A significantly higher percentage of the mothers who gave up smoking in early pregnancy admitted a knowledge of the possible hazards to the fetus than those who smoked continuously throughout pregnancy. The percentages admitting knowledge of the dangers to the fetus and to themselves were significantly higher among those who acknowledged receiving advice from their medical advisers. The poster campaign instituted by the Health Education Council had no significant effect. As an avoidable risk factor, more efforts need to be made to dissuade pregnant mothers from the smoking habit. (Auth. Abs. Mod.)

82–0139. Harrison, K. L.; Robinson, A. G. **The Effect of Maternal Smoking on Carboxyhemoglobin Levels and Acid-Base Balance of the Fetus.** *Clinical Toxicology* 18(2): 165-168, 1981.

A correlation of combined pH, pO_2, pCO_2, and COHb levels with extent of maternal smoking and potential effect on fetal weight is explored. Cord blood from 55 infants was collected and analyzed, revealing lower pO_2, and higher pCO_2 and hematocrits. All parameters showed an effect related to dosage. Infants of smoking mothers show many characteristics of infants born at high altitudes. This evidence of chronic uterine hypoxia provides a potential mechanism for the reduced birth weight found in the infants of mothers who smoke.

82–0140. Huel, G.; Boudene, C.; Ibrahim, M. A. **Cadmium and Lead Content of Maternal and Newborn Hair: Relationship to Parity, Birth Weight, and Hypertension.** *Archives of Environmental Health* 36(5): 221-227, September-October 1981.

Since it is known that specific toxic metal trace elements may become bioconcentrated in mammalian hair, head hair samples were acquired from 110 mothers and their newborn infants and examined for cadmium and lead content. Several chemical and metallurgical factories are located in the area where the study was conducted. Data on the mothers' smoking habits showed only 13 mothers smoked during pregnancy and most smoked less than five cigarettes/day. A positive association was found between mothers and newborns for cadmium content, but not for lead. Although no relationship was found in this study between maternal smoking and the trace metals, the study refers to earlier reports of cadmium in cigarette smoke becoming a part of the total body burden of cadmium. The possibility that maternal smoking can lead to reduced infant birth weight is also mentioned.

82–0141. Karki, N. T.; Pelkonen, O.; Tuimala, R.; Kauppila, A.; Koivisto, M. **Aryl Hydrocarbon Hydroxylase Induction in Maternal and Cord Blood Mitogen-Treated Lymphocytes.** *Developmental Pharmacology and Therapeutics* 2(1): 32-43, 1981.

The inducibility of aryl hydrocarbon hydroxylase (AHH) was measured in mitogen-activated peripheral lymphocytes from smoking and nonsmoking mothers and their newborn infants (cord blood). The mean inducibility ratio in lymphocytes from smoking women was 4.02 and that for nonsmokers 2.87. Benz(*a*)anthracene-induced and noninduced AHH activities were 3-6 times higher in the lymphocytes from the cord blood than in those from the mothers. The inducibility ratio in the cord blood lymphocytes from the smoking mothers was 3.65 and did not differ significantly from that for the nonsmokers, 3.54. There was a significant correlation in the induction ratio between maternal and cord blood lymphocytes from the nonsmokers, but not in the smokers. Thymidine incorporation was about twice as high on average in the cord blood lymphocytes as in the maternal lymphocytes. The results demonstrate that the extent and distribution of inducibility were very similar in the maternal and cord blood lymphocytes. (Auth. Abs.)

82–0142. Kuzma, J. W.; Kissinger, D. G. **Patterns of Alcohol and Cigarette Use in Pregnancy.** *Neurobehavioral Toxicology and Teratology* 3(2): 211-221, 1981.

Maternal smoking and drinking have been implicated as factors in the adverse outcome of pregnancies. In a California study of women ($n = 12{,}406$) in a broad socioeconomic range, a large proportion were found to smoke (35 percent) or drink (51 percent) during pregnancy. Drinking habits appeared to be associated with age, ethnicity, marital status, income, education, church attendance, and prenatal care; but not with gestational age, breast-feeding, or self-reported health status. Smoking showed an association with income and education, with women of higher economic and educational status being less likely to smoke. Drinking and smoking are closely interrelated, showing strikingly parallel associations with such factors as use of caffeine or illicit drugs. Subgroups of pregnant women who exhibited differing drinking habits were found not comparable on certain other characteristics which potentially influence the health of neonates. The importance of utilizing large populations, adjusting for confounding variables, and paying particular attention to smoking habits should be emphasized. Forthcoming multivariate analyses of the data set may disentangle effects of the confounding variables. (Auth. Abs. Mod.)

82–0143. MacKenzie, K. M.; Angevine, D. M. **Infertility in Mice Exposed in Utero to Benzo(*a*)pyrene.** *Biology of Reproduction* 24(1): 183-191, February 1981.

The effect of prenatal exposure to benzo(*a*)pyrene (BP) on pregnancy maintenance, fetal development and survival, and postnatal development and reproductive function of F_1 male and female CD-1 mice was evaluated. Daily oral doses of 0, 10, 40, and 160 mg BP/kg maternal body weight were administered on days 7-16 of gestation. Total sterility was observed in 97 percent of F_1 mice exposed prenatally to 40 or 160 mg BP/kg. Fertility was markedly impaired in F_1 animals exposed in utero to 10 mg BP/kg. After 6 months on a breeding study, female mice exposed to this dose of BP in utero gave birth to significantly fewer and smaller litters. Male mice exposed to 10 mg BP/kg impregnated 35 percent fewer females than did control males. The impaired fertility in F_1 mice exposed to BP in utero was associated with marked alterations in gametogenesis and folliculogenesis and a dramatic decrease in the size of the gonads. Germ aplasia was observed in males as well as a reduction in the size of the seminiferous tubules and an increase in the quantity of interstitial tissue. Microscopic examination of ovarian tissue from F_1 females exposed to 10 mg BP/kg revealed marked hypoplasia with very few corpora lutea or follicles. The data demonstrate a sensitivity of fetal gonads to BP which is similar to effects observed with another polycyclic aromatic hydrocarbon, 9,10-dimethyl-1,2-benzanthracene. (Auth. Abs. Mod.)

82–0144. Rowell, P. P. **The Effect of Maternal Cigarette Smoking on the Ability of Human Placental Villi To Concentrate α-Aminoisobutyric Acid *in Vitro*.** *Research Communications in Substance Abuse* 2(3): 253-266, 1981.

The ability of isolated human placental villous tissue to concentrate the nonmetabolizable neutral amino acid α-aminoisobutyric acid (AIB) was determined in placentas from smoking and nonsmoking mothers. Placental villous fragments were incubated in ^{14}C-AIB for periods up to 90 minutes. It was found that after 60 minutes of incubation, the tissue from nonsmokers had a 24 percent higher intracellular concentration of AIB compared to smokers. The major factor contributing to the lower intracellular concentration in the tissue from smokers was a depressed active uptake of AIB from the medium. A contributing factor was also a slightly elevated intracellular volume in the tissue from smokers, probably attributable to more edematous placentas. Measurements of the Na-K ATPase activity in the villous tissue indicated that there was no statistical difference between smokers and nonsmokers. It is suggested that the decreased availability of amino acids to the fetus, which would result from a lower placental intracellular concentration, could contribute to the lower birth weights of babies from mothers who smoke cigarettes during pregnancy. (Auth. Abs.)

82–0145. Rush, D. **Cigarette Smoking, Nutrition, Social Status, and Perinatal Loss: Their Interactive Relationships.** In:

Porter, I. H.; Hook, E. B. (Editors). *Human Embryonic and Fetal Death.* New York, Academic Press, 1980, pp. 207-219.

An analysis of the interactive relationships of smoking and other factors to the outcome of pregnancy reveals that much of the "social class" effect on perinatal mortality is due to maternal smoking. In the English-speaking countries, lower status women smoke more often and at higher levels. After controlling for the amount smoked, the effects of maternal smoking on the fetus are greater among the poor. The mechanism for this more intense effect is not obvious, but probably involves nutrition. Women of lower weight at conception are more vulnerable to the effects of tobacco, and it seems likely that maternal smoking can affect the fetus through altered nutrition and weight gain. When weight gain is sustained, much of the effect of smoking on fetal growth (and possibly on perinatal loss) may be ameliorated. Preliminary observations suggest that the upper status smoker may be protecting her fetus by sustaining her diet and weight gain. (Auth. Abs. Mod.)

82-0146. Sram, R. J. **Geneticke Riziko Koureni. [Genetic Risk of Smoking.]** *Prakticky Lekar* 61(2): 49-53, 1981, Czechoslovakian.

Mutagens in cigarette smoke, testing of mutagenic activity of cigarette smoke, effect of smoking on genetic material in smokers as evidenced by analysis of mutagenic activity of chemical substances, and the relationship between smoking and outcome of pregnancy are discussed. Research has indicated an increased risk of fetal development abnormalities in smoking mothers. However, the difference between smoking and nonsmoking mothers with respect to the frequency of fetal abnormalities was not evident, if the mother was exposed simultaneously to chemical substances at work. Genetic damage appears to be highly significant in children of smoking fathers. In the case of workers exposed simultaneously to mutagens and carcinogens (e.g., asbestos, uranium), smoking increases by a factor of 10 to 15 the genetic risk from a single substance. It is concluded that smoking presents a high genetic risk for present and future generations.

82-0147. Terrin, M.; Meyer, M. B. **Birth Weight-Specific Rates as a Bias in the Effects of Smoking and Other Perinatal Hazards.** *Obstetrics and Gynecology* 58(5): 636-638, November 1981.

Maternal smoking was found to increase perinatal mortality rates in the data examined from the Ontario Perinatal Mortality Study conducted during 1960 to 1961. The records of 31,788 births among English-speaking Canadian-born women were examined. Overall perinatal mortality rose from 22.0 to 28.2 per 1,000 if the mother smoked, a 27 percent increase. When perinatal mortality was computed in birth weight specific subgroups (less than 2,500 g, and the normal 2,500 g or more), the rates for smokers were lower than those for nonsmokers in both birth weight subgroups, though the rate difference for the normal birth weight subgroup was not considered statistically significant. These seemingly paradoxical results occurred because maternal smoking had an even stronger effect on birth weight than on perinatal mortality. Smoking had a small to moderate effect on the percentage of deaths under 2,500 g, and a large effect on the percentage of total births under 2,500 g. Smoking increased the total percentage of births in the low weight subgroup, from 4.5 percent for nonsmokers to 9.4 percent for smokers. The percentage of perinatal deaths in the low weight subgroup increased from 64.7 percent for nonsmokers to 73.7 percent for smokers. Although smoking increased the percentage of deaths in the low weight subgroup, the increase was not as large proportionately as the 106 percent increase in total births under 2,500 g associated with smoking. The data confirmed the hazard of maternal smoking for the unborn. The mechanisms by which maternal smoking influences birth weight and perinatal mortality may include physiological events such as fetal oxygenation or clinical events such as abruptio placentae and premature rupture of membranes. (Auth. Abs. Mod.)

82-0148. Yeung, D. L.; Pennell, M. D.; Leung, M.; Hall, J. **Effects of Maternal Cigarette Smoking During Pregnancy on Birthsize, Growth of Infants and Infant Feeding Practices.** *Nutrition Reports International* 23(5): 887-900, May 1981.

A longitudinal nutrition survey of 403 infants was conducted in Toronto and Montreal between 1977 and 1979. Infants who were born to mothers who smoked cigarettes during pregnancy (CS) were found to be 230 g lighter and 1.0 cm shorter than infants whose mothers did not smoke (NCS). The greater the number of cigarettes smoked, the smaller the birth size. The CS infants were also smaller than the NCS infants in weight, length, and head circumference at 6, 12, and 18 months. However, there was no difference in body fatness between the two groups of infants at the specified time of study. There was no difference in the postnatal rate of weight and length gain between the two groups of infants, implying that no catch-up growth occurred among CS infants. The survey's results suggest that cigarette smoking not only has an effect on the outcome of pregnancy, but may also have a long-term effect on the progeny. (Auth. Abs. Mod.)

82-0149. Yeung, D. L.; Pennell, M. D.; Leung, M.; Hall, J. **Breastfeeding: Prevalence and Influencing Factors.** *Canadian Journal of Public Health* 72(3): 323-330, September-October 1981.

Between 1977 and 1979, a Toronto and Montreal survey of infant nutrition indicated that mothers who smoked were less likely to breast feed than those who did not smoke. Other factors measured for their influence on breast feeding were parents' education, total family income, ethnic identity, and birth weight. During the first week after birth, 71 percent of all surveyed infants were breast fed by their mothers vs. 21 percent at 6 months and 5 percent at 12 months. Survey data found that 76.8 percent of the nonsmoking mothers initially breast fed their babies, compared to 58.0 percent of the mothers who smoked. The median termination time of breast feeding was 2.7 months for smokers, compared to 4.1 months for nonsmokers. Possible factors suggested to explain this reduced duration of breast feeding were: (1) some mothers may have wanted to protect their infants from possible harmful effects of smoking, and (2) smoking may have reduced milk production. Infants born to mothers who smoke tend to have lower birth weights, which may also discourage breast feeding.

82–0150. Zenk, K. E. **An Overview of Perinatal Clinical Pharmacology.** *Clinics in Laboratory Medicine* 1(2): 361-375, June 1981.

The disposition of drugs in pregnancy, in preterm and term neonates, and in breast milk is discussed, including nicotine, one of the constituents ingested during smoking. Maternal smoking's adverse effects on the fetus are proportional to the number of cigarettes smoked each day. Mothers who smoke experience an increased incidence of stillbirth and of low birth weight in infants, although by the end of the first year, the small infants usually catch up in growth. During pregnancy, the fetus is an inadvertent recipient of drugs given to the mother. The placenta allows passage of most drugs at varying rates from maternal to fetal circulation. In neonates, the pharmacokinetics of drugs are affected by gestational age, postnatal age, previous exposure to drugs in utero, concomitant administration of drugs, and the existence of pathologic conditions in the infant such as acidosis, renal and/or liver dysfunction, and hyperbilirubinemia. The increased interest in the breastfeeding of infants requires greater attention to pharmacokinetic studies of maternal, milk, and infant drug concentrations.

OTHER DISEASES AND CONDITIONS

82–0151. Burrows, B.; Halonen, M.; Barbee, R. A.; Lebowitz, M. D. **The Relationship of Serum Immunoglobulin E to Cigarette Smoking.** *American Review of Respiratory Disease* 124(5): 523-525, November 1981.

Data from adults enrolled in a general population study in Tucson, Arizona, indicated that smokers have higher serum IgE concentrations than nonsmokers, despite a lower rate of allergy skin test reactivity, at least after 35 years of age. After 15 years of age there was a significant decline in serum IgE with age in both allergy skin test positive and skin test negative nonsmokers, but no significant age decline was noted in similar groups of smokers. Ex-smokers showed a decline in serum IgE concentrations since quitting cigarettes. Also, the marked increase in geometric mean IgE that was seen in nonsmokers during the spring was not observed in smokers. The data suggested that smoking leads, directly or indirectly, to an increase in serum IgE and that this excess IgE may be qualitatively different from that seen in nonsmokers. Possible mechanisms for the effect are discussed. The observations raised the possibility that some of the adverse health effects of smoking might be immunologically mediated. (Auth. Abs.)

82–0152. Carter, N. L.; Keen, K.; Waugh, R. L.; Murray, N.; Bulteau, V. G. **The Relations of Eye Colour and Smoking to Noise-Induced Permanent Threshold Shift.** *Audiology* 20(4): 336-346, 1981.

Effects of smoking on noise-induced permanent threshold shift were only found to be statistically significant at 1.0 kHz frequency in the left ear, although there is a suggestion that smoking might potentiate noise at the higher, more noise-sensitive frequency. This was concluded from a study of 118 otologically normal Australian soldiers conducted to determine the relationship between eye color and hearing level. The soldiers were 40 years of age or less with at least 8 years service. Each soldier was classified by eye color pigmentation and questioned concerning his medical history, noise exposure, and smoking habit. It was hypothesized that cigarette nicotine would impair the protection given to hearing by the presence of melanin in the ears of brown-eyed persons, but have little or no effect on the susceptibility of blue- or grey-eyed persons, since they lack melanin in their eyes. However, results show that the effect of smoking may even have been less among brown-eyed individuals with melanin eye color pigmentation.

82–0153. Christen, A. G. **The Case Against Smokeless Tobacco: Five Facts for the Health Professional To Consider.** *Journal of the American Dental Association* 101(3): 464-469, September 1980.

Five facts concerning the use of smokeless tobacco are presented. First, smokeless tobacco is heavily advertised in today's mass media, with special emphasis directed toward male youth. Such advertisements often use professional athletes and entertainers to promote the products. Second, the tobacco companies through careful manipulation of the public have worked to make tobacco chewing and dipping more socially acceptable. The spitting that is concomitant with tobacco chewing is presented not as a disgusting habit but as part of the pleasure of smokeless tobacco. Advertisers also point out the allegedly lower prices of smokeless tobacco compared to cigarettes. Third, the use of smokeless tobacco is rapidly increasing, especially among male high school and college students and athletes. Sales of smokeless tobacco have increased appproximately 11 percent each year since 1974, with a total of about 22 million users in 1980. Fourth, advertising implies that smokeless tobacco products are "safe," although they are not. A person introduced to smokeless tobacco habits can easily become dependent on nicotine and may even switch to smoking. One common result of tobacco chewing and dipping is oral leukoplakia, a white lesion on oral tissues that is considered precancerous. It has a malignant transformation rate of between 3 percent and 5 percent. Smokeless tobacco is also associated with pharyngeal, laryngeal, and esophageal cancers. Fifth, smokeless tobacco has significant dental and oral effects on both soft and hard tissues. It is associated with bad breath, discolored teeth, and excessive wear or abrasion of the incisal and occlusal surfaces of teeth. Health practitioners are urged to advise their patients about the hazards of smokeless tobacco and to take a public stance in opposition to current advertising campaigns. The health professions should demand that smokeless tobacco products carry health warning labels similar to those printed on cigarettes.

82–0154. Clement, F. **Les Polyglobulies. [Polycythemias.]** *Schweizerische Medizinische Wochenschrift* 111(33): 1196-1201, August 15, 1981, French.

Classification of various known polycythemias (PC) is presented. Evolution, physiopathologic consequences, symptoms, diagnostic complications, prognosis, and treatment of PC are described, especially in the case of the primary polycythemia vera. Usually, the secondary hyposcic PC vera is observed in heavy smokers, when oxygen transport by hemoglobin is disturbed by carboxyhemoglobinemia. According to recent studies, spurious chronic PC is frequent and has a relatively high mortality rate and therefore, its treatment presents a problem. Certain authors believe that smoking cigarettes or cigars is the major cause of the spurious chronic PC (smokers' polycythemia). Physiopathology of this PC type involves carbon monoxide inhalation which either slightly increases or slightly decreases red cell mass.

82–0155. Council on Scientific Affairs. **Marijuana, Its Health Hazards and Therapeutic Potentials.** *Journal of the American Medical Association* 246(16): 1823-1827, October 16, 1981.

The health hazards of marihuana are discussed in relation to: ages and implications for users; pulmonary, cardiac, psychomotor, genetic, immune-response, and reproductive functions; therapeutic potentials; and implications of ongoing research. In comparing the hazards of marihuana usage to those of tobacco smoke, it is noted that marihuana has poor combustibility and therefore its smoke has up to 50 percent more polyaromatic hydrocarbons than tobacco smoke. Thus marihuana can cause more severe degenerative changes in lungs than tobacco. Also, marihuana is usually smoked down to a small butt, and the smoke is retained in the lungs for a longer period of time. One human study reported that less than one joint per day diminished vital capacity as much as 16 cigarettes daily. The combination of tobacco and marihuana smoking, a common practice, is probably additive in its pulmonary side effects.

82–0156. Hedin, C. A.; Ronquist, G.; Forsberg, O. **Cyclic Nucleotide Content in Gingival Tissue of Smokers and Nonsmokers.** *Journal of Periodontal Research* 16(3): 337-343, May 1981.

The total amount of cAMP (cyclic adenosine monophosphate) and cGMP (cyclic guanosine monophosphate) was determined in gingival tissue of smokers ($n=30$) and nonsmokers ($n=26$). The mean gingival tissue content from all patients was 1.80 pmol cAMP/mg protein and 0.56 cGMP/mg protein. There was no significant difference in nucleotide levels between smokers and nonsmokers. The 16 heavy smokers (>15 cigarettes/day, sample mean 19.7), however, were found to have significantly elevated nucleotide levels compared to the 14 moderate smokers (≤ 15 cigarettes/day, sample mean of 6.8). The heavy smokers had mean nucleotide levels of 2.30 pmol cAMP/mg protein and 0.65 pmol cGMP/mg protein, compared to 1.36 pmol cAMP/mg protein and 0.49 pmol cGMP/mg protein for the moderate smokers. Smokers also had a significantly lower gingival exudate flow compared to nonsmokers. The findings suggest that tobacco smoking may change the cellular metabolism in human gingival tissue. (Auth. Abs. Mod.)

82–0157. Kauffmann, F.; Brille, D. **Bronchial Hypersecretion, Chronic Airflow Limitation, and Peptic Ulcer.** *American Review of Respiratory Disease* 124(5): 646-649, November 1981.

A relationship was detected between peptic ulcers and smoking (and associated respiratory symptoms) in a study conducted on 1,049 men, of whom 7.1 percent reported a history of peptic ulcers. Age, social class, and country of origin were lifestyle factors that had no significant effect on the prevalence of ulcers in the study population. The smoking categories examined were nonsmokers, ex-smokers, moderate noninhaling smokers, moderate inhaling smokers, heavy noninhaling smokers, and heavy inhaling smokers. Men with ulcers were not more likely to smoke than the others, but were more often inhalers when they did smoke. The largest percentages of peptic ulcers in the smoking categories were among the inhaling moderate smokers (10.3 percent) and the inhaling heavy smokers (9.4 percent). The lowest percentages were among the nonsmokers (5.4 percent) and the noninhaling heavy smokers (1.3 percent). There was also a relationship between ulcers and chronic phlegm in all smoking categories. Among ulcer and nonulcer individuals, the percentage of those with chronic phlegm was often higher for heavy smokers and inhalers than for moderate smokers, noninhalers, and nonsmokers. The relationship between smoking and ulcers appeared to be greater in bronchial hypersecretors than in nonhypersecretors. Chronic airflow limitation was measured by FEV_1, which was not lower in the peptic ulcer patients, contrary to expectations, although the lower FEV_1 relationship was observed with inhalation and phlegm. The vital capacity was higher among the peptic ulcer subjects, even when adjusted for age, height, and weight.

82–0158. Lazaratou, H.; Moschovakis, A.; Armaganidis, A.; Kapsambelis, V.; Kiburis, J.; Kephalas, T. A. **The Pharmacological Effect of Fractions Obtained by Smoking Cannabis Through a Water-Pipe. II. A Second Fractionation Step.** *Experientia* 36(6): 1407-1408, 1980.

The catatonic activity, prolongation of phenobarbital sleeping time, convulsant action, and disruption of nest-building activity were assessed in mice subjected to four cannabis pyrolysis products and their tobacco analogs. All but one of the cannabis fractions prolonged the pentobarbital sleeping time and disrupted the nest-building activity of mice in a way not related to their content in the main cannabinoids. Nest-building activity seems to be the most valid assay used so far in examining behavioral disruptions. (Auth. Abs.)

82–0159. Yeomans, N. D.; Williams, D. R.; Mackinnon, M. A.; McLeish, A. R.; Smallwood, R. A. **Effect of Cigarette Smoking on Duodenogastric Reflux of Bile Acids.** *Australian and New Zealand Journal of Medicine* 11(4): 347-350, August 1981.

The possibility that smoking induces duodenogastric reflux was examined in 13 healthy male volunteers, but no apparent reflux was found from smoking one cigarette. Duodenogastric reflux of bile could injure gastric surface cells, and may be involved in the pathogenesis of idiopathic gastric ulcer. Gastric juice was aspirated for four consecutive 20-minute periods, and reflux quantitated by measuring total bile acids (TBA), chenodeoxycholic acid (CDCA), and glycocholic acid (GCA) in the juice. One cigarette was smoked during either period 2 or period 3. Amounts of bile acids (μmoles) refluxing into stomach during the presmoking, smoking, and postsmoking periods respectively were: TBA—4.5±1.1, 5.4±3.0, and 3.9±1.6; CDCA—1.7±0.6, 1.5±0.9, and 2.0±1.1; GCA—1.8±1.1, 1.1±0.6, and 1.0±0.6. Paired analysis revealed no significant effect of smoking on any of these parameters. The findings, based on quantitative methods, did not confirm those of previous unblinded and semiquantitative studies. Results appeared to indicate that smoking one cigarette did not provoke duodenogastric reflux. It was considered possible that smokers who do develop gastric ulcers comprise a subgroup who behave differently. It was also suggested that smoking several cigarettes in a row might provoke reflux. (Auth. Abs. Mod.)

See also, **82–0039**

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

82–0160. Ashley, M. J. **Smoking Habits, Knowledge, and Attitudes of Final Year University Students in the Health Professions.** *Preventive Medicine* 10(5): 645-654, September 1981.

A questionnaire survey was undertaken to determine the smoking habits of final year university students in the health professions, their knowledge about the health consequences of smoking, and their attitudes toward smoking control measures and specific professional roles. Students of medicine, nursing, dentistry, physical and occupational therapy, pharmacy, and physical and health education were studied. Students of arts and science, and applied science and engineering served as controls. Smoking was uncommon in all groups, the great majority of students never having smoked. Although knowledge about the relationship of smoking to specific diseases varied according to both the disease and the discipline, substantial proportions of all groups failed to appreciate the significance of this health hazard. Strong support for smoking control measures was lacking, as was a clear commitment to specific professional roles. These findings have implications for those involved in the education of students in the health professions. (Auth. Abs.)

82–0161. Canadian Tobacco Grower. **Smoking Habits of Canadians.** *Canadian Tobacco Grower* 29(6): 51, June 1981.

Results of a survey of Canadian smoking trends over a 15-year period (1965-1979) show a 9.9 percent increase in the number of nonsmokers, and a decrease of 8.6 percent in the number of regular smokers, and 1.3 percent in the number of occasional cigarette smokers and pipe and/or cigar only smokers. Between 1975 and 1979 the percentage of ex-regular smokers increased from 11.7 to 14.9 percent. Although the percentage of regular smokers who smoke cigarettes of less than 14 mg of tar increased by 12.4 percent between 1977 and 1979, the average number of cigarettes smoked per day also increased from 18.2 to 18.6. Further, it was found that in 1979, the highest percentage of smokers were 20 to 44 years of age, were from the Quebec region, and had 0 to 8 years education (males) or some secondary education (females).

82–0162. Chapman, S. **Understanding Cigarette Advertising—A New Approach to Anti-Smoking Education.** Department of Preventive and Social Medicine, Commonwealth Institute of Health, University of Sydney, Australia, Health Services Research and Teaching Paper No. 6, September 1981, 35 pp.

An Australian antismoking campaign is described, which uses an educational approach focusing on cigarette advertisements and their methods, as well as adopting some of the advertising's own principles to "unsell" smoking for schoolchildren. Adolescent smoking appears to be an antiauthoritarian gesture or a response to smoking's image as a glamorous or fun-filled activity. After a background description, the antismoking report discusses the methodological issues of cigarette advertising and smoking. Problems which hinder acquiring causal data include a lack of control populations unexposed to advertising; isolation of the effects of advertising from other factors; decision on whether advertising generated smoking or high smoking levels generated advertising; and the insensitivity of research methods dependent on respondents admitting to being influenced by advertising. A study was conducted to determine whether "demystifying" cigarette advertisements would reduce smoking prevalence in schoolchildren. Three sequential 40-minute lessons examined the mechanisms and promises of advertising. A high level of student participation was required. In the program's 16 randomly selected secondary schools, a questionnaire was administered before and again 8 months after. The expected patterns of brand preference were found: just four brands accounted for 78.7 percent of cigarettes smoked. Smokers had also been expected to be more familiar than nonsmokers with cigarette advertisements, which was found to be true. The suggestion that smokers would smoke those brands most familiar to them from advertisements and that those brands most heavily advertised would be the ones most smoked was generally borne out. The questionnaire results after 8 months indicated that although the program was considered interesting and enjoyable by teachers and students, it had no significant effect on the sample population. It is recommended that any future use of the program be either part of a social studies or mass communications course, or as one component of a wider health education program.

82–0163. Conway, T. L.; Vickers, R. R., Jr.; Ward, H. W.; Rahe, R. H. **Occupational Stress and Variation in Cigarette, Coffee, and Alcohol Consumption.** *Journal of Health and Social Behavior* 22(2): 155-165, June 1981.

The impact of occupational stress on self-reported cigarette, coffee, and alcohol consumption was investigated in a longitudinal field study. Substance consumption and subjective stress indicators were measured repeatedly for 34 men performing a job with known systematic variation in stress. Habitual cigarette smoking and coffee drinking were positively associated with chronic tendencies to perceive high stress; no associations were found between chronic alcohol consumption and stress perceptions. Consumption of all three substances varied significantly across days that differed in perceived stress level. On the average, there was more cigarette smoking and more coffee drinking, but less alcohol consumption, under high stress. These general effects of stress appeared to depend largely on the behavior of only a few of the participants, as the association between subjective stress indicators and substance consumption within individuals was not consistent across all of them. These findings suggest that there can be important individual differences in the tendency to increase or decrease habitual substance consumption in response to varying levels of stress. The individual differences may include factors such as: (1) individuals may differ in sensitivity to stress, the more sensitive persons producing stronger correlations; (2) people may differ in the way they change behavior to cope with stress (smokers may smoke more cigarettes or take more puffs per cigarette when under high stress); (3) people may differ in their mode of response to stress, e.g., some may smoke more cigarettes as stress increases, while others may drink more coffee; and (4) what may have begun as a response to stress may develop into a habit that is not responsive to stress, such as a person with a chronic smoking habit whether under stress or not. Such individual differences should be considered when constructing models that include behavioral responses to stress. (Auth. Abs. Mod.)

82–0164. Crutchfield, R. D. **An Examination of the Demographic, Mental Health, and Social Role Correlates of Adult Drug Use.** Doctoral Dissertation, Vanderbilt University, Nashville, University Microfilms International 80-18868, May 1980, 298 pp.

Drug use by normal adults—persons not under medication, and not using any combination of drugs where use disrupts functioning—was studied. The use of alcohol, cigarettes, tranquilizers, marihuana, sleeping pills, and stimulants is explored as a coping strategy in adult behavior. Results indicate that those who drink rarely use other stimulants; smokers tend to be multiple drug users; tranquilizers are most frequently used by middle-aged women; and those who use marihuana, tranquilizers, sleeping pills, and stimulants tend to use them in conjunction with at least one other drug. Cigarette use is negatively related to age, more common among men, lowest among the married, highest among the divorced, not related to income, and more common at lower educational levels. Those in poor mental health are more prone to drug use than those enjoying mental well-being. Tranquilizers tend not to be used in response to everyday difficulties, while cigarette use tends to be constant; and alcohol has elements of both patterns.

82–0165. Davidson, F.; Choquet, M. **La Notion de Risque, Issue de l'Etude des Deviances de l'Adolescent. [The Concept of Risk Resulting From the Study of Deviancy in an Adolescent.]** *Archives Francaises de Pediatrie* 37(Supplement, 1): 3-8, 1980, French.

Four epidemiological surveys were conducted among adolescents within a general population of French high schools, vocational colleges, and among young workers and youngsters hospitalized for suicide attempts. The purpose of these comparative surveys was an early detection of deviancy risks and discovery of the factors which have a deviancy predicting value. Personality profiles of the high-risk types of adolescents were obtained by multifactorial analysis of the epidemiological data. Regular heavy smoking was found to be one of the early signs of potential deviancy from a normal behavior. This conclusion was made on the basis of the findings that one high school student out of five used to smoke one or more cigarette packs/day versus one out of three suicide attempts and one out of two repeated suicide attempts. Among the 250 postal employees of the Paris region, aged 21 or younger, 21 percent of smokers of over 10 cigarettes/day, but only 5 percent of nonsmokers formerly took illicit drugs. Among the 250 students of high schools and technical colleges, 24 percent of smokers of over 10 cigarettes/day versus only 3 percent of nonsmokers used to take illicit drugs. Thus, a regular smoking habit may be considered as an indicator of a state which could evolve into a state more detrimental to an adolescent.

82–0166. Fisher, D. A.; Magnus, P. **"Out of the Mouth of Babes..." The Opinions of 10 and 11 Year Old Children Regarding the Advertising of Cigarettes.** *Community Health Studies* 5(1): 22-26, 1981.

One hundred and seventy-five 10- to 11-year-old children from five Australian primary schools were surveyed to determine the effects of cigarette advertising. Forty-five percent (79) of the children were from three schools which are situated in areas with a low socioeconomic level, a multiracial composition, and a higher than average proportion of children with physical, emotional, and learning problems. The remainder of the children (54.9 percent) were from schools in predominantly middle-class areas. Results show that the children feel cigarette advertisements are intended to lead people, including children, to take up smoking. The fact that the children identified the heavily advertised cigarettes as those most popular with adults and children appears to indicate that cigarette brand preferences of children are determined by advertising. The survey data also provide evidence of underestimation by the children of the hazard that cigarette smoking presents to life.

82–0167. Gallup, G. **Smoking Level Declines as More Perceive Health Hazard. Part Two.** Gallup Poll, August 31, 1981, 2 pp.

In the past decade, the percentage of cigarette smokers has declined in the United States from 43 percent in 1972 to 35 percent in the present survey, the lowest recorded level in 37 years of regular polling. As cigarette smoking declined, the percentage of those who believe there is a causal relationship between smoking and illness increased. In the current survey, 74 percent of those polled believe smoking is a cause of heart disease, compared to 68 percent in 1977 and 60 percent in 1969. Those who see a link between smoking and lung cancer comprised 83 percent of the current population compared to 71 percent in 1971. Those who favor stronger measures against smoking are also more numerous. In the current study, 46 percent of all persons surveyed believe Federal and State taxes on cigarettes should be increased, compared to 38 percent in 1977. A complete ban on cigarette advertising is supported by 43 percent, compared to 36 percent 4 years ago. A complete ban on cigarette sales is opposed by 74 percent overall (89 percent of smokers and 66 percent of nonsmokers).

82–0168. Gallup, G. **Percentage of Smokers Lowest in 37 Years. Part One.** Gallup Poll, August 30, 1981, 2 pp.

The percentage of smokers in the United States (i.e., those who smoked in the week preceding the survey) in 1981 is 35 percent, the lowest level recorded in 37 years of regular assessments. This reflects a continuing downtrend that began in 1972 when 43 percent were classified as smokers. The decline since 1972 was greatest among those in the 18 to 29 age group, declining by 17 points. A decline is also more pronounced among those with a college education than those with a high school or grade school educational background. The overall decline was 10 points for men and 5 for women. From 1977 to 1981, the proportion of heavy smokers (more than two packs per day) declined from 13 to 2 percent. The proportion of those who smoked one to two packs increased from 45 to 59 percent, while those consuming less than one pack decreased from 41 to 38 percent. Adults aged 18 to 29 were more likely to smoke less than one pack (50 percent) than those aged 30 to 49 (36 percent) or those 50 and older (30 percent). In 1978 and again in 1981, 66 percent of smokers indicated they would like to stop, with heavy smokers (more than one pack a day) being less likely to say they would like to quit. No less than 84 percent of smokers in 1977 and 83 percent in 1981 had given up smoking for at least 1 day. Those who quit for up to 1 week measured 28 percent in 1977 and 29 percent in 1981. Those who quit for 1 year or more totalled 15 percent in 1977 and 13 percent in 1981. In 1981, 22 percent of teenagers were smokers, similar to the figures for 1977 and 1979. Slightly more girls (24 percent) were smokers than boys (20 percent) in 1981. Teenage smokers tend to be light smokers compared to adults, with about two-thirds smoking half a pack of cigarettes a day or less.

82–0169. Gibinski, K.; Lelkowa, E.; Orzechowska, R.; Rys, M. **Charakterystyka Palaczy Tytoniu w Wojewodztwie Katowickim. [Characteristics of Smokers in the Katowice District.]** *Polski Tygodnik Lekarski* 35(14): 485-488, 1980, Polish.

Questionnaires were administered to adult employees of various industrial plants, health services, transportation, and schools to determine the smoking incidence in a highly industrialized region of Poland. A profile of the smoking population and its knowledge of and motivation for smoking is presented. The answers of 8,912 people revealed that 57 percent of the total population sample were smokers (67 percent of the men and 43 percent of the women). About 70 percent of smokers of both sexes were 20 to 40 years old, with a higher percentage of women than men. About 27 percent of the sample smoked more than 20 cigarettes/day. The last two findings point to a highly significant risk of smoking-related disease, particularly for young women. Among the smokers, 15.2 percent were not aware of the dangers of smoking compared to only 5.8 percent among nonsmokers. As to motivation for smoking, 17 percent of smokers "did not know why they smoke" and nearly 30 percent smoked because they "couldn't stop smoking." This fact indicates that antismoking campaigns may achieve a measure of success with many smokers. Difference in motivation for smoking was not significant between the sexes or between professional groups.

82-0170. Goldberg, S. R.; Spealman, R. D.; Goldberg, D. M. **Persistent Behavior at High Rates Maintained by Intravenous Self-Administration of Nicotine.** *Science* 214(4520): 573-575, October 30, 1981.

Previous studies have shown that long and orderly sequences of responding can be maintained by scheduled presentations of environmental stimuli that have been associated with intravenous injections of drugs such as morphine and cocaine. The schedules of reinforcement relating responding to consequent presentations of the stimuli and injections of drugs have been termed second-order schedules. In this study, squirrel monkeys pressed a lever at high rates under a second-order schedule of reinforcement in which lever pressing produced a brief visual stimulus that was occasionally contiguous with an intravenous injection of nicotine. The rate of lever pressing could be markedly reduced either by substituting saline for nicotine injections or by blocking the effects of nicotine with mecamylamine. The rate of lever pressing could also be reduced by eliminating the brief visual stimulus. These results show that nicotine can function as an effective reinforcer under a second-order schedule of drug self-administration and that an environmental stimulus associated with nicotine intake can contribute to the maintenance of persistent drug-seeking behavior. (Auth. Abs. Mod.)

82-0171. Harburg, E. **Handedness and Drinking-Smoking Types.** *Perceptual and Motor Skills* 52: 279-282, 1981.

Results of a survey of 1,153 men and women (10.3 percent left-handed), between the ages of 18 and 69, to establish differences in drinking and smoking habits due to handedness, are reported. The study population (which excluded siblings, past drinkers, and ex-smokers) consisted of the following groups: (1) never smoked or drank (17 percent of females, 8 percent of males); (2) drank, but did not smoke (52 percent of females, 44 percent of males); (3) smoked, but did not drink (1 to 2 percent for both sexes); and (4) both smoked and drank (29 percent of females, 47 percent of males). It was found that a higher percentage of sinestrals (left-handed) of both sexes both drank and smoked than did dextrals (right-handed): 60 percent of male sinestrals vs. 46 percent male dextrals; and 40 percent of female sinestrals vs. 28 percent female dextrals. There were also fewer abstainers among the sinestrals: 3 percent male sinestrals vs. 8 percent male dextrals; and 5 percent of female sinestrals vs. 18 percent of female dextrals. The greater use of alcohol and tobacco among sinestrals may be due to the stress of living in a right-handed world. Alcohol may be used to relax or to be "social," while smoking may be a response to tension.

82-0172. Henningfield, J. E.; Griffiths, R. R. **Cigarette Smoking and Subjective Response: Effects of *d*-Amphetamine.** *Clinical Pharmacology and Therapeutics* 30(4): 497-505, October 1981.

Administration of *d*-amphetamine was found to induce large increases in tobacco-product consumption and changes in subjective response in eight adult smokers, who had reported smoking an average of 26 cigarettes/day for an average of 15 years. One subject smoked a low-yield brand of 0.2 mg nicotine and 1 mg tar, while the others smoked moderate-strength brands with an average of 0.94 mg nicotine. Each smoker, acting as his own control, was administered four drug dose levels (0, 5, 15, and 25 mg) according to five randomized block sequences. Increased drug dosages resulted in an increase in the cigarettes smoked, puffs per smoking session, time spent smoking, cigarette duration, total burned tobacco, and satisfaction derived from smoking. There were dose-related increases in after-session expired air CO levels, indicating that the inhalation of smoke also increased. Mean cigarette butt weights and interpuff intervals were not affected by the drug doses. After each session the subject completed a short form of the Addiction Research Center Inventory (ARCI) and the Profile of Mood States (POMS). The ARCI questionnaire included scales sensitive to various classes of abused drugs, while POMS used scales for vigor, fatigue, and tension-anxiety. As the dose of *d*-amphetamine increased so did the score in the morphine-benzedrine group of the ARCI. Individual subject analysis indicated that the ARCI scales were less sensitive to *d*-amphetamine than behavioral measures of smoking. The drug doses had no effect on the tension—anxiety scale of POMS, suggesting that stress was not a factor in increased smoking. Two possible explanations are suggested for the increases: (1) *d*-amphetamine may induce stimulation or be antagonistic to the sedative effects of nicotine, and (2) *d*-amphetamine can act as a general behavioral stimulant that promotes smoking behavior. Interaction with nicotine may not be necessary for increased smoking to occur. The second explanation was strengthened by the fact that one smoker used low-nicotine cigarettes and another was a noninhaler whose expired air CO levels were not raised when smoking rates increased. It is suggested that *d*-amphetamine not be used during treatment intervention for smoking.

82-0173. Huba, G. J.; Wingard, J. A.; Bentler, P. M. **Intentions To Use Drugs Among Adolescents: A Longitudinal Analysis.** *International Journal of the Addictions* 16(2): 331-339, 1981.

Data on the actual and intended use of psychoactive substances, including cigarette smoking, were obtained from 1,634 students in the 7th, 8th, or 9th grade of Los Angeles area schools. All participants were recontacted 1 year later, and 1,177 (72 percent) responded. A confirmatory methodology tested whether the information contained in intentions to use drugs provided a sufficient increment over behavioral data in predicting future drug use. While the increment was statistically significant, the strength of the additional information was minimal. The results are interpreted as showing that little variance in adolescent substance use can be attributed to long-term cognitively purposeful behavior unique from previous drug-taking experience. It seems a reasonable conclusion that the initiation of habits such as smoking is not controlled by mental calculation of risk and benefits, but rather by more immediate factors such as role models or peer values. It may be more reasonable for health education programs to strengthen an individual's defenses against peer pressures and attitudes than simply to present the risks of smoking or drug habits. (Auth. Abs. Mod.)

82–0174. Johnston, L. D.; Bachman, J. G.; O'Malley, P. M. **1979 Highlights. Drugs and the Nation's High School Students. Five Year National Trends.** U.S. Department of Health and Human Services, Public Health Service, National Institute on Drug Abuse, DHHS Publication No. (ADM) 81-930, 1980, 80 pp.

In this, the third in a series of reports on drug use and related attitudes of high school seniors, some 74 percent of the seniors report having tried cigarettes at some time, and about 34 percent smoked to some extent in the month preceding the survey. Data were collected from seniors at 125 to 130 private and public high schools during the spring of each year, from 1975 through 1979. Statistics are tabularly reported for: the prevalence of drug use, recent trends in drug use, use at earlier grade levels, degree and duration of highs, attitudes and beliefs about drugs, and the social milieu. The 11 separate classes of drugs covered are: marihuana (including hashish), inhalants, hallucinogens, cocaine, heroin, natural and synthetic opiates other than heroin, stimulants, sedatives, tranquilizers, alcohol, and cigarettes. Also included, for the first time, are PCP and the amyl and butyl nitrites. Results related to smoking show: (1) cigarettes are used daily by more of the respondents (25 percent) than any of the other drug classes, with 17 percent saying they smoke half a pack or more per day; (2) 17 percent of the females and 15.4 percent of the males smoke half a pack or more per day; (3) 10 percent of the college-bound and 23 percent of the noncollege-bound smoke half a pack or more a day; and (4) a substantial and steadily increasing number of students think pack-a-day cigarette smoking involves great risk to the user.

82–0175. Kellner, H. **Das Schlechte Beispiel der Rauchenden Kollegen. [The Bad Example of a Smoking Colleague.]** *Munchener Medizinische Wochenschrift* 123(32/33): 36, August 7, 1981, German.

Examples of physicians and nurses smoking cigarettes in hospitals and clinics during visits to patients are often cited by the patients to justify their own smoking. Some established physicians are seen with a lighted cigarette, cigar, or pipe during office hours or visiting their patients. At a recent German Congress for Education of Physicians in Berlin, it was announced that a maximum 0.005 percent of the sanatorium patients were discharged because of smoking, while most of the smoking patients were only reprimanded by the physicians in charge. The end result is that a smoking patient with circulatory, peripheral, or pulmonary deficiencies gets a significantly prolonged paid vacation every second year instead of a 4-week cure. Often, the sanatorium patient who continues heavy smoking during treatment blames the sanatorium physician who was smoking himself and didn't enforce the smoking prohibition at the sanatorium.

82–0176. Lall, K. B.; Singhi, S.; Gurnani, M.; Chowdhary, B.; Garg, O. P. **An Epidemiological Study of Smoking in Urban School Boys of Ajmer.** *Indian Journal of Pediatrics* 47(388): 403-408, September-October 1980.

A survey of 1,000 school boys from 8 to 16 years of age revealed 52 (5.2 percent) to be smokers. Of the 30 who were regular smokers, 27 began smoking between 10 and 14 years of age. Only six children thought that they smoked for pleasure. None of the children whose mothers had university or higher vocational education smoked ($p<0.01$). Parental smoking also seemed to influence smoking behavior of the children. Type of family—nuclear or joint—did not affect smoking prevalence, but children occupying special position in the family smoked more frequently ($p<0.001$). Most of the smokers belonged to middle or poor socioeconomic groups and none to the upper socioeconomic group ($p<0.001$). (Auth. Abs.)

82–0177. Lynch, P.; Woodward, D.; Waters, M.; Kirk, I.; MacLean, A.; Rockcliffe, W.; Reid, P. **Smoking Behaviour of Tasmanian High School Pupils.** *Australian Paediatric Journal* 16(4): 255-259, December 1980.

A survey of smoking behavior and attitudes among Tasmanian high school students revealed that smoking is more prevalent among girls than boys in the 14- to 16-year-old age group. Girls at this age also smoke more heavily than boys. The mean age for experimentation was age 10 for boys and a year later for girls. By age 16, 80 percent of the pupils had at least tried cigarettes. An association was present between smoking behavior and attitudes toward health outcomes of smoking. The sample of 646 boys and 565 girls was drawn from 14 Tasmanian high schools.

82–0178. Maisto, S. A.; Caddy, G. R. **Self-Control and Addictive Behavior: Present Status and Prospects.** *International Journal of the Addictions* 16(1): 109-133, 1981.

The application of self-control techniques to the treatment of obesity, smoking, and alcohol and drug abuse is reviewed. Although the literature suggests a commonality in the etiological and maintenance factors of these addictive behaviors, self-control techniques have not been applied equally in treatment. Unlike the alcoholic, obese individual, and drug user, the smoker traditionally has not been considered to be suffering from a lack of will power, possibly because smoking apparently does not have the same immediate or short-term negative effects. It is hypothesized that this differential application of self-control procedures can be accounted for by an examination of three sets of variables: pharmacological properties of the substance abused, models of the behaviors under investigation, and social consequences of abusing a specific substance. The pharmacological property of particular interest is the capacity of the substance to induce physical dependence. It is generally accepted that nicotine ingestion leads to dependency, but the abstinence syndrome associated with nicotine is not considered severe. Explanations for addictive behavior include a range of sociological or biological factors. Currently there is no dominant model to account for smoking behavior. Smoking has had less social consequences than some of the other addictive behaviors. Traditionally, public attitudes have generally accepted the use of nicotine, though there appears to be an increasing trend to restrict smoking in public places. It is suggested that if models of addictive behavior and the procedures that stem from these models are revised to take into account the range of internal and external stimuli that affect an individual's continued use of a substance, then maintenance and generalization of behavior change occurring during treatment would be more likely. Self-control procedures may be advantageous if they induce the client to develop behavior modulation strategies which can theoretically be independent of any therapist or situation. (Auth. Abs. Mod.)

82-0179. Newman, I. M.; Duryea, E. J. **Adolescent Cigarette Smoking and Tobacco Chewing in Nebraska.** *Nebraska Medical Journal* pp. 243-244, October 1981.

A higher percentage of Nebraska adolescents smoke, and predict they will still be smoking in 5 years, than is found in the national sample. In a 1980 survey of 2,616 Nebraska youths (12 to 18 years of age), 14.0 percent of the girls and 13.5 percent of the boys were smokers. Of these, 88.3 percent of boys and 86.5 percent of the girls reported smoking daily. In the 17- to 18-year-olds, the percentages of smokers were 19.3 for boys and 26.2 for girls, who tended to smoke longer cigarettes, while the boys reported higher use of "dangerous" cigarettes. Lowest rates were found among farm youth, followed by town/city youth. Highest rates were reported by nonfarm rural youth. Over 95 percent of all cigarettes smoked by both boys and girls were filtered. A few of the adolescents reported chewing tobacco; and although use is increasing, it does not appear to be a new fad for this group.

82-0180. Office of Population Censuses and Surveys. **General Household Survey. Cigarette Smoking: 1972 to 1980.** *OPCS Monitor* 7 pp., July 28, 1981.

General Household Survey figures in the United Kingdom show an overall decline in smoking prevalence from 1972 to 1980. Among men, the proportion of total current smokers dropped from 52 percent in 1972 to 42 percent in 1980. Among women, the proportion declined from 41 percent in 1972 to 37 percent in 1980. The overall decrease between 1972 and 1980 reflected a decrease in the proportions of male and female light smokers (fewer than 20 cigarettes a day). The proportion of male heavy smokers (20 or more cigarettes a day) also decreased between 1974 and 1980, but the proportion of female heavy smokers remained fairly constant over the same period. General prevalence and consumption patterns between different age groups did not greatly alter from 1972 to 1980. Prevalence and consumption continued to be lower than average among the young and elderly of both sexes. Between 1976 and 1980, consumption per male smoker declined in most age groups, while consumption among women remained at about the same level. Smoking prevalence continued to be higher in 1980 for both male and female manual worker groups than nonmanual groups. The fall in smoking prevalence between 1972 and 1980 was most pronounced among professional groups. Between 1972 and 1976, average weekly cigarette consumption increased for male and female smokers in all socioeconomic groups. Between 1976 and 1980, consumption declined among male smokers in all socioeconomic groups except the unskilled manual group where consumption remained constant. Among female smokers, changes in cigarette consumption between 1976 and 1980 were too small to suggest any recent trends within particular socioeconomic groups.

82-0181. Ossip, D. J.; Epstein, L. H.; McKnight, D. **Modeling, Coffee Drinking, and Smoking.** *Psychological Reports* 47(2): 408-410, October 1980.

A group of undergraduate smokers (n = 30) was examined for the relative effects on cigarette smoking of coffee and of the presence of a smoking model. Of these, 15 were classified as light smokers (5 to 15 cigarettes/day) and 15 were considered heavy smokers (16 or more cigarettes/day). All subjects drank at least one cup of coffee per day. A between-groups design was used. Presentation of coffee or water and presence of a smoking or nonsmoking model were varied over a 1-hour period. The three conditions tested were: water plus nonsmoking model, coffee plus nonsmoking model, and water plus smoking model. Ten subjects (five heavy smokers and five light smokers) were tested in each condition. Subjects were permitted to smoke freely during the 1-hour period. The smoking rate was not significantly affected by coffee or modeling. The mean number of cigarettes smoked for subjects in the water—nonsmoking model, coffee—nonsmoking model, and water—smoking model groups were 1.70, 1.10, and 1.80 cigarettes, respectively. The lack of significant findings from the coffee and smoking model variables may be because the subjects and models were specifically instructed not to interact with each other, but the present investigation does suggest that the effects of coffee and modeling on smoking may not be as great as might have been expected from previous research in the field.

82-0182. Philip, L. **Morphological Constitution and Smoking Behaviour.** *Indian Journal of Public Health* 24(2): 59-63, April-June 1980.

Two body measurements, height and weight, and one bodily ratio, weight/height^2, were used to study morphological characteristics in relation to smoking status. The subjects were of the administrative, technical, and managerial elite (social class I) of Calcutta, India; 182 men of all ages, categorized into light, medium, and heavy smokers and nonsmokers. Although decimal differences occurred, there was no significant difference in any of the variables between any of the smoking categories. This was possibly due to the small number of subjects studied and to lack of age differentiation.

82-0183. Radius, S. M.; Dielman, T. E.; Becker, M. H.; Rosenstock, I. M.; Horvath, W. J. **Health Beliefs of the School-Aged Child and Their Relationship to Risk-Taking Behaviours.** *International Journal of Health Education* 23(4): 227-235, 1980.

A survey of 224 schoolchildren in Washtenaw County, Michigan, aged 6 to 17, was conducted to determine the students' perspective on health concerns and whether such concerns affect their behavior. Prior use of cigarettes was admitted by 30 percent of the sample, of which 25 percent identified themselves as current smokers. Even the youngest children had tried cigarettes and were anticipating their future use. More males (34.7 percent) than females (24.3 percent) reported experimenting with cigarettes, but current use among all those who had ever smoked was higher for females (30.8 percent) than males (21.4 percent). Analysis of the data indicated that current cigarette smoking was somewhat related to the measures of children's health beliefs. Age had the strongest impact on risk-taking behavior, with smoking being more prevalent among older youths. Other characteristics affecting the profile of current smokers included having a relatively low level of general health concern, being female, being more preventively oriented, believing greater severity to accompany illness, and perceiving relatively greater parental concern for their health. It is suggested that health educators continue to stress the relationship between health-risk behaviors such as smoking and its ramifications.

82-0184. Schmidt, D.; Diallo, A. D.; Tiendrebeogo, H.; Roudaut, M. **Enquete sur la Consommation Tabagique en Milieu Africain a Abidjan. [Smoking Habits of Africans in Abidjan.]** *Poumon-Coeur* 37(2): 87-94, 1981, French.

A questionnaire about smoking habits was administered in 1977 to a representative sample of 500 black residents of Abidjan, Ivory Coast, of both sexes, aged over 15 years. The residents were selected in four urban districts, based on their income evaluated according to the type of dwellings. Among those surveyed, only 24.4 percent were smokers, mostly of cigarettes. The average age of the smokers, almost exclusively men, was 27 years. The average daily consumption was 12.7 cigarettes per smoker, with 82.6 percent of the smokers using less than one pack per day. The highest percentages of smokers were found among craftsmen or merchants (43 percent) and employees (31 percent). The great majority (69.7 percent) of the smokers lived in a slum district (45.6 percent) or in the urbanized district (apartments) of the city. Significantly, more smokers were found among single than among married subjects. Only 33.6 percent of the smokers inhaled smoke. More smokers of affluent districts than those of poor districts (92.3 vs. 26.5 percent) were aware of smoking risks. It is concluded that the young and the lower socioeconomical class are the two population groups which must be targets of preventive and educational antismoking measures.

82-0185. Shor, R. E.; Williams, D. C.; Shor, M. B. **An Investigation of Reported Symptoms and Attitudes on Tobacco Smoke Pollution as a Function of Expositional Context, Smoking Status, and Gender.** *Addictive Behaviors* 6(4): 271-282, 1981.

A social support theory of smoking behavior was examined in this questionnaire study of 691 University of New Hampshire undergraduates who reported on symptoms from tobacco smoke pollution and pertinent attitudes about smoking. The three independent variables were the explicit versus nonexplicit expositional context of the nonsmokers' rights movement, smoking status (current smokers versus nonsmokers), and gender. Moderate to extremely high similarity was found in the patterns of relative mean magnitudes on all three variables. Significant, although in some instances very small, effects were found on all three variables in regard to both symptoms and attitudes, and a significant interaction of expositional context and smoking status was found in regard to symptoms. However, the essential patterns of findings remained stable across contexts. Alternative explanations for the context effect and interaction and directions for further research are discussed. (Auth. Abs.)

82-0186. Spence, V.; Ho-Yen, D. O. **Puff Volume Increases When Low-Nicotine Cigarettes Are Smoked. (Letter).** *British Medical Journal* 283(6293): 730-731, September 12, 1981.

A previous comment (*British Medical Journal* 283(6289): 498, August 15, 1981) questioning an earlier article (*British Medical Journal* 283(6285): 187-189, July 18, 1981), which concluded that smokers who switched to cigarettes that deliver lower nicotine yields took larger puffs, thereby maintaining nicotine levels while increasing exposure to combustion products, is refuted. It is stated that while the original conclusion may be unjustified from the evidence presented, the reasoning seems consistent with the observations of patients with peripheral vascular disease, who usually smoke 20 to 40 cigarettes a day and who are told either to stop smoking, to change to a brand with a lower tar:nicotine ratio, or to reduce the number of cigarettes smoked. Smokers tend to be reluctant to switch to a lower nicotine yield; and of those who do, many feel "unsatisfied" and do not persist with the change. Those who succeed only in reducing their smoking are often unable to reduce consumption to much below 10 cigarettes a day and admit to inhaling more deeply, retaining the smoke longer, and smoking more of each cigarette, if possible. It is suggested that in patients with cardiovascular disease such changes in smoking behavior may be lethal by increasing carboxyhemoglobin levels in the blood. Since many cardiovascular patients cannot stop smoking, it is important to ascertain the risks of changing brands or reducing the number of cigarettes smoked, with the concomitant change in smoking behavior. See also 81-1946 and 81-1932.

82–0187. Sutton, G. C.; Stock, S. L. **Passive Smoking and Lung Cancer. (Letters).** *British Medical Journal* 282(6265): 733-734, February 28, 1981.

Two responses to Hirayama's paper (*British Medical Journal* 282: 183-185, January 17, 1981) that concluded that passive smoking increases one's risk of lung cancer are presented. The original findings were that the risk of lung cancer from passive smoking is dose-related, and is about a third to a half that of active smoking. Sutton feels that if that is correct, the effect of smoking would be compounded from three sources: one's spouse, one's workmates, and oneself. This is based on findings that smokers tend to marry smokers, work among smokers, and congregate in smoking areas of movies, trains, etc. Further, it is suggested that if passive smoking does present a high risk, then a large element of the risk of active smoking is in rebreathing one's own fumes in a smoky room, whereupon one could conclude that more powerful ventilation or smoking out of doors could perhaps reduce lung cancer mortality. Stock, on the other hand, supports Hirayama's findings, and adds that the degree of exposure necessary to produce an effect requires urgent attention, taking into account differences in frequency of the smoking habit among spouses and environmental surroundings. He recommends that further studies measure the risk to nonsmokers with increased precision and that smoking in public be controlled. See also 81-1088.

82–0188. Vertes, L. **Rauchen und Hoheres Lebensalter. [Smoking Among Elderly People.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 156(3): 272-276, 1981, German.

The importance of the problem of smoking among the elderly has been increasing because of the steadily growing old-age fraction of total population and the increase in smoking by elderly people. In Hungary alone, people over 60 make up 18.8 percent of total population. This fact in turn increases the importance of elderly people as role models for young people. The common trend in old people to develop several diseases simultaneously (multiple morbidity), primarily cardiovascular diseases, requires the consideration of smoking as an important risk factor of these diseases. It is stated that individual counseling is the most important method of health education of elderly smokers. In addition, small group therapy and written information (pamphlets, brochures) may be helpful. Abstinence from smoking or, in certain cases, reduction of smoking is indicated as helpful in treating multiple diseases of old age. The rising trend toward isolation of old people also contributes to their smoking as their only pleasure, thus offering a bad example to youth. Intervention of government and social organizations is believed necessary to make nonsmoking the generally accepted social norm of behavior.

82–0189. Weinberger, M.; Greene, J. Y.; Mamlin, J. J.; Jerin, M. J. **Health Beliefs and Smoking Behavior.** *American Journal of Public Health* 71(11): 1253-1255, November 1981.

Smoking histories and beliefs about smoking were obtained for 120 ex- and current smokers. Ex-smokers (n = 42) viewed smoking as a serious health problem, did not report having a physician recommend they stop, and felt personally susceptible to its adverse effects. The 33 moderate smokers (1-10/day) also viewed smoking as serious, but did not see themselves as vulnerable to its adverse effects. The data suggest that certain attitudes can discriminate between groups of current smokers, as well as smokers from ex-smokers. (Auth. Abs.)

82–0190. Wilcock, A. R.; Milsum, J. H.; Laszlo, C. A.; Varga, L. E. **Evaluation of Relative Health-Risk Levels of a Group of Impaired Drivers Through Health Hazard Appraisal.** *Canadian Journal of Public Health* 72(4): 264-268, July-August 1981.

The observation that persons convicted of impaired driving show evidence of multiple lifestyle hazards was confirmed by comparing test and control groups for significant differences in lifestyle variables. It was found that the test group (90 persons convicted of impaired driving) had diminished life expectancy; higher risk of death due to smoking and alcohol-related disease; drove more miles per year but used seat belts less often than the controls; and, in general, pursued a more hazardous lifestyle. Data were obtained through a Health Hazard Appraisal (HHA) of both groups, and mortality risk and composite risk factors were determined. Smoking cessation or reduction was found to be of similar significance to alcohol as a recommendation for risk reduction in the test group. Other factors were far less significant. It was concluded that the HHA, when subjects are assured of confidentiality, can be a useful tool in the context of a defensive driving course.

See also, **82–0213**

SMOKING PREVENTION AND INTERVENTION

82-0191. Brooks, J. (Editor). **Introducing Tobacco Education in the Elementary School K-4.** Kent, Ohio, American School Health Association, 1978, 20 pp.

A Tobacco Education Curriculum for kindergarten through fourth grade, developed by the American School Health Association, is presented. The goal of the curriculum is to teach the relatedness of intellectual, physical, emotional, and social well-being, emphasizing the potential for a full and meaningful life. General information on tobacco and health education, suggested classroom activities, and social concepts involved in smoking behavior are discussed. A resource paper with a more detailed description of the immediate and long-term effects of smoking on health is provided, which includes a partial listing of the chemicals found in tobacco smoke. Suggested agencies to contact for further information and a bibliography of relevant publications on smoking and health are also included.

82-0192. Bureau of Drug Education, New York State Education Department. **Guidelines for Development of School Policies Regarding Smoking.** University of the State of New York, State Education Department, Bureau of Drug Education, Albany, New York, 1980, 53 pp.

A new tobacco education curriculum, distributed by the New York State Department of Education to all schools in the State, is described. The curriculum is designed to help teachers meet the requirements of the tobacco education law requiring school programs which discourage the use of tobacco by developing desirable health attitudes. The curriculum includes separate booklets for kindergarten through sixth grade, junior high, and senior high school, and can be used directly by the teacher. There is also a book of guidelines for the development of school policies regarding smoking, designed to facilitate clarifying the school's role in smoking education, containment, enforcement, and modification of smoking behavior. The policy booklet covers topics such as laws and regulations, smoking surveys, current literature, peer approach to smoking education, and smoking cessation programs.

82-0193. Chu, F. Z.; Day, R. G. **Smoking Recognition by Family Physicians.** *Journal of Family Practice* 12(4): 657-660, April 1981.

The frequency with which physicians recognize the smoking habits of their patients was investigated among 22 family practice residents and 10 faculty members in a two-part study begun in the fall of 1979. Subjects were categorized as smokers, nonsmokers, or smoking status unknown. A smoking patient was considered recognized if the physician made any reference to smoking in the progress notes and/or if smoking was mentioned as a problem on the patient's chart. The initial smoking recognition rate by family physicians was 18 percent. An educational program was presented to all study participants, consisting of two 1-hour conferences; and antismoking and smoking cessation literature was made available to patients and physicians. A followup study, conducted 1 year later, showed a significant improvement in the smoking recognition rate (51 percent), particularly for smokers with medical conditions known to predispose them to higher mortality and morbidity, including respiratory and cardiovascular conditions, hypertension, diabetes, pregnancy, and use of birth control pills. This result suggested that the physicians behaved in a more disease-oriented than preventive manner, and that large numbers of smoking patients were still not recognized. It is suggested that family physicians need to become better prevention specialists and that they document the smoking habits of all their patients before trying to counsel or intervene with smoking cessation programs.

82-0194. Dawley, H. H., Jr.; Finkel, C. **The Great American Smokeout: A Follow-Up Report.** *Addictive Behaviors* 6(2): 153-154, 1981.

In a telephone sample of participants in the Great American Smokeout (November 15, 1979), 18 percent of those who stopped had not returned to smoking at the 2-month followup. The Great American Smokeout is an annual event operated by the American Cancer Society in which people are solicited to sign a pledge stating that they will not smoke on the day of the smokeout. Telephone surveys by the American Cancer Society for last year's smokeout suggest that about one-third of the nation's smokers participated, with about one-third succeeding in making it through the day without smoking. This year's telephone survey indicated that 66 percent of those who signed up actually participated, and of those, 73 percent succeeded in not smoking for that day. (Auth. Abs. Mod.)

82-0195. Denson, R.; Stretch, S. **Prevention of Smoking in Elementary Schools.** *Canadian Journal of Public Health* 72(4): 259-263, July-August 1981.

A successful Canadian smoking prevention program in six schools, which characterized adult smoking as not only destructive to physical health, but also as a form of drug addiction, is discussed. The addiction hypothesis was considered of special value since it made adult behavior (a parent who smoked warning about the dangers of smoking) more comprehensible. Knowledge of the mechanisms of drug addiction apparently allowed children to see smoking as a weakness, rather than as being associated with strength, maturity, and independence. When study results were compared to those from six control schools, the recruitment to smoking rate over a 2-year period (beginning of seventh grade to the end of eighth grade) was 4.0 percent for program schools and 17.5 percent for control schools. The four program sessions, which combined films, slides, lectures, and discussion, are outlined and the questionnaire administered to all students is reproduced. It was concluded that, unlike other short courses on the prevention of smoking onset which have been ineffective, emphasis on the addictive aspect of smoking in this study was a factor in the significant reduction in smoking onset. Total teaching time for the program was under 5 hours.

82-0196. Evans, R. I.; Rozelle, R. M.; Maxwell, S. E.; Raines, B. E.; Dill, C. A.; Guthrie, T. J.; Henderson, A. H.; Hill, P. C. **Social Modeling Films To Deter Smoking in Adolescents: Results of a Three-Year Field Investigation.** *Journal of Applied Psychology* 66(4): 399-414, 1981.

The effectiveness of a social-psychological strategy to deter the onset of frequent smoking in junior high school students is evaluated. A smoking deterrence strategy was developed within a social-learning and persuasive-communication framework consisting principally of a series of films. In the films, detailed techniques for coping with social influences (peers, parents, media) on smoking are depicted. Dependent measures of knowledge gained from the films, of intentions to smoke, and of smoking behavior utilizing a novel nicotine-in-saliva analysis were administered. Students ($n = 1,352-3,296$) in 13 junior high schools were assigned to three experimental and four control groups. Seventh- through ninth-grade smoking deterrence interventions and dependent measures were administered in the experimental schools, and the dependent measures only were administered in the control schools. At the end of the eighth and ninth grades, analyses of the data revealed that the experimental group subjects smoked less frequently and intended to smoke less frequently than did the control group subjects, and that knowledge generated by the films was significantly related to smoking intention and behavior. (Auth. Abs.)

82-0197. Flagle, J.; Wheeler, J. **Helping Teenagers Become Nonsmokers. Summary Report.** Division of Health Education, Center for Health Promotion and Education, Centers for Disease Control, U.S. Public Health Service, September 1981, 14 pp.

Five programs designed to prevent smoking in adolescents by helping students resist peer pressure to smoke, providing nonsmoking role models, changing attitudes about the supposed rewards of smoking, and educating youth about the hazards of smoking are reviewed. The five programs examined are: (1) the antismoking component of "Body Power," a health curriculum for sixth graders developed by the Chicago Heart Association; (2) Counceling Leadership About Smoking Pressures (CLASP) created for seventh graders by Stanford University; (3) a life skills training program developed for seventh graders by the American Health Association; (4) pregnant teenagers education program developed at the University of Minnesota; and (5) the New Hampshire Lung Association's School Health Education Program (SHEP). All programs include use of films and biofeedback equipment to emphasize the immediate and long-term health effects of smoking. Although quitting smoking is encouraged, the programs are prevention-oriented; four of the five focused on life skills training, such as resisting peer pressure, decisionmaking, interpersonal communications, and environmental awareness, with one of the four especially designed for pregnant teenagers. The fifth program is designed around health and scientific methodology and the experimental process, for older, achieving students.

82-0198. Griffiths, A.R.B.; Jost, S.; Smith, D.; Rogers, F.; Cole, R. J. **No Smoke Without Advertising. (Letters).** *New Scientist* 89(1235): 94, January 8, 1981.

A series of letters are presented in reply to a previous article (*New Scientist* 88(1231): 691, December 11, 1980) advocating a tax on tobacco advertisements in Great Britain, with revenues to be spent on antismoking advertising. The first letter warns that specific taxes are too often lost in the general taxation system and, further, that administrative overhead would be needed to collect the special taxes. Instead, it is suggested that for each tobacco advertisement, the advertiser finance equal space and time for antismoking advocates. The second letter acknowledges a conflict between the freedom to advertise and public health benefits, but states that the value of improved public health should have precedence, and suggests increased taxation on tobacco products, increased public education on smoking, and widespread advertising against smoking. The third letter argues the proposal may establish an unwise precedent leading to other selective taxes, such as a tax on alcohol to finance alcoholism warnings. It is also suggested that stopping tobacco advertising might have little effect on smoking in Great Britain. For example, cigarette advertisements are not allowed in Italy and the U.S.S.R.; yet consumption there has risen during most of the past 40 years. The fourth letter notes the adverse association between maternal smoking and fetal and child health such as birth defects and early life cancers. See also 81-2047.

82-0199. Kim, S. **How Do We Know Whether a Primary Prevention Program on Drug Abuse Works or Does Not Work?** *International Journal of the Addictions* 16(2): 359-365, 1981.

A primary prevention program is defined as one which attempts to keep infrequent, irregular, or curiosity-oriented drug experimenters (such as smokers) from becoming regular users. A program named Ombudsman was implemented from Spring 1975 to Fall 1977 among students ($n > 1,000$) who were mainly in Grades 5 and 6. The program, presented as a 50-minute session twice a week for 14 weeks each semester, has three phases: (1) self-awareness, (2) group skills, and (3) a students' project within their community or school. A survey was then conducted among students ($n = 14,117$) in Grades 5 through 12 asking if the participants had taken Ombudsman and if they still engaged in behavior such as smoking. The primary prevention program seemed to have greater effectiveness with "hard" drugs that are less socially accepted than with "social" drugs such as cigarettes. The proportion of Grade 5 students who answered that they no longer used cigarettes was 11.6 percent for Ombudsman recipients vs. 5.2 percent for non-Ombudsman students, indicating the program had some effectiveness against the smoking habit. Positive results were also found in grades 7 through 9, with the respective figures being 16.8 percent vs. 15.0 percent (Grade 7), 19.4 percent vs. 18.5 percent (Grade 8), and 20.6 percent vs. 18.1 percent (Grade 9). This was not, however, the case in the other grades. For Grade 6, the proportion of students who answered that they no longer used cigarettes was 9.7 percent for Ombudsman recipients vs. 11.5 percent for non-Ombudsman students. The respective figures were 9.3 percent vs. 19.5 percent in Grade 10, 13.2 percent vs. 16.4 percent for Grade 11, and 10.4 percent vs. 14.2 percent for Grade 12.

82-0200. McAlister, A.; Puska, P.; Koskela, K.; Pallonen, U.; Maccoby, N. **Psychology in Action. Mass Communication and Community Organization for Public Health Education.** *American Psychologist* 35(4): 375-379, April 1980.

A nationwide program to facilitate smoking cessation conducted in Finland, through cooperation between psychologists and public health researchers in the United States and Finland, is described. The program consisted of a series of televised counseling sessions and an effort to organize volunteer-led, self-help viewing groups, which were included as part of the program because it was felt that televised counseling efforts without social support or interaction would not provide cues for action: passive viewers might participate only cognitively, learning from the program but not applying it to themselves. The television shows (seven sessions of about 45 minutes each) were broadcast nationwide in February or March of 1978, each program featuring a group of 10 voluntary smokers. Two leaders gave direct counseling to the group and to the television audience. In the province of North Karelia, special community efforts and self-help groups were organized with one community leader per 300 to 400 smokers. Local actions were not consistently organized in other provinces of Finland. A survey of panels of smokers from North Karelia (n=258) and the neighboring province of Kuopio (n=294) showed that North Karelia had almost twice the viewership and program participation. The percentages of actual reported success in the two provinces, however, did not differ significantly at a 1-month or 6-month followup, perhaps because of the small sample sizes. Study data suggest that about 40,000 adult smokers may have actively participated and that about 10,000 (approximately 1 percent of the smokers in Finland) achieved at least 6 months of cessation. If the estimate is accurate, then the results were unusually cost effective, with about 1 dollar spent for each 6-month "success." (Auth. Abs. Mod.)

82-0201. McGuire, W. J. **Behavioral Medicine, Public Health and Communication Theories.** *Health Education* 12(3): 8-13, May-June 1981.

A seven-step strategy for designing a public health communications campaign against the smoking habit and other health risks is discussed. The first step is to analyze the current health concerns to determine whether: (1) a particular problem is sufficiently severe, (2) a corrective or coping behavior is available whose benefits outweigh any negative side effects, and (3) such corrective behavior can be promoted by a communications campaign. Since smoking is a self-inflicted health risk, urging smokers to stop has high priority, although a communications professional might suggest a campaign would be more effective in preventing nonsmokers from starting. The second step is to use one's own sense of ethics to decide means and effects of the campaign which will create more benefits than costs. Third, the sociocultural context should be examined for factors that will help or impede the campaign. Pressures to smoke and procedures for resisting them should be examined. Fourth, there should be psychological mapping and surveying of the matrix of attitudes, feelings, and actions that instigate and sustain smoking behaviors. The fifth step involves analyzing the information acquired from the previous steps in order to choose the most promising targets and techniques for the campaign. Constructing persuasive communications is the sixth step. The target population may be more effectively persuaded if they receive the message singly rather than in a group, and by lay persons similar to themselves, rather than by experts. The seventh step is to evaluate the campaign's effectiveness at each successive step. The health communications professional must act effectively in several scientific and social fields to develop a successful antismoking campaign.

82-0202. Miller, G. H. **SOS Stop Smoking Clinic: A One-Year Report on the Program at the Cummins Engine Company.** *Journal of the Indiana State Medical Association* 74(5): 292-294, May 1981.

Of 37 smoking employees introduced to the SOS Stop Smoking Clinic on August 20, 1979, 33 decided to attend the clinic, which consists of up to 15 sessions, with lectures on the adverse effects of smoking, selected films, guest speakers, talks by former smokers, group counseling, and individual counseling. The participants met for 10 sessions during the first 3 weeks, followed by 5 reinforcement sessions. These occurred after a 1-week interval, a 2-week interval, and three 1-month intervals. Measurements were taken for carbon monoxide exhalation to determine if the individual participant had actually stopped smoking. Persistent smokers did not attend the reinforcement sessions. Most health professionals recognize a 1-year period without smoking as the criterion for success in a stop smoking clinic. Year-end results indicated that 6 of the 33 participants (18 percent) were back to full-time smoking; 9 (27 percent) were still trying to quit or were occasional smokers; and 18 (55 percent) reported that they were not smoking. Smoking had been resumed by five members during the last few months of clinic attendance, with three indicating that reinforcement sessions would have made the difference in their not returning to smoking. Weight gain, cited as the main factor by one member who reported smoking again, was felt to be a contributing factor by three others. Those most likely to be unsuccessful were those who had smoking wives, had been to hypnotists, or had used nicotine reduction devices. A 55 percent success rate compares favorably with a more common rate of 10 to 30 percent, but additional reinforcement sessions are recommended for future clinics. A company that sponsors successful stop smoking clinics will reportedly gain economically from less absenteeism, fewer cases of sickness, and increased productivity.

82–0203. Pinney, J. M. **Warning Citizens About the Hazards of Smoking: Where We Are in 1981.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 241-245.

Despite the Surgeon General's report issued 16 years ago on the health hazards of smoking, tobacco remains a major industry in the United States. In 1965, 977 million pounds of cigarette tobacco were grown, a figure that rose to 1.8 billion pounds in 1980, showing a profit of 1.6 billion dollars in 1977. Two changes have been occurring in the U.S. tobacco industry, however. First, the domestic market is getting smaller, as more people quit and fewer take up the habit. To counteract this trend, tobacco companies are increasing their exports to foreign countries. Second, low-yield cigarettes have become popular, with those yielding 15 mg of tar or less making up almost half the current U.S. domestic market. Government intervention has been minimal; a 1969 law banned cigarette advertising on television and radio and required warning labels on cigarette packages, but only modest amounts have been spent on antismoking education. The Federal tax on cigarettes remains at its 1952 level and tobacco price supports are still in place. State governments raised cigarette taxes in the late 1960s and early 1970s, but the trend seems to have slowed. State and local governments have also passed regulations concerning smoking in public places. In 1980, the Public Health Service reviewed the health consequences of low-yield cigarettes and of tobacco additives such as flavor enhancers, concentrating on the relevance of past information: how well machine-measured tar and nicotine levels correspond to actual intake by the smoker; whether tar and nicotine alone are adequate indicators of hazard (some 4,000 compounds are known in cigarette smoke), and the varying effects of switching to low-yield cigarettes on the risks of developing disease. At present, the Public Health Service advises that health hazards may be reduced by switching to low-yield cigarettes (provided smokers don't compensate by smoking more often or more deeply), although smoking even the lowest yield cigarettes presents health hazards greater than not smoking at all.

82–0204. Puska, P.; Neittaanmaki, L.; Tuomilehto, J. **A Survey of Local Health Personnel and Decision Makers Concerning the North Karelia Project: A Community Program for Control of Cardiovascular Diseases.** *Preventive Medicine* 10(5): 564-576, September 1981.

The North Karelia project was established to carry out a comprehensive community program for control of cardiovascular diseases (CVD) in North Karelia, a county in eastern Finland. The objectives were immediate reduction of known CVD risk factors, high blood pressure, serum cholesterol, and smoking. The intervention activities were integrated with the county's health services and social organizations. Health personnel were involved in antismoking and nutritional health education activities. Public health nurses were more active in smoking intervention than were physicians. Survey results for the 5-year period reported the following net reductions in the proportion of smokers: 15 percent for physicians, 6 percent for public health nurses, 6 percent for decisionmakers, and 4 percent for males and 9 percent for females in the general population.

82–0205. Reed, C. E. **What Are Schools Doing To Discourage Cigarette Smoking?** *Health Education* 12(2): 16-17, March-April 1981.

A randomized questionnaire survey of 451 freshmen (196 males, 255 females), at Central Michigan University, to determine what high schools are doing to discourage smoking, is reported. Approximately 19 percent of the sample were smokers (compared to a national norm of 20 percent), and 6 percent were ex-smokers. Based on responses, it was concluded that: (1) there is a serious omission of planned experiences within the curriculum of most high schools regarding the effects of smoking on health; (2) very few high schools are helping prepare teachers to teach the facts which are consistent with current scientific findings about human health and smoking; (3) in many high schools, teachers, visitors, and students are still permitted to smoke in the buildings; (4) more than half of all high school students are being taught to smoke by the example of one or both parents; and (5) about half of the students believe the general atmosphere of the school discouraged more than encouraged smoking. Whenever a program is to be implemented at all levels in a school system, the impetus must come from the policy makers and the policy enforcers; therefore, it is recommended that school boards initiate policies to discourage cigarette smoking among students. These policies should provide for instruction on the effects of smoking, and should prohibit smoking on school property.

82–0206. Spitz, J. B. **Curbing Cigarette Smoking. (Letter).** *New England Journal of Medicine* 305(16): 962-963, October 15, 1981.

A reader expresses agreement with a previous editorial regarding ways to curb cigarette smoking (*New England Journal of Medicine* 304(24): 1486-1487, June 11, 1981). Special reference is made to the suggested prohibition of smoking in hospital rooms. Patients would be accustomed to doing without cigarettes, possibly allowing them to leave the hospital as nonsmokers. Conversely, the American Medical Association (AMA) is criticized for holding stock in tobacco companies; it is suggested the stock be sold. An editorial reply notes the AMA has completed the sale of its 1.4 million dollars of stock in R. J. Reynolds Industries and Philip Morris. See also 81-1675.

82–0207. Stencel, S. **Anti-Smoking Campaign.** In: Gimlin, H. (Editor). *Editorial Research Reports on National Health Issues.* Washington, D.C., Congressional Quarterly Inc., 1977, pp. 85-102.

Spurred by the activism of groups such as GASP (Group Against Smokers Pollution) and ASH (Action on Smoking and Health), the battle between smokers and nonsmokers is getting hotter. Since 1973, 30 States and the District of Columbia have enacted laws that ban smoking in public places such as offices, production lines, buses, post offices, and supermarkets. The claim that tobacco smoke is a health hazard to nonsmokers has been questioned by the tobacco industry, and many smokers claim antismoking laws are an infringement of their rights. Antismoking campaigns have existed continuously since tobacco was introduced to the civilized world over 400 years ago (Czar Michael of Russia cut off offenders' noses). Recent controversy has centered on the health consequences of smoking, the final turning point being the 1964 Surgeon General's Report, which linked smoking to coronary artery disease, bronchitis, emphysema, heart disease, and lung cancer. Most Americans were shocked; and sales dropped sharply, only to rise again as the shock wore off. Since then, pressures have increased on the cigarette industry: advertising is limited; tar and nicotine levels and health warnings must be posted; and research is ongoing to develop safer cigarettes for those who can't quit.

82–0208. Tones, B. K. **Health Education: Prevention or Subversion?** *Royal Society of Health Journal* 101(3): 114-117, 1981.

Four philosophical approaches to health education for smoking and other risk factors are presented: educational, preventive, radical, and self-empowerment. The educational approach involves the exploration and clarification of relevant beliefs and values with the client audience, allowing the client to choose freely; however, many people find it difficult to make a such a choice about smoking, due to social pressures and parental role modeling. The need to persuade or motivate people to modify smoking behavior underlies the preventive approach to health education, which assumes that prevention is better than cure, and that smoking is involved in the etiology of modern disease. The stages of the prevention approach are: (1) to prevent onset of disease or to detect it in the asymptomatic stage; (2) to prevent the development of disease and to reverse the process where possible, through early diagnosis, compliance with therapy, and utilization of medical services; and (3) rehabilitation or acceptance of impending death. The radical approach considers the roots of health problems such as smoking to be social, economic, and political factors, with poverty and disadvantages being major correlates of ill health. It can be argued that apparent government reluctance to alienate tobacco manufacturers or to endanger tax revenues indicates lack of concern about tobacco-related disease rates. The self-empowerment approach may be able to avoid the passivity of the educational, the coercion of the preventive, or the posturing or subversive action of the radical approaches, by creating a genuinely free and informed choice. It includes promoting attitudes for deferring present immediate gratification (such as smoking) in favor of future benefit; challenging beliefs that life and health are controlled by others; and promoting self-esteem which would help in responding to health education and in resisting pressures for unhealthy practices such as smoking. The four approaches to health education do not need to be separate and, in fact, often overlap in practice.

82–0209. Wynder, E. L.;Hoffmann,D. **Tobacco and Health: A Societal Challenge.** In: Finger, W. R (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 247-266.

For over a quarter of a century tobacco smoking has been associated with a variety of illnesses, such as cancers and heart disease. A three-point approach is suggested to address this social problem: youth antismoking programs to prevent people from starting the habit, smoking cessation programs to help current smokers quit, and less harmful cigarettes for those who continue to smoke. For adolescents faced with the problem of peer group pressure in favor of smoking, programs can adopt the informational approach to improve students' knowledge about smoking and its risks; but such programs often have minimal impact on smoking behavior. Programs that adopt social and psychological approaches, teaching students to cope with the factors and pressures that could lead to smoking, appear more promising. Antismoking programs aimed at adult smokers have been more successful than those directed at children and teenagers. Of the traditional approaches to smoking cessation (hypnosis, individual and group counseling, and strategies derived from learning theory), group therapy may have better long-term results than hypnosis or individual therapy. In the development of less harmful cigarettes, reduction of tar, nicotine, and carbon monoxide may be accomplished by elimination of certain tobacco ingredients before cigarette manufacture, selection of specific tobacco varieties, or various methods of smoke filtration or dilution. For the individual who achieves total cessation of smoking, a rapid reduction in the risk of myocardial infarction may be expected, and a more gradual reduction in the risk of lung cancer. It is recommended that in the future: antismoking efforts include more large-scale prevention programs; school health programs be coordinated nationally; smoking cessation techniques and therapies be more readily available; and all such activities receive increased governmental support.

See also, **82–0169, 82–0178**

SMOKING CESSATION METHODS

82–0210. Beaver, C.; Brown, R. A.; Lichtenstein, E. **Effects of Monitored Nicotine Fading and Anxiety Management Training on Smoking Reduction.** *Addictive Behaviors* 6(4): 301-305, 1981.

An experimental smoking reduction program found that combining gradual reduction in cigarette nicotine yields with an additional program to manage any resulting anxiety or stress tended to be less effective than a program of reducing nicotine only. Nicotine fading is a procedure whereby smokers change weekly to progressively lower nicotine content brands. Anxiety management teaches subjects to use relaxation instead of smoking as a response to stress. Subjects (28), recruited through radio and newspaper ads, had smoked brands containing at least 0.7 mg nicotine at the rate of pack/day for at least a year. Subjects were divided into high and low anxiety groups, matched according to daily nicotine intake, and randomly assigned to the nicotine fading only or nicotine fading plus anxiety management programs. The subjects met once a week for 6 weeks. Followup assessments were carried out at 1-week, 1-month, and 6-month intervals. Results showed that subjects who received anxiety management did not do as well as the nicotine fading only subjects, the poorest results being from the high anxious-anxiety management group. At the 3-month followup, 6 of the 8 subjects (26 responding) who reported abstinence were from the nicotine fading only group. At 6-month followup, 4 subjects (25 responding) were still abstinent, of whom 3 (out of 11) were from the nicotine fading only group. Of the 21 subjects who were smoking at the 6-month followup, 18 were smoking a reduced nicotine yield cigarette, median reduction 0.6 mg. Subjects did not appear to increase their smoking rate to compensate for reduced nicotine intake. It is suggested that the ineffectiveness of the anxiety management program might be due to overly complex, inappropriate, or poorly executed methods.

82–0211. Carosone, G. **Cigarette Holder.** United States Patent No. 4,275,745, June 30, 1981, 4 pp.

A cigarette holder for assisting a smoker in stopping smoking is described. It has an outer tube whose rear end is provided with a mouthpiece and whose front end is provided with a metal snuffing collar. Inside this outer tube is another tube that acts as a stop for a cigarette inserted into the outer tube, and that is provided with break formations such as grooves or notches so that it can be shortened. This inner tube is therefore periodically shortened so that the amount of cigarette that can be inserted or received within the holder and in back of the snuffing collar increases. The inner and outer tubes may be made of a heat-resistant synthetic resin, the outer tube being made of a transparent resin if desired. The extinguishing collar may be made of metal or any high heat content and heat conductive substance. When the cigarette burns down to the collar or ring, the cigarette is snuffed out. The amount of each cigarette that can be smoked with the holder can be decreased step-wise to allow the user to reduce the amount of each cigarette smoked. Ultimately cigarette smoking can thus be stopped. (Auth. Abs. Mod.)

82–0212. Dobler-Mikola, A.; Gmur, M.; Angst, J. **Erfolgsdeterminanten bei der Behandlung von Rauchern durch den Handaufleger Hermano. [Determinants of Success in the Treatment of Smokers With the Hermano Therapy.]** *Archiv fuer Psychiatrie und Nervenkrankheiten* 230(1): 31-40, 1981, German.

A 1973 smoking cessation treatment program (532 subjects) employing the Hermano method of chirothesia (laying on of hands) is evaluated. The subjects' smoking behavior, social background, and personality were documented before treatment, and correlated with followup information obtained through questionnaires 4 months and 5 years posttreatment. Statistical analysis showed the highest success rates with moderate smokers, but little success with addicted smokers or those classified as neurotic. Social and demographic variables (age, civil status, sex) and personality characteristics together contributed only 12 percent to prognosis of treatment success, with neuroticism the only apparently relevant personality characteristic. No specific indicators were found for the successful application of the Hermano therapy method.

82–0213. Evans, D.; Lane, D. S. **Smoking Cessation Follow-Up: A Look at Post-Workshop Behavior.** *Addictive Behaviors* 6(4): 325-329, 1981.

Results of a long-term followup from nine smoking cessation workshops indicate that compared to smokers, ex-smokers are more likely to avoid other smokers, and to cope with smoking urges by denial or by thinking of personal reasons for stopping rather than using distractions or substitute rewards. Of the 590 participants in the workshops, 67 percent (372) responded to the survey. The smoking cessation rates declined to an average of 25 percent by the first year post-workshop, and remained relatively constant thereafter; however, only 67 percent of those not smoking at followup had abstained continuously from workshop to followup. In addition to the coping techniques used by ex-smokers to maintain abstinence, it was noted that ex-smokers were less likely to follow workshop recommendations to change habits or routines associated with smoking such as coffee breaks. It is suggested that early confidence of success could have reduced participants' willingness to change smoking associated habits, or that the change in the associated habits induced a further sense of deprivation and encouraged a relapse. No apparent association was found between not smoking and other compliance or situation variables, or with sociodemographic variables, habituation, health beliefs, or commitment to quitting. It is concluded that further analysis of the techniques used by successful ex-smokers may be useful.

82–0214. Fichera, A. T. **Tobacco Smoking Inhibitor.** United States Patent No. 4,276,890, July 7, 1981, 10 pp.

A method of inhibiting or destroying the desire to smoke tobacco products is presented, which comprises administering for an extended period of time a gamma pyrone selected from the class of maltol and ethyl maltol and in a quantity initially of not less than about 500 milligrams per day. The reduction appears to be spontaneous and does not seem to be accompanied by any typical symptoms of nicotine withdrawal. By incorporating a maltol in relatively large amounts in gum, candy, or other physiologically acceptable media, the desired amount of maltol can be taken by reasonable daily consumption of such media. Releasing the maltol in the mouth at least 10 minutes for about 12 to 15 times per day is important. A lozenge or troche that lasts over 10 minutes in the mouth and releases ethyl maltol at a rate in excess of about 0.2 mg/min is preferred. When this is done the pyrone should be administered at more than about 75 mg/day. The treatment may be maintained for as little as 10 days, although 20 to 30 days is preferred. The rate of reduction of smoking during treatment can vary, due to individual variations in nicotine susceptibility and in responsiveness to gamma pyrone, as well as psychological and social factors. (Auth. Abs. Mod.)

82–0215. Glasgow, R. E.; Schafer, L.; O'Neill, H. K. **Self-Help Books and Amount of Therapist Contact in Smoking Cessation Programs.** *Journal of Consulting and Clinical Psychology* 49(5): 659-667, October 1981.

Two self-help behavior therapy books for stopping smoking, *Become an Ex-Smoker,* by B. G. Danaher and E. Lichtenstein (See 80-0834) and *Break the Smoking Habit. A Behavioral Program For Giving Up Cigarettes,* by O. F. Pomerleau and C. S. Pomerleau (See 78-0370), were evaluated against a widely available minimal treatment program, which utilized the *I Quit Kit,* published by the American Cancer Society. A 3 x 2 factorial design was employed to evaluate the three treatments under both self-administered and therapist-administered conditions. Eighty-five chronic cigarette smokers participated in the 8-week treatment programs and a 6-month followup. Dependent variables included self-monitored number of cigarettes smoked daily, carbon monoxide concentrations in expired breath samples, dosage scores based on topographical aspects of smoking behavior, and adherence and cost-effectiveness measures. Under self-administered conditions, the minimal treatment program was at least as effective as the behavior therapy books. On the self-report measures, therapist contact was associated with improved treatment outcome for the behavior therapy programs but not for the control condition. Reliable between-groups differences were not consistently observed on carbon monoxide levels. It is not known how well the study's results would apply to other populations and conditions of testing. In particular, there are significant differences between the study's self-administered treatments and the way such programs are acquired in bookstores or libraries. The study's results suggest that a two-stage process be used for smoking cessation, starting with a self-administered program and followed, if necessary, by behavior therapy administered by a therapist. (Auth. Abs. Mod.)

82–0216. Lowe, M. R.; Green, L.; Kurtz, S.M.S.; Ashenberg, Z. S.; Fisher, E. B., Jr. **Self-Initiated, Cue Extinction, and Covert Sensitization Procedures in Smoking Cessation.** *Journal of Behavioral Medicine* 3(4): 357-372, December 1980.

Two studies which tested the utility of self-control procedures and covert sensitization as alternatives to rapid smoking in smoking cessation are described. The surfeit effect of the rapid smoking procedure can give encouraging results in antismoking programs, but poses possible health risks. Self-control and covert sensitization (CS) procedures rely on symbolic aversive stimuli rather than actual aversive stimuli such as rapid smoking. In CS, subjects imagine smoking behavior accompanied or followed by aversive images such as nausea, fear, or bodily harm. A program was initiated in which the subjects were at least 21 years of age and had smoked at least 1 pack/day for at least 5 years. Relaxation training and discussion of goals were given in three meetings per week for 2 weeks prior to subject-chosen quit dates. A total of 10 sessions were held during the 90 days following cessation. In Study I, both groups received self-control strategies; one group also received CS. At 3-month followup, 33 percent of those without and 27 percent of those with CS were abstinent. At 6-month followup these percentages were 33 and 13, respectively. CS apparently added nothing to the effects of the self-control package. In Study II, one group received the same combination of self-control manual and the basic package as in Study I; a second group received the basic package with cue extinction procedures designed to extinguish associations between desires for cigarettes and cues paired with previous smoking; and a third group received a combination of both sets of procedures. At 3-month followup, 71 percent and 60 percent respectively of the first two groups and only 31 percent of the combination group were abstinent. At 6-month followup, the percentages were 29, 27, and 8, respectively. The low abstinence rate for the combination group may reflect problems associated with presenting too much material in the 2 weeks prior to cessation, since too many procedures or ideas may dilute the impact of each component. Although the long-term abstinence rates appear low, they may be judged more favorably if the great costs of smoking-related illnesses are borne in mind. (Auth. Abs. Mod.)

82–0217. Martin, G. P.; Waite, P.M.E. **The Efficacy of Acupuncture as an Aid to Stopping Smoking.** *New Zealand Medical Journal* 93: 421-423, June 24, 1981.

Results of a study involving 405 subjects suggests that acupuncture can help between 5 and 15 percent of the population to stop smoking for at least 6 months. According to classical acupuncture, the exact placing of the needles is important. Using chronically implanted press needles, however, the study showed that previously reported "effective" auricular acupuncture points for smoking cessation were not better than "placebo" auricular points. Additional electroacupuncture on the hand neither increased the probability of stopping smoking nor enhanced the reduction in smoking at 3-week, 3-month, or 6-month followup periods. It is concluded that the effectiveness of acupuncture in the cessation of smoking is the result of a large psychological component. (Auth. Abs. Mod.)

82–0218. Ockene, J. K.; Nutall, R.; Benfari, R. C.; Hurwitz, I.; Ockene, I. S. **A Psychosocial Model of Smoking Cessation and Maintenance of Cessation.** *Preventive Medicine* 10(5): 623-638, September 1981.

The long-term cessation rates obtained in smoking control programs have been disappointing. The behavior pattern of smoking goes through the stages of initiation, transition, maintenance, and termination or cessation. Initiation may be promoted by psychological factors such as self-esteem, social needs, and other personal needs. During transition the external social factors become less important, giving way to intrinsic psychological motivation. The habit is maintained when it becomes an intrinsic part of the person's life, becoming dependence-producing or addictive. To provide a better understanding of the problems of smoking cessation and maintenance of cessation, and to facilitate the development of more effective smoking control programs, a model of smoking cessation is offered in which stress, or "life events," is a major determinant of success. The model assumes that chronic smoking is a maladaptive behavior, helping the smoker to deal with the discomfort generated by stress by providing a means he can use to maintain a "vital balance." A smoker's ability to handle stress without cigarettes is conditioned by the presence of personal and social resources or "psychosocial assets." Personal security, an individual's belief that he can control what happens to him, an ability to respond to stress with a low level of negative effect, and the availability of social supports provide a cushion or buffer against the effects of stress. Therefore, the combined effect of stress and the psychosocial variables determines whether or not a smoker can successfully eliminate smoking from his repertoire of coping responses. (Auth. Abs. Mod.)

82–0219. Ogle, J. **The Stop Smoking Diet.** New York, M. Evans and Company, Inc., 1981, 168 pp.

The Stop Smoking Diet which assumes that nicotine is the addictive component of cigarettes is detailed. Diets containing acid-forming foods such as meats, poultry, and fish encourage faster elimination of nicotine from the system, while diets with base-forming foods such as vegetables, fruits, and milk products result in a slower release of nicotine. The base-forming smokers' diet (including limited amounts of grains) is intended to prevent weight gain and help the smoker to quit. In addition, a Fresh Start Diet for smokers who have quit allows more meat, poultry, fish, eggs, and grains, but is still moderate in calories. Nicotine alternatives suggested as aids to smoking cessation include dry snuff, moist snuff, chewing tobacco, nicotine chewing gum, and a pipe or cigar if not inhaled. A Stop Smoking Plan for quitting the habit includes the Stop Smoking Diet. It is concluded that for cessation of smoking success, an individual must be motivated, in addition to such aids as dieting, nicotine alternatives, and social and psychological supports.

82–0220. Owens, M. V.; Samaras, J. T. **Analysis of the Damon Smoking Control Program. A Study of Hypnosis on Controlling Cigarette Smoking.** *Journal of the Oklahoma State Medical Association* 74(3): 65-70, March 1981.

A study of 468 smokers (average, 35 cigarettes/day), who voluntarily attended a single hypnotherapy session of the Damon Smoking Control Program, is described. Cassette tapes were made available as a reinforcement tool. Questionnaires were used in evaluation interviews 6 to 9 months after the group hypnotherapy. An overall quit rate of 27.53 percent (n=128) was achieved, while 53.33 percent had not changed their smoking status, 15.05 percent were smoking less, and 4.09 percent were smoking more. Of the 128 who quit, 40 had smoked for more than 21 years, 24 for more than 31 years, and 7 for over 41 years, indicating that even those who have smoked for a long time can be motivated to quit. Of those who quit, 63.57 percent had smoked more than one-and-a-half packs/day. A positive relationship was found between the number of cigarettes smoked daily and smoking status after attending the hypnotherapy session. A majority of those who quit (22.07 percent of the total number of smokers) were between 25 and 54 years old. Of the 399 who stated they had tried to quit before, 111 were able to quit after attending the program. In rating the program, 160 termed it "very successful," 85 "moderately successful," 94 "not very successful," and 117 "quite unsuccessful." The quit rate for males was 32.99 percent compared to 23.42 percent for females. Only 14 of those who quit used a cassette tape as reinforcement. It is concluded that additional sessions might increase the number of nonsmokers since over half of the respondents expressed such a desire. It is also apparent that the individual must be motivated, at least enough to attend an antismoking session.

82–0221. Paxton, R.; Scott, S. **Nonsmoking Reinforced by Improvements in Lung Function.** *Addictive Behaviors* 6(4): 313-315, 1981.

Early improvement in lung function was found to reinforce abstinence behavior among 42 subjects in a smoking cessation program. Participants in a Smoking Clinic were divided into a Success group and a Failure group. The Failure group (5 men and 10 women) had smoked again within 8 weeks of the stop smoking date. The Success group (16 men and 11 women) had not smoked during the same period. Self-reports of abstinence were verified by random collection of urine samples for nicotine analysis. Lung function was measured for FEV_1 (volume of air produced in the first second of forced exhalation), and FVC (total volume of air yielded by a forced exhalation). Measurements were taken before cessation of smoking, weekly during the first month without cigarettes, and at set intervals thereafter. The participants were informed of the test results and told whether they had improved after each test. The Success group showed greater improvement in FEV_1 during the first 3 weeks than the Failure group. It was not clear whether the verbal feedback after each test session or improved fitness was responsible, but it is concluded that those with improved lung function were probably influenced by both effects. Future investigation of this question will monitor lung function without informing subjects of the results.

82–0222. Poole, A. D.; Sanson-Fisher, R. W.; German, G. A. **The Rapid-Smoking Technique: Therapeutic Effectiveness.** *Behaviour Research and Therapy* 19(5): 389-397, 1981.

The effectiveness of the rapid-smoking technique in terms of two measures of outcome—posttreatment abstinence rate and reduction in smoking achieved by nonabstinent subjects—is examined. A total of 75 volunteers (40 men and 35 women) participated in the treatment program and were randomly allocated to one of four conditions: rapid-smoking alone; rapid-smoking and relaxation training; rapid-smoking, relaxation, and contingency contracting; or contingent rapid-smoking. No significant differences between treatment conditions were obtained. For the total sample, abstinence was achieved by 64 percent of the subjects immediately following treatment; but the probability of remaining abstinent 12 months later was only 0.33. Immediately after treatment the nonabstinent subjects had reduced their rate of cigarette consumption to 34.2 percent (SD=29.3) of baseline smoking, but by 12 months average consumption had increased to 82.4 percent (SD=28.3) of baseline. It is concluded that rapid-smoking gives rise to substantial short-term changes in smoking behavior, but these effects are not sustained. (Auth. Abs.)

See also, **82–0202, 82–0223**

TOBACCO PRODUCT ADDITIVES

82–0223. Corbett, L. O. **Anti-Smoking Device.** United States Patent No. 4,269,203, May 26, 1981, 5 pp.

A device is described for injecting an unpleasant odoriferous substance into cigarettes contained within a package, which conditions the smoker to regard smoking as an unpleasant experience. One problem with persuading people to stop smoking is that some regard it as a pleasant experience. The smoker may enjoy the tobacco aroma, smoking may provide a sense of well being, or it may reduce anxiety. The present invention includes a hollow carrying case with an upper chamber for receiving a package of cigarettes and a lower chamber containing the odoriferous substance. Hollow needles are arranged to extend from the lower chamber into the upper chamber for penetration into the cigarettes. The cigarettes are penetrated as the package is inserted into the upper compartment. Fewer needles are present than cigarettes in the package, they are arranged in an irregular pattern, and have different lengths. As a result, some cigarettes remain untreated while the odoriferous portions of the treated cigarettes will vary. The foul smell and uncertainty associated with such treated cigarette packs would hopefully have a negative psychological effect, discouraging the smoker from smoking, for fear of smoking a "treated" cigarette. (Auth. Abs. Mod.)

82–0224. Sprecker, M. A.; Hall, J. B.; Schmitt, F. L. **Benzodioxanones and Organoleptic Uses Thereof.** United States Patent No. 4,294,266, October 13, 1981, 16 pp.

Use of selected bicyclic compounds for the organoleptic enhancement of smoking tobacco products is described. The compounds provide novel sweet, spicy, vanilla-like, woody, hay, tobacco-like aromas before smoking, and sweet, spicy, hay, tobacco-like aromas during smoking. The aroma characteristics may be varied and controlled to produce the desired uniform flavoring characteristics. The compounds may be added to tobacco or to suitable substitutes such as dried lettuce leaves. Other flavor and aroma additives may also be added either separately or in mixture with the compounds, including selected synthetic materials or natural oils. The compounds plus any extra additives may be added to the smoking tobacco material, the filter, the leaf itself, or the paper wrapper. A proportion by weight of 250 to 1,500 ppm of the selected compounds in the smoking tobacco material is recommended. The compounds may be applied by spraying or dipping the tobacco material; by brushing or rolling it on the paper or leaf wrapper; or by spraying, dipping, or coating the filter.

82–0225. Wilson, R. A.; Schreiber, W. L.; Mookherjee, B. D.; Kiwala, J.; Vinals, J. F.; Vock, M. H.; Stork, G.; Schmitt, F. L. **2,6,6-Trimethyl-α-(Iso)Propenyl-1-Cyclohexene-1-Methanols and -1,3-Cyclohexadiene-1-Methanols and Organoleptic Uses Thereof.** United States Patent No. 4,278,098, July 14, 1981, 31 pp.

Selected compounds which provide novel smoking tobacco compositions having sweet-musty, hay/tobacco-like, fruity-raspberry, ionone-like aromas before smoking and fruity-tobacco-like, Virginia tobacco-like aromas and tastes during smoking are described. These effects may be controlled and maintained at a desired uniform level before and during smoking, regardless of tobacco blend variations or the nature of the filter. In addition to these aroma and flavor additives, other selected synthetic materials or natural oils may be added to the smoking tobacco or suitable substitute (such as dried lettuce leaves). The aroma and flavor additives may be added to the smoking tobacco material, to the paper or leaf wrapper, or to a filter which is part of the smoking article. They may be applied by brush or roller on the paper or leaf wrapper of the smoking product or to the filter by either spraying, dipping, or coating.

See also, **82–0226**

TOBACCO MANUFACTURING AND PROCESSING

82–0226. Baskevitch, N. **Continuing Upward Trend in Low Tar and Nicotine Cigarettes.** *Tabak Journal International* 4: 304-306, 308, 310, August 1981.

The cigarette industry has devoted considerable research and development to reducing tar and nicotine levels to meet a growing demand while still maintaining desirable organoleptic properties. Those who supply cigarette paper, filter wrapping papers, and reconstituted tobacco have a role in this endeavor. A cigarette paper having increased natural porosity will allow greater dilution of smoke. This results in a reduction of tar and nicotine, as well as of carbon monoxide. Combustion salts such as monoammonium phosphate, sodium and potassium citrate, sodium acetate, potassium nitrate, or potassium and sodium tartarate may be added to cigarette papers to modify the taste of smoke and to affect combustion. Dilution systems have been developed to promote ventilation at the filter level, which also reduces amounts of nicotine, tar, and carbon monoxide. Reconstituted tobacco may also be produced with a highly porous structure and a satisfactory burn rate, reducing the tar and nicotine levels. Although the possibilities for yield reduction are more limited in plain, unfiltered cigarettes, modified cigarette paper and reconstituted tobacco made with the paper process may be used. Additional ways of reducing yields include employing genetically selected natural tobaccos, simple or multiple filters, and expanded tobaccos. Development of low-yield cigarettes with satisfactory taste implies the use of several techniques, each used in moderation.

82–0227. Becker, J. J. **Tricyclic Lactones.** United States Patent No. 4,273,714, June 16, 1981, 4 pp.

Selected tricyclic lactones to be used as perfume and flavor agents for tobacco are described. The lactones are 10,10-dimethyl-4-oxa-tricyclo (7.1.1.$0^{3,8}$) undec-3 (8) -en- 5 -one and 10,10- dimethyl - 4-oxa-tricyclo (7.1.1.$0^{3,8}$) undecan- 5 -one. The perfume or aroma of the compounds are characterized as reminiscent of tobacco, honey, or tonka beans, being sweet, herb-like, and slightly spicy. The compounds produce sweet, flowery, woody, herbal, or coumarin-like flavors. A level of 100 ppm to 200 ppm is recommended for aromatization of tobacco products; however, greater or lesser amounts may also be used.

82–0228. Becker, J. J.; Ohloff, G. **Tricyclic Lactones.** United States Patent No. 4,271,076, June 2, 1981, 4 pp.

Novel tricyclic lactones which are useful perfume and flavor agents are described. The lactones presented are 10,10-dimethyl-3-oxa(7.1.1.$0^{2,7}$)undec-2(7)-en-4-one and 10,10-dimethyl-3-oxa-tricyclo(7.1.1.$0^{2,7}$)undec-4-one. The perfume or aroma qualities are described as being sweet, herb-like, and slightly spicy, reminiscent of tobacco, honey, and tonka beans. The flavors are characterized as sweet, flowery, woody, herbal, or as resembling coumarin. To achieve organoleptic enhancement, recommended levels of use are 100 to 200 ppm of the weight of the tobacco material. Higher or lower proportions may be used, however, especially when more particular effects are desired.

82–0229. Conrad, L. J.; White, J. L. **Process and Apparatus for Expanding Tobacco.** United States Patent No. 4,270,553, June 2, 1981, 17 pp.

A process for expanding tobacco is described, during which tobacco is impregnated with an expansion agent and rapidly heated in the presence of hot inert solid particles in a fluidized state, bringing about greater tobacco expansion than that achieved by previous processes. The preferred expansion agent would be an organic compound essentially chemically inert to the tobacco and able to rapidly vaporize or expand at process temperatures. Expansion agents can include selected aliphatic hydrocarbons and corresponding unsaturated hydrocarbons, aliphatic alcohols, ketones, cycloaliphatic hydrocarbons, or halogenated hydrocarbons. Inert solid particles may include beads of ceramic materials, metals, alumina, silica, or similar materials, with dimensions between 0.3 and 2 millimeters. The particles should not give rise to unfavorable taste or aroma in the tobacco, and should be stable at relatively high temperatures. Their total weight should be about 3 times the dry weight of the tobacco and have a temperature between 100°C and 300°C at the time of contact with the impregnated tobacco. To avoid charring, the tobacco should be kept in the heating zone for 2 minutes or less, depending on the temperature of the particles. Tobacco expansion usually occurs within a short time after initial contact with the hot solid particles.

82–0230. Eicher, T.; Muller, F. **Smokable Products.** United States Patent No. 4,296,762, October 27, 1981, 8 pp.

The described smokable products, based on cellulose and combustion-modifying substances, yield a reduced content of harmful substances in the smoke and/or improved organoleptic properties. The smokable products contain: (1) 0.1-10 percent by weight, based on the total weight of the smokable product, of finely divided solids. Their average particle diameter is less than 2 μ, in a distribution which is heterogeneous over the cross section of the smokable product, with an enrichment in or on the surfaces; and (2) 0.5-15 percent by weight, based on the total weight of the smokable product, of salts of monobasic or polybasic carboxylic acids. The salts may be simple or complex, including the ammonium, alkali metal, alkaline earth metal, iron, aluminum, manganese, and zinc salts of oxalic acid, tartaric acid, citric acid, lactic acid, and glycolic acid. The products are produced by saturating, spraying, or coating a combustible, prefabricated material which is based on cellulose and optionally contains fillers customarily used in smokable products. The material can be used as a smokable product with a dispersion of solids, in a solution of salts of monobasic or polybasic carboxylic acids. (Auth. Abs. Mod.)

82–0231. Horsewell, H. G.; Phelpstead, J.W.P. **Tobacco-Smoke Filters.** United States Patent No. 4,300,577, November 17, 1981, 3 pp.

A two-component filter which appears to remove a higher proportion of volatile aldehydes and hydrogen cyanide than would be expected from their individual components is described. During puffs, the first component weakly retains vapor-phase constituents such as aldehydes, while between puffs the constituents desorb from the first and combine with the second to yield substantially nonvolatile reaction products. The first component may be a porous mineral earth, such as magnesium silicate or silica gel in porous granular form. The second component may be an ion-exchange resin or polyethylene imine. The first component can be granular and dispersed in a fibrous or filamentary second component. Alternatively, both first and second components can be granular, mixed together and dispersed in a fibrous or filamentary material such as cellulose acetate, or placed between first and second plugs of such material.

82–0232. Jenkins, J. M.; Pedder, C. T.; Robertson, N.G.D. **Process and Apparatus for Producing Paper Reconstituted Tobacco.** United States Patent No. 4,270,552, June 2, 1981, 11 pp.

A method and apparatus for producing reconstituted tobacco by a papermaking process are presented. Tobacco material is mixed with aqueous tobacco extract or water to wet the tobacco. The resulting slurry is fed to a deflaker in which the bundles of tobacco fibers are reduced to small fiber bundles. The slurry is then directed to an extraction stage after which the water soluble and fibrous components of the slurry are separated. The fibrous material in the form of a slurry is preferably passed through a second deflaker to further separate the fiber bundles and through refiners to prepare the material for web formation. The fibrous material is diluted and passed to a papermaking machine in which it is processed to form a base sheet of reconstituted tobacco, and the sheet is impregnated with a concentrate of the water soluble component separated at the extraction stage. The latter component may be subjected to a high shearing force to lessen its viscosity and facilitate the impregnation process. The resulting product is a reconstituted tobacco of good quality produced in an efficient and economic manner. (Auth. Abs. Mod.)

82–0233. Kassman, A. J.; Geiszler, W. A., Jr. **Filter Cigarette With Inlet Vent Zones.** United States Patent No. 4,294,265, October 13, 1981, 4 pp.

The design for a filter cigarette having inlet vents in the filter mouthpiece divided into two groups is described. The distance between the two groups of inlet vents is selected to give an increased resistance to draw without appreciably decreasing dilution or total particulate matter. Prior methods of increasing the amount of ventilation sometimes led to a decreased resistance to draw, possibly causing smoker frustration. A denser filter could be used to increase resistance to draw, resulting, however, in excessive filtration. (Auth. Abs. Mod.)

82–0234. Mookherjee, B. D.; Wilson, R. A.; Schmitt, F. L.; Vinals, J. F.; Kiwala, J. **Use for Augmenting or Enhancing the Aroma and Taste of Smoking Tobacco and Smoking Tobacco Articles by Adding Thereto 1-(2,6,6-Trimethyl-1,3-Cyclohexadien-1-YL)-1,3-Butanedione.** United States Patent No. 4,271,853, June 9, 1981, 19 pp.

A detailed description for a compound for promoting organoleptic qualities in tobacco or tobacco articles is presented. The compound imparts sweet, fruity, berry-like, fresh squeezed fruit (apple and grape) juice-like, hay-like, and tea-like aromas prior to smoking. During smoking it imparts sweet, hay, or tea aromas in the mainstream smoke, and sweet, tobacco-like, and "fresh cigarette pack" aromas in the sidestream smoke. In addition to the compound, other flavor and aroma agents such as selected synthetic materials or natural oils may also be added to the tobacco or substitute material (such as dried lettuce leaves). The compound plus any extra additives may be added to the smoking material, to the filter, or to the leaf or paper wrapper. Applications of between 50 ppm and 1,500 ppm by weight of the compound in the smoking material are recommended.

82–0235. Siceloff, B. **Tobacco for Protein: A Revolutionary Upheaval?** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, Lexington Books, 1981, pp. 109-115.

A process for removing the protein from tobacco leaves, discovered during research to develop a safe cigarette, is described. A high-quality protein called Fraction-I, and others, are abundant and easily extracted from tobacco leaves, and surpass in quality and quantity those found in soybean or milk. However, the flue-cured tobacco farmers are worried; the scientists may also succeed in developing an inexpensive form of smoking tobacco from the byproducts of the deproteinization process. Tobacco companies have informally stated they could use such a product to blend into cigarettes and reduce the flue-cured portion, provided it did not have an objectionable odor. The result would be economic upheaval in North Carolina. The protein researchers view farming tobacco for protein as a dual system, coexisting alongside tobacco crops for smoking. However, the North Carolina tobacco industry leaders view the new process as a threat and are advocating less lucrative uses for the deproteinized byproducts, such as methanol production or animal fodder.

82–0236. Swanson, G. T.; Trenary, J. **Smoking Filters.** United States Patent No. 4,273,142, June 16, 1981, 9 pp.

Several prior approaches to cigarette filtration have led to such disadvantages as increased draw resistance, weakening of the taste, or retention of an offensive smell in the filter. The present invention is a filter apparatus designed to avoid the deficiencies of the previous approaches while accommodating their more positive aspects. It has an elongated tubular shell with one interior end portion shaped to define a coupling to the end portion of a cigarette. Longitudinally along the coupling, a tubular wall of predetermined cross-section is defined that is partially closed by a transverse wall with a central aperture. An air inlet extends through the outer wall of the shell. The filter element is of a porous material which is inhibitive to the passage of substances such as tar and nicotine. The element is in the form of an elongated hollow sleeve, with one end closed and the other end open. A laterally projecting shoulder is defined on the sleeve to have the aforementioned predetermined cross-section so as to seat frictionally within the tubular wall and against the transverse wall with a portion of the sleeve projecting through the aperture. The invention is especially directed toward cigarette use, but may also be used for other means of smoking tobacco as well as for the smoke produced by other substances. (Auth. Abs. Mod.)

82–0237. van der Loo, H. E.; Wiener, C.; Higginbotham, J. D. **Smoking Articles Containing Thaumatin or Monellin.** United States Patent No. 4,300,576, November 17, 1981, 4 pp.

The incorporation of sweet proteins (thaumatin or monellin) in a cigarette or cigar filter or in a stem of a pipe, so that the proteins do not come in contact with the mouth, is said to improve the flavor of tobacco. Cooling and smoothing, thus reducing tobacco harshness, are produced in the smoke which passes through the protein. Impregnation can be by aqueous solution, or dry powder, a cellulose acetate filter medium being preferred as it appears to absorb the protein efficiently. Since the protein does not appear to have any useful effect when burned, it is suggested that it not come in contact with the burning tobacco. Suggested amounts of protein for various smoking articles are given, and combined effects with other flavorants are discussed.

82–0238. Van Tilburg, J. **Smoke Filters.** United States Patent No. 4,273,141, June 16, 1981, 9 pp.

A ventilated filter capable of reducing carbon monoxide yields is described. The device consists of a tobacco smoke filter having at least one directly ventilated cavity which provides for the formation, over a region of the filter extending inwardly of its peripheral region, of a mixture of at least the major proportion of the smoke and at least a major proportion of the ventilating air entering the said cavity or cavities. The extensive and uniform mixing of smoke and ventilation air gives a satisfying smoke with a low tar count and high nicotine/tar ratio, and can be obtained with the invention due to appropriate shaping of the cavity, or asymmetric positioning of the air ventilating vents, or by means of a flow-disturbing insert mounted in the filter, or a combination of two or more of these factors. Preferably a major part of the smoke is constrained to enter the cavity at an upstream peripheral extremity thereof and air vents are also concentrated at this extremity. This filter allows air dilutions of up to 80 percent and higher, while maintaining a smooth smoke of acceptable taste. The filter can also give a high CO reduction. Compared to a cigarette with the same amount of tobacco and a conventional filter, it can give up to a 30 percent increase in smoking duration. Conversely, compared to a conventional cigarette of the same smoking duration, it can give up to 25 percent saving in tobacco. The filter can be made of the usual filtering materials, such as cellulose acetate filaments or fibers, usually along with a plasticizer or paper. Different filtering materials can be used for different parts of the filter. (Auth. Abs. Mod.)

82–0239. Yamaguchi, T. **Cigarette Filter.** United States Patent No. 4,269,204, May 26, 1981, 5 pp.

An improved cigarette filter is described, wherein dry and active green algae in the form of chlorella in a granular or powdery form can physically trap and remove such toxic substances in cigarette smoke as nicotine, tar, and carbon monoxide. The toxic components are deposited on a porous columnar carrier carrying the green algae. When dry and active chlorella are in a granular or powdery condition, they are highly porous with a number of very fine pores. The rate of contact of the passing cigarette smoke with the chlorella is as high as with the conventionally used activated granular charcoal. The chlorella's capacity for physically trapping the toxic substances is also high. In addition, the algae can biologically denature and change the toxic components to harmless form. The filter is used as combined with a cigarette holder or directly with a cigarette. With the carrier, preferably, a total weight of 20 to 50 mg of granular or powdery chlorella is used alone or as combined with such inorganic porous adsorbents as fine granular or powdery zeolite, silica gel, iron oxide, or activated charcoal. (Auth. Abs. Mod.)

82–0240. Yeatts, R. E. **Composite Tipping Structure for Use on an Air-Ventilated Cigarette and Method of Manufacturing Same.** United States Patent No. 4,295,478, October 20, 1981, 6 pp.

A composite tipping structure which uses air dilution of the smoke of a cigarette to produce a milder smoking product is described. The intention is to provide a ventilated tipping paper in which the porosity can be controlled and which will adhere to the filter plug without leaving large areas free of adhesive, especially around the edge of the tipping. The composite structure includes a nonporous tipping paper coated with a heat-activatible adhesive in a selected pattern, having areas void of adhesive and microscopic openings through the tipping paper within the void areas. The method of manufacturing the composite structure includes applying a nonconductive, heat-activatible adhesive coating on the paper web in a specific pattern having void and adhesive coated areas and forming perforations in the void areas of the paper web. (Auth. Abs. Mod.)

TOBACCO ECONOMICS

82–0241. Adams, F. **Vegetable and Fruit Crops: Viable Alternatives for Tobacco Farmers.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 93-102.

Fewer farmers today can afford to grow tobacco due to the increasing size and mechanization of tobacco operations. The production of tobacco is becoming less labor-intensive and more capital-intensive. The small tobacco farmer can respond by borrowing money and investing in the tobacco operation, hoping to keep the operation going; by taking an off-farm job; or by trying to change crops and remain on the farm. The change to other crops presents prospects and problems. Tobacco farmers are accustomed to guaranteed market outlets and sales price levels through the Federal support system, and coping with new crops can be a challenge, especially in marketing. Tobacco farmers cannot switch to soybeans, corn, wheat, or other grains, which require large acreage to make a profit, but vegetable and fruit crops are viable alternatives. At present, alternative crops do not get the same attention as tobacco from Federal and State programs and research; however, the Frank Porter Graham Center in Wadesboro, North Carolina, a private, nonprofit research project of the National Sharecroppers Fund, is working to provide viable models for small farmers who wish to continue working their land. The Center's research suggests that changing to alternative crops would not require additional machinery or labor, would probably use less fuel or pesticides, and would not be adversely affected by the soil and climate of tobacco regions, although berries and some vegetable crops might require irrigation. The lack of a stable market (unlike tobacco) and distribution system may be the biggest deterrent to tobacco farmers wishing to switch to other crops. Prices for most alternative crops fluctuate from year to year. Better distribution is needed to get the vegetable and fruit produce to potential consumers. One possible method open to farmers is to work in cooperative efforts. A co-op could pool individual harvests into quantities large enough to attract established bulk buyers such as jobbers, wholesalers, and processors, who sell to large populations of consumers. One such group in North Carolina is the Piedmont Vegetable Marketing Cooperative, Incorporated. If it proves successful, it may serve as a marketing model for farmers in other areas.

82–0242. Badger, A. J. **Early Efforts To Control the Market—And Why They Failed.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 3-12.

Individual tobacco growers faced severe economic difficulties in the decades leading up to the 1930s. Most growers, whether owners or tenants, were in debt from January 1930 onward, their crop being their only security for year-long credit. Since banks were reluctant to make short-term credit available to farmers, the farmers had to turn to the time merchant, who charged high rates of interest and credit prices higher than cash payment prices. Since tobacco is a perishable product and the individual grower could not afford redrying, he could not withhold his crop from the market if prices were low. Prices paid for tobacco by buyers were affected by grading, the grades being secret and varying from company to company. Manufacturers had 2 or 3 years' supply of tobacco at hand, and could wait if prices were up in a given year. There was a lack of competition among buyers. A chronic tendency to overproduce also led to poor prices for farmers. Before the New Deal there were four main efforts to improve the economic status of growers: (1) the Tri-State Growers' Cooperative, organized in 1921 and 1922 in an attempt to persuade farmers to turn over their tobacco to the cooperative to be graded, redried, and stored until a favorable time for selling. It failed due to opposition from warehousemen and creditors, and because it never controlled enough of the crop; (2) North Carolina Governor O. Max Gardner's 1929 "Live at Home" campaign, encouraging crop diversification so that less tobacco acreage would cause a price rise allowing farmers to at least live by subsistence farming and be less dependent on tobacco. It had little effect on the price of tobacco, and did not appeal to the large commercial farmer and landlord; (3) the Federal Farm

Board's cooperative attempt in 1929, failing because warehousemen and time merchants remained hostile. Growers themselves were distrustful and not willing to submit to the discipline of belonging to a cooperative; and (4) the 1931 attempt to restrict production by interstate agreement or individual State legislation, which was opposed by many State leaders on practical and constitutional grounds. Apparently a reduction of acreage was needed to adjust supply to demand, but voluntary and local efforts to do so had failed. In 1933 the North Carolina tobacco growers had to wait for the Federal New Deal.

82–0243. Berardi, G. **Can Tobacco Farmers Adjust to Mechanization? A Look at Allotment Holders in Two North Carolina Counties.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 47-61.

Flue-cured tobacco has remained a labor intensive crop longer than most others in modern American agriculture. In the last 20 years, however, several factors have altered patterns of tobacco production, and reduced the number of tobacco farms and farmers. Two "push" factors are technological innovations and changes in the Federal support program; e.g., chemical controls reduce the labor needed to remove sucker leaves, and transplanting is less labor-intensive due to mechanical transplanters. Mechanical harvesters, in use since the early 1970s, have a break-even capacity of 40 to 50 acres, thus encouraging tobacco farming units to consolidate or enlarge. Curing in bulk barns has also complemented harvester use. U.S. Department of Agriculture data (1975) found that in 1972 only 1 percent of the tobacco acreage was harvested mechanically and 8 percent cured in bulk barns. It predicted that by 1978 mechanical harvesting would grow to 23 to 36 percent and 65 to 80 percent for bulk barns. Three major changes in the Federal support program also promoted larger farm units and less labor. In 1961, intracounty lease-and-transfer of flue-cured allotments was allowed on a limited basis. In 1965, the flue-cured allotment system changed from an acreage to a poundage basis, so that leaf left in the field by mechanization was not a loss. In 1968, changes in the marketing of flue-cured tobacco to "sheets" rather than "hands," required less labor. A "pull" force was increasing employment in nonfarm occupations. In North Carolina nonfarm employment increased from 1.4 to 2.1 million from 1965 to 1977. In the summer of 1980, a study of 116 tobacco allotment holders was conducted in Greene and Wayne counties, to document socioeconomic characteristics of allotment holders no longer producing their quota, and to determine how former tobacco growers had made the transition to nontobacco income. About half (46.7 percent) of the households made less than 10,000 dollars in 1979. Most of these incomes, on a per capita basis, are below the North Carolina State income average of 7,359 dollars. The allotment holder population has tended to replace tobacco earnings by combining income from four sources: lease and transfer of quota (95.3 percent of the population), special services such as Social Security (56.6 percent), off-farm employment (31.1 percent), and crop and livestock production (29.2 percent). More research is needed on the adjustment of populations to the new tobacco trends.

82–0244. BfAI-Report. **Considerable Production Increase Within the Algerian Tobacco Industry.** *Tabak Journal International* 4: 286-287, August 1981.

Between 1975 and 1979, the Algerian tobacco industry experienced a considerable rise in production of: cigarettes from 9,668 tons to 14,678 tons with an expected 1980 total of 16,242 tons; chewing tobacco and snuff from 4,685 tons to 6,705 tons with an anticipated 1980 level of 7,686 tons; and cigars from 14.9 tons to 29.0 tons, with the 1980 level expected to be 33.7 tons. However, the production of cigarette tobacco and pipe tobacco fell from 265 to 98 tons. During the 1975-1976 and 1977-1978 crop years, Algerian tobacco production rose from 1,900 tons to 3,300 tons, but imported tobaccos are still being used to a large extent, partly because the Algerian public continues to prefer foreign cigarettes (particularly French) which have considerable prestige value, and efforts to persuade consumers to switch to similar domestic brands have not been successful. In 1979, 16,450 tons of tobacco and tobacco goods valued at 125.0 million Algerian Dinars were imported, while tobacco goods are only a minor export product. From 1975 to 1979 the domestic consumption of cigarettes increased from 9,448 tons to 14,659 tons, and further increases are expected in spite of a current antismoking campaign. Cigar consumption in Algeria rose during the same period from 19.9 tons to 29.0 tons, and chewing tobacco and snuff consumption rose from 4,219 tons to 6,266 tons. Pipe and cigarette tobacco consumption decreased from 265 tons to 98 tons. Tobacco goods in Algeria are not customarily sold from automatic vending machines but from kiosks, tobacconists' shops, and nonauthorized itinerant traders.

82–0245. Burry, R. D. **Aggressive Marketing Needed To Sustain 1980's Cigarette Upturn.** *Tobacco International* 183(7): 8-9, 11, April 3, 1981.

The reported performances, full-year trade association data, and government statistics of the major tobacco companies for 1980 indicate significant shifts in total consumption trends, the mix between cigarettes of differing tar and nicotine levels, and market share patterns. Cigarette shipments between wholesalers and retailers were used as the most accurate measure of overall domestic consumption. In 1980 total consumption increased 2 percent, reversing an earlier trend toward declining growth rates, and necessitating increased production and reduced exports. Factors limiting cigarette consumption include slow growth in the under age 25 population where usage traditionally increases, public health awareness, greater restrictions on smoking in certain areas, and retail price increases. Factors promoting usage include increased numbers of female smokers, lessening public prominence of the health issue, and the growing popularity of low-tar cigarettes; however, the market must still be closely watched through the early 1980s. The low-tar segment of the cigarette market continued to expand in 1980, with unit volume growing from 6 to 7 percent. Low-tar cigarettes, which accounted for almost half of 1980 industry shipments, are expected to increase their market share over high-yield brands, but not to alter fundamentally the volume or share relationship between major companies. The top two companies, Philip Morris and R. J. Reynolds, continue to extend their dominance of low-tar shipments, with most of the low-tar share acquired in 1980, being captured from the two weakest producers, Lorillard and Liggett. In overall unit volume of cigarette production, Philip Morris increased its market share by 8.1 percent in 1980, accounting for 31.1 percent of industry shipments.

82-0246. Campbell, J. S. **American Leaf Exports on Decline: Imperial Tobacco Limited Closes Its Last American Primary-Processing Plant.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 145-149.

The U.S. flue-cured tobacco industry is suffering losses in the world market. This was dramatized by Imperial Tobacco Limited's decision in the spring of 1980 to close its last American processing facility; the United Kingdom has traditionally been a leading buyer of American leaf. However, a diminishing demand for high quality leaf has forced prices to unrealistic levels, and in recent years the United Kingdom has used far less American tobacco. The demand for flue-cured tobacco has declined for many reasons. Research has reduced waste (less leaf required for each cigarette) and discovered ways to replace high-cost leaf with cheaper, low-quality leaf, while increased knowledge of health consequences has created a growing market for low-tar brands. Manufacturers no longer rely on a small number of grades for production; but for the U.S. tobacco industry to protect its market, the Federal program must be changed to enable farmers to increase production while maintaining competitive prices in the world market.

82-0247. Campbell, J. S. **Agricultural Economics and the U.S. Tobacco Industry.** *Tobacco International* 183(7): 32, 34, 37, April 3, 1981.

The 1981 meeting of the Tobacco Workers' Conference paid special attention to domestic and foreign leaf and product sales as they relate to producers and marketing of their products. One recent trend discussed was the increased use of labor saving bulk barns and mechanical harvesters, with a corresponding drop in harvest labor. Management units decreased in number and increased in size, rising from 9.5 acres in 1972 to 13.8 acres in 1979. An estimated 50 percent of flue-cured acreage and 25 to 30 percent of Burley will be rented in 1981, with higher rent prices in counties where there is a greater concentration of tobacco acreage and higher yields per acre. Tobacco is one of the few farm commodities experiencing a declining market, despite strong world demand. High support prices in recent years have constrained consumption and encouraged leaf imports, resulting in shrinking acreage and declining market shares. This has led to quota reductions which increase the pressure to keep rising support levels to protect grower incomes. It was predicted that in the European Community nationalism would keep each country's cigarettes chiefly in the country that smokes them, and that the Common Agricultural Policy will tend to reduce imports, especially when the Community is enlarged. Future challenges may come in 15 to 20 years when the new "developed world," including South Korea, Taiwan, Malaysia, Brazil, Venezuela, the Arabian Gulf States, and others, may initiate major changes in cigarette blends and place more emphasis on individual tastes of smokers. In the United States, current worries about smoking and health continue to affect industry growth and may account for the present emphasis on low-tar cigarettes, now at 50 percent of the market. World cigarette output during the past 3 years has declined to a growth rate of 2 percent or less. Such a small increase coupled with a 1 percent decline in tobacco use per cigarette could mean that demand for cigarette leaf would grow by only 1 percent a year. Production of flue-cured, Burley, and Oriental tobaccos is nevertheless projected to increase about 2 percent a year during the 1980s. As cigarette consumption lags behind leaf supplies, there may be a downward pressure on prices and production adjustments in all countries.

82-0248. Clairmonte, F. F. **World Tobacco: A Portrait of Corporate Power.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 203-219.

The global tobacco economy is dominated by seven transnational tobacco conglomerates (TTCs), who produce more than 39 percent of the world's cigarettes, with worldwide sales exceeding 56 billion dollars in 1979. The seven TTCs are: British American Tobacco Company (BAT), Imperial Tobacco Company (ITC), R. J. Reynolds (RJR), Philip Morris (PM), the South-African-controlled Rupert Rembrandt/Rothmans group, American Brands, and Gulf and Western. The BAT company is the largest, with 1979 sales exceeding 7.2 billion pounds. Since BAT's beginning in 1902, it has shared with the Imperial Tobacco Company joint ownership in several corporate bodies. The ITC has more than 60 percent of Britain's cigarette market. It is diversifying into food and beverages, as are other tobacco conglomerates. In 1970, the Reynolds Tobacco Company became a fully owned subsidiary of R. J. Reynolds Industries, which has engaged in systematic acquisition of foreign enterprises. It has also broken into the food industry and agribusiness. Philip Morris in 1979 controlled 29 percent of the U.S. cigarette market and 6 percent of international tobacco sales. Gulf and Western owns Consolidated Cigar, one of the world's leading cigar corporations, controlling about one-third of the U.S. market and selling its Spanish, Dutch, and American cigars in some 100 countries. There are several mechanisms of corporate power: (1) conglomeration has resulted in tobacco industries spreading through modern economies and acquiring member industries in a wide range of nontobacco products; (2) cross-subsidization deploys masses of capital from one profit center to another to acquire control in other industries; (3) appropriation of technology and innovation have generated ever-larger economies of scale, contributing to the increase in capital concentration; (4) finance capital are provided by global working relations between financial institutions and the tobacco industry; and (5) the TTCs' global advertising costs are now over 2 billion dollars a year and rapidly increasing. The TTCs, working for further corporate growth, especially in foreign markets such as the Third World, continue to compete against new entrants, avoid active price competition, generate massive surplus cash flows, cross-subsidize to boost new product lines, and promote mutual relations with other corporations.

82–0249. Dalton, R. **Resources on Tobacco Production and Marketing.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 75-89.

The tobacco program in the United States is administered and affected by a variety of organizations in the Federal and State governments, as well as in private industry. The U.S. Department of Agriculture has several involved divisions: (1) the Secretary, who makes the final administrative decisions; (2) the Agricultural Stabilization and Conservation Service, with its 600 county offices, which is chiefly responsible for implementing the program; (3) the Agricultural Marketing Service (Tobacco Division), which defines tobacco grades and grades the tobacco at market centers; (4) the Commodity Credit Corporation, which makes nonrecourse loans to stabilization cooperatives; (5) the Foreign Agricultural Service (Tobacco, Cotton, and Seeds Division), which works to maintain and increase tobacco exports; (6) the Economic Research Service, involved in a variety of projects related to the tobacco industry; and (7) other divisions, including the Statistical Reporting Service, the Agricultural Cooperative Service, the World Agricultural Outlook Board, and several operations in Science and Education Administration, including Extension Services. The North Carolina Department of Agriculture has several divisions, including an Office of the Commissioner, involved in marketing, research, market news, a reporting service, and regulatory functions. The State also has two land-grant universities, North Carolina State University at Raleigh and North Carolina Agricultural and Technical State University at Greensboro, which engage in agricultural research and provide agricultural extension services. Several industry organizations also exist, including the Tobacco Association of the United States (TAUS) and the Tobacco Institute. TAUS, founded in 1900 and the oldest tobacco trade association, promotes the growth, sale, distribution, manufacture, and consumption of domestic tobacco in the United States and overseas. The Tobacco Institute, a nonprofit, noncommercial organization founded in 1958 by 11 U.S. tobacco product manufacturers, encourages public understanding of the smoking and health controversy, and the historic role of tobacco and its place in the national economy.

82–0250. Dalton, R. **Changes in the Structure of the Flue-Cured Tobacco Farm: A Compilation of Available Data Sources.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 63-74.

In the past decade, farms growing flue-cured tobacco have become larger, more mechanized, requiring less labor, and relying on more leased quota, four factors which are interrelated and interdependent. According to U.S. Census of Agriculture data, flue-cured farm size increased from about 5 acres in 1964 to 13.8 acres in 1979, with the percentage of acreage harvested mechanically increasing from 1 percent in 1972 to 19 to 33 percent in 1979. The percentage of acreage that was bulk-cured increased from 8 percent to 61 percent. The North Carolina Agricultural Extension Service and the Tobacco Association of the United States (TAUS) agree with the USDA on the extent of bulk barn use for curing, and tend to give even larger estimates of the pace of mechanization. Labor requirements have declined due to devices such as the mechanical harvester and weed-control chemicals, with USDA estimates of flue-cured harvest workers dropping from 325,000 in 1972 to 211,000 in 1979. In 1961, Congress voted to allow the lease and transfer of tobacco quota within counties, removing in 1967 the 5 acre limit on leasing to any one farm. Surveys by the USDA in North Carolina show a large growth in lease and transfer since 1966, both in raw numbers (pounds of quota and acreage of allotment assigned to each farm) and in percentages. From 1966 to 1979 the amount of quota increased from 79.7 million to 280 million pounds and allotment leased from 42,200 to 147,600 acres. Similar trends occurred throughout the flue-cured belts, with the quota leased increasing from 138 million pounds in 1966 to 477.3 million pounds in 1979 and allotment leased from 80,100 acres to 267,700 acres. In 1972, 19 percent of farm operators beltwide owned the entire tobacco quota that they produced, declining to 16 percent in 1979. These four changing factors have a vital role in influencing the structure of flue-cured farms.

82–0251. Federal Trade Commission. **Report to Congress, Pursuant to the Federal Cigarette Labeling and Advertising Act for the Year 1979.** Federal Trade Commission, 1979, 27 pp.

Trends in cigarette advertising and changing consumption patterns for the United States, including military overseas, are presented in this annual statistical report (1979) of the Federal Trade Commission. Although total cigarette sales increased, per capita consumption declined slightly, for the seventh straight year. The market share for filter cigarettes rose (91 percent in 1979, 1 percent over 1978, and 33 percent higher than in 1963), as did that of menthol brands (29 percent in 1979, compared to 16 percent in 1963). Market growth also continued for the longer cigarettes (95-101 mm), while the newer 110-121 mm brands remained steady at 2 percent. The greatest gains, however, have been in "low-tar" brands (15.0 mg or less) which have increased from a 2 percent share of the market in 1967 to 40.9 percent in 1979; however, 90 percent of all cigarettes sold in the United States in 1979 had "tar" levels of over 9.0 mg. The most illuminating figures occur in advertising expenditures (newspapers, billboards, and magazines) which have grown dramatically since the ban on radio and TV advertising: from 71 million dollars in 1970 to approximately 663 million dollars in 1979. Filter cigarette advertising now accounts for 99 percent of expenditures (up from 75 percent in 1963), with "low-tar" brands receiving a disproportionate share: 30.9 percent of advertising budgets allocated to brands holding only a 10.6 percent of market shares.

82–0252. Finger, W. R. (Editor). **The Tobacco Industry in Transition.** Lexington, Massachusetts, Lexington Books, 1981, 339 pp.

Several policy areas in today's domestic tobacco industry, including agriculture, farmer alternatives, leaf marketing, manufacturing, health, and politics, are addressed. The agriculture section describes the Federal tobacco program and the great changes that tobacco farming has undergone in recent years, such as increased mechanization and consolidation of farmland. Alterations of the Federal program are proposed to meet changes in tobacco farming. In the second section, alternatives suggested for farmers who wish to quit tobacco farming include growing vegetable and fruit crops or moving into industrial work. An alternative use for tobacco as a source of protein is also suggested. The third section describes expanding world leaf sales and the declining U.S. share of the market. Many developing countries are increasing their own tobacco agriculture, but also may represent a new market for U.S. tobacco. The next section reviews corporate diversification and international expansion, and details are given for the larger U.S. tobacco companies. The fifth section discusses the smoking and health controversy, including legal and policy issues. The final section covers the political issues concerning tobacco, including arguments between tobacco industry proponents and opponents.

82–0253. Finger, W. R. (Editor). **Appendix 2A: An Economic Interpretation of the Current Tobacco Program.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 27-29.

An economic portrayal of the current tobacco program is presented, including a diagram displaying traditional supply-and-demand curve dynamics. Information is provided about an inelastic aggregate demand curve for tobacco at the farm level, the average price support for a given year, the open market price, the willingness of present quota holders to supply tobacco at various prices, the national marketing quota, a kinked supply curve, and rental costs. The tobacco quota is restricted at a level expected to yield a price at or above the support rate. If demand is misjudged so that prices bid on the market do not exceed the support rate, funds are advanced to acquire excess tobacco. In subsequent years, quotas have to be adjusted downward to allow the excess stocks to move into trade and to prevent long-term government losses. Adjustments in the quota level may be used to compensate for changes in demand. Changes in demand can be caused by antismoking efforts affecting consumer tastes, population changes, income changes, changes in conditions of international trade, cigarette manufacturing techniques, and taxes.

82–0254. Foreign Agriculture. **Commodity Update.** *Foreign Agriculture* 19(4): 5-6, April 1981.

In the United Kingdom, cigarette prices increased 18 percent as of March 14, 1981, due to a tax increase announced in the 1981-1982 budget. A pack of 20 king-size cigarettes will now cost 2.10 U.S. dollars. The cigarette excise tax went up 34 percent, reaching the equivalent of about 40 U.S. dollars/1,000. The Value Added Tax remains at 15 percent and the ad valorem tax component at 21 percent, although the actual amount of taxes will increase, since the higher specific tax raises the base for both taxes. The specific excise tax on cigars increased 34 percent to 34.29 pounds (81.37 U.S. dollars) per kilogram. The tax surcharge on high-tar cigarettes was abolished. In 1980, U.S. leaf exports to the United Kingdom were 14,728 tons valued at 78.2 million dollars, a decrease of 52 and 44 percent respectively, compared to 1979. The U.S. leaf exports are expected to decrease further, due to high domestic taxes in the United Kingdom as well as a drop in cigarette consumption and attempts by United Kingdom manufacturers to obtain lower cost leaf from other suppliers.

82–0255. Herget, J. B. **Industrial Growth: An Alternative for North Carolina's Tobacco Farmers.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 103-108.

During the 1970s, in North Carolina, the small farm gave way to agribusiness, and thus affected the last major cash crop grown on small farms—tobacco. During 1978, only 52,000 were growing tobacco in North Carolina, which has a labor force of 2.6 million. By 1979, there were only 46,000, a decline of 12 percent. In an effort to offer alternatives to tobacco farmers who have been squeezed out by industrialized methods, the State has been promoting new industry in rural counties. An attempt has also been made (successfully) to diversify the industrial base, protecting the State economy against violent swings, and to create new jobs in areas previously dependent on tobacco farming for economic survival.

82–0256. Hill, D. **Export Sales Gain in Importance to U.K. Industry Besieged by Taxes.** *Tobacco International* 183(5): 48, 51, 53, July 24, 1981.

In 1981, the British tobacco industry has had to contend with the effects of two governmental tax increases: (1) the initial increase which added 14p to the price of a packet of cigarettes, a similar amount on an ounce of pipe tobacco, and 9p on a packet of small cigars; and (2) the second, a few weeks later, which added an additional 3p to 20 cigarettes, with linked increases on other products. The initial drop in consumption after the latest price increase is estimated at about 13 percent for cigarettes and 8 percent for cigars. Cigarette sales in Great Britain had already been steadily decreasing from their peak levels in 1974, and as a result, the tax increases adversely affected an industry already burdened by economic recession and the health controversy. In response to government pressure, the tobacco industry has agreed to emphasize low-tar brands in its advertisements, to introduce new lower tar brands, and to reduce tar yields in existing brands. A voluntary agreement that will remain in force until mid-1982 includes further restrictions on advertising and more emphasis on health warnings. Consumers continue to switch to filtered brands, which now comprise about 93 percent of the domestic market, with the king-size filter brands dominating sales. With the sagging domestic market, British manufacturers are increasingly turning to exports which in 1980, totaled 41,023 tons of cigarettes valued at 264 million pounds, up from 35,881 tons (210 million pounds) in 1979. In unit terms, British cigarette exports climbed steadily from 25.9 billion in 1976 to a record 36.9 billion in 1980. In value terms, American leaf still topped the list of tobacco sources. Despite difficulties in the home market, British clearances of unmanufactured tobacco rose to 147,800 tons, or 5,000 tons above the 1979 figure, a result of increased exports and the trend toward king-size brands.

82-0257. Huebner, A. **Making the Third-World Marlboro Country.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 151-156.

Transnational tobacco companies are seeking new markets in the Third World, to compensate for stagnating markets in the developed world; e.g., U.S. market growth has been only 1 percent annually for the past 5 years. While the United States and European countries continue antismoking campaigns, the Third World offers a new market with a vast population, and governments unable or unwilling to fund antismoking campaigns, and reluctant to give up the increasing tax revenues from cigarette sales. Direct evidence of health hazards has not yet begun to show in their morbidity and mortality tables. In the past decade, Philip Morris' overseas cigarette sales, in more than 160 countries, have increased 18 percent annually. British-American Tobacco, the world's largest cigarette maker, distributes 300 brands in 180 countries, and other companies are entering the market. According to the United Nations Food and Agriculture Organization (FAO), cigarette consumption in the Third World is growing at a rate of 5 percent a year. Advertisements seek to present smoking as an adult, healthy, or sexy activity. Tobacco companies also sponsor Third World sports competitions, as well as using testimonials from athletes. Western governments also promote the spread of the smoking habit in the Third World. Apart from domestic subsidies and price-stabilization loans, the U.S. Government has shipped tobacco valued at hundreds of millions of dollars to Third World countries under the Food for Peace program; the European Economic Community continues to subsidize its tobacco farmers; and China, the world's largest tobacco producer, recently displayed its brands at a Philippine trade fair. Even the FAO and the United Nations Development Program promote tobacco as a cash crop in many Third World countries, to the distress of the World Health Organization. The trade-off appears to be between health and economic growth. The hopes for economic growth may not be realized, however, as mechanization spreads to the Third World, reducing labor demands. Tobacco acreage also ties up arable land that could grow food. Although tobacco could be grown for export as a cash crop, worldwide smoking trends suggest that an increasing proportion grown in Third World countries is smoked within their borders. The health hazards associated with smoking could be especially damaging to Third World countries, which are least able to afford the related health care costs.

82-0258. Kiger, H. C. **Open Trade and Modernized Tobacco Program: The Keys to an Expanded U.S. Flue-Cured World Market.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 131-144.

World trade is important to the U.S. tobacco economy and is likely to become more so due to decreasing domestic use of U.S. flue-cured tobacco. From 1973 to 1979 domestic use fell from 703 million pounds to 563 million pounds. The U.S. tobacco economy's increasing dependence on exports has also been promoted by a shift to lower yield cigarettes (which need less tobacco), a disparity in price between American and foreign leaf, and higher U.S. taxes on cigarettes. The United States is the world's largest tobacco exporter, as well as the third largest importer; however, from 1966 to 1979 the United States' share of world trade in flue-cured tobacco fell from 60 percent to about 28 percent, due to increased competition from other exporting countries. U.S. tobacco exporters feel that tobacco programs need to be modified to make U.S. flue-cured prices more competitive on the world market, and that freer trade should be encouraged to ensure that the United States gains a larger share of the world market. The high price of U.S. flue-cured leaf is largely the result of the domestic tobacco program. The U.S. tobacco industry gained its world trade dominance during the past 50 years while the United States had a relatively free trade policy. The United States has participated in the General Agreement on Tariffs and Trade, which seeks to promote nondiscriminatory trade. It is suggested that the U.S. tobacco economy should support trade legislation and policies to reduce trading barriers on a reciprocal basis. The world demand for tobacco will probably continue to grow in the 1980s, particularly in Third World nations with expanding economies. Thus, policies that encourage more competitive U.S. prices and greater access to foreign markets may result in expanded exports, increased domestic use, higher farm income, and an increase in the U.S. balance of payments.

82-0259. Kinney, J. A. **Tobacco's Global Economy: Is North Carolina Losing?** In: Finger, W. R. (Editor). *The Tobacco*

Industry in Transition. Lexington, Massachusetts, Lexington Books, 1981, pp. 119-129.

The North Carolina tobacco farmer faces a variety of challenges for the 1980s: (1) a 1 to 2 percent annual decline in domestic demand as a result of health hazard awareness and higher taxes; (2) technological advances which reduce manufacturing wastage; (3) rising demand for low-yield cigarettes which use less tobacco; and (4) increasing use of foreign tobaccos by U.S. manufacturers (from 11 percent in 1965 to 30 percent in 1980). Although total U.S. exports grew from 259,000 metric tons in 1975 to 358,000 in 1980, the actual U.S. share of the expanding world market dropped from 61 to 29 percent between 1960 and 1979. Many developing nations, while increasing their tobacco production as a good cash crop, are also potential markets, since consumption is still low, populations and incomes are rising, and the health hazards of smoking have drawn little attention as yet. With the lifting of international trade sanctions, at least one major Third World producer, Zimbabwe, may be expected to greatly increase its export of tobacco leaf, which is currently considered to be second in quality only to U.S. leaf, at about half the price. The continued support of the Federal Government will be needed to help U.S. farmers meet the international challenge. Such support includes providing market information, sponsoring market development efforts, negotiating favorable tariff and trade agreements, and financing loan and credit programs. The effect of the price-support program may have discouraged export growth; however, if the global economy continues its gradual growth, the United States may be expected to retain its dominant role as the world's leading tobacco exporter, at least through the 1980s.

82-0260. Louis, A. M. **The 150-Million Dollar Cigarette.** *Fortune* 102(10): 121-122, 125, November 17, 1980.

The Brown and Williamson Tobacco Corporation (BWTC), in a dramatic business gamble, is risking an estimated 150 million dollars on advertising and promotion for a single new product: an ultra-low-tar cigarette called Barclay. Promotion costs include free cartons of Barclay, which could total up to 80 million if the offer is continued. BWTC is a steadily declining subsidiary of BAT Industries Ltd., the largest cigarette producer in the noncommunist world. BWTC has previously promoted and failed to establish a market for two other ultra-light brands, but now claims to have solved the problems of draw and taste which have plagued ultra-lights to date. The promotion of Barclay is viewed as BAT's final attempt to reverse BWTC's downward trend. Since newspaper publication of the findings of a 1978 study which concluded that lower tar cigarettes were safer for health, sales of ultra-lights have nearly tripled, with room, theoretically, for six more brands on the market. A different approach to the question of tar content, stressing the absence rather than tar level ("99 percent tar-free"), may make Barclay a success.

82-0261. Mann, C. K. **The Tobacco Franchise for Whom?** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 37-46.

In 1933 Congress authorized a program to halt the drop in tobacco leaf prices, limiting the overall amount grown by establishing a national quota and determining growers by assigning allotments to the land planted in tobacco in the 1930s. A tobacco farm's value thus came to be judged less by the quality of the land than by the allotment assigned to it, and encouraged farmers to increase the yields on their allotment instead of putting more of their land into tobacco. The system also arrested the 1930s shift of the tobacco belt out of Virginia and the North Carolina Piedmont into the more fertile and productive coastal plains of North and South Carolina. Although the allotment system has remained essentially unchanged, three important modifications have had effects. In 1961, Congress allowed lease-and-transfer of allotments within a county, allowing consolidation of acreage, promoting increased mechanization, and reducing labor opportunities. In 1965, the allotment system was changed from an acreage to a poundage base, also promoting mechanization. In 1968, the "tying" requirement for flue-cured tobacco was dropped, allowing the stacking of "sheets" of leaf which required less labor, and promoted bulk curing, also complementing increased mechanization. Bulk-barn curing acreage rose from 8 percent in 1972 to 61 percent in 1979, while average farm size increased from 9.5 to 13.8 acres, and harvest labor use fell from 187 to 118 hours per acre. Mechanization is increasing pressures for statewide leasing. If greater allotment mobility were allowed, tobacco production would move into the coastal plains and become more efficient. It is suggested that all tobacco farmers should be included in the program, including laborers. Those farmers who do not have an allotment benefit little from the existing program. Other proposed changes in the program include allowing anyone with a history as a tobacco grower to be granted an allotment in relation to that history. The government could purchase allotments from present owners wishing to leave before parceling them out to tenants. Allotment programs could be reviewed or modified perhaps every 5 years, to avoid the "freezing" process that took place in the 1930s. Former tobacco workers who are unemployed could be retrained and given other worker-oriented assistance.

82-0262. Maxwell, J. C., Jr. **Valuable Vehicles for Long-Term Gains.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 229-231.

Since 1950 the cigarette industry has increased overall unit sales by 74 percent; however, future domestic growth may be limited by demographic variables and health concerns. While the average person forms a smoking habit in their early twenties, and usually begins to cut down after age 40, the demographic impact of the postwar "baby boom" is fading, reducing the number of potential smokers. In addition, growing awareness of the health hazards of smoking has encouraged some smokers to reduce or quit smoking. As a result, there may be little growth, or even a decline in the domestic market in the future. However, the possibilities worldwide may be more promising, since per capita income, which shows a direct correlation with cigarette consumption, is rising throughout the world. In recent years, industry growth rates outside the United States have been around 2 to 4 percent, and U.S. exports have been increasing: Philip Morris cigarette sales rose from 18.13 billion units in 1975 to 39.10 billion in 1979; and Reynolds sales rose from 20.50 billion units to 32.50 billion during the same period. In addition to pursuing the international tobacco market, major American companies have also turned to diversification during the past decade, investing in such areas as shipping, oil, beer, department stores, pet foods, whiskey, insurance, and gold products. This diversification is due more to a high cash flow than to declining investor confidence, and it is expected that the preponderant share of company earnings will still come from tobacco sales for the immediate future.

82–0263. Maxwell, J. C., Jr. **The Maxwell Fact Book. A Tobacco Report.** New York, Lehman Brothers Kuhn Loeb Inc., June 1981, 78 pp.

The Maxwell Fact Fook, published in June 1981, tabularly reviews the status of the tobacco industry, with emphasis on tobacco consumption, cigarette taxes, tobacco leaf usage by types, imports and exports, and world production of flue-cured and Burley tobacco and cigarettes. Information is also given on cigarette brands (history and prices), a 10-year summary of cigarette sales, expenditures for tobacco products, tobacco manufacturers, cash receipts, tobacco acreage and yield, cigarette leaf usage, cigar leaf usage by types, the smokeless tobacco industry, and an 8-year summary of cigar sales. The U.S. Department of Agriculture data are generally limited to the U.S. tobacco industry.

82–0264. Mayrhofer, K. **Tabak von der Verarbeitung bis zum Endverbraucher—Erfahrungen am Verbrauchermarkt. [Tobacco From Processing to the Ultimate User—Experience With the Consumer Market.]** *Der Deutsche Tabakbau* 61(4): 37-39, February 15, 1981, German.

Development, testing, packing, advertising, and distribution are described as necessary steps on the long way from conception to marketing of a new tobacco blend or a new cigarette brand. An overview of the 1979 West German tobacco products market is presented. Total 1979 West German consumption of cigarettes was about 133 billion or an average of 20 cigarettes/consumer/day. From 1970 to 1979, the nicotine and tar content of the 50 leading cigarette brands, representing 95 percent of the German market, decreased by about 26 and 22 percent, respectively. However, a few, mainly young, smokers are switching increasingly to moderately strong or strong cigarettes, e.g., Marlboro. In 1977, the upturn in the market of tobacco products (fine cut, pipe tobacco, chewing tobacco, snuff) coincided with a stiff increase in the cigarette tax. From 1978 to 1979, sales of cigars and cigarillos increased by 7.6 percent. Difficulties inherent in introducing a new cigarette brand on the market are considered. Thus far, none of the 15 new brands marketed in 1979 took as much as 1 percent of the market.

82–0265. Milmoe, S. R. **U.S. Farm Exports at Record 41.3 Billion Dollars in 1980.** *Foreign Agriculture* 19(4): 19-20, April 1981.

Overall agricultural exports from the United States rose to another record-breaking level in 1980, with an export volume of almost 163 million metric tons (MT) and shipments valued at almost 41.3 billion dollars. Tobacco experienced an increase in export volume and value in 1980, though not to the point of breaking records. In 1976, tobacco exports were 269,000 MT, increasing to 285,000 MT in 1977 and 318,000 MT in 1978. In 1979, the figure decreased to 257,000 MT, but increased 6 percent in 1980 to was 940 million dollars, increasing to 1.094 billion dollars in 1977 and to 1.358 billion dollars in 1978. In 1979, the figure decreased to 1.184 billion dollars, but increased 13 percent in 1980 to 1.334 billion dollars.

82–0266. Neuber, D.; Rizio, D. **Light in Tar—High in Quality.** *Tabak Journal International* 4: 332, 334, 337, August 1981.

Since 1976 Lorillard sales have increased 25 percent, expanding its share of the total domestic cigarette market from 8.1 percent to 9.9 percent in 1980. Cigarettes account for 97 percent of company sales, the principal brands being Kent, Golden Lights, Triumph, Newport, True, Old Gold, and Max 100 mm's. The company, presently number five in the domestic market, also produces little cigars, small cigars, and chewing tobacco. In 1976, Lorillard began to concentrate its efforts on low-tar cigarettes, reducing the tar content of Kent from 25 mg to 12 mg, and that of True from 11 mg to 5 mg. Currently, 80 percent of all cigarettes sold by Lorillard are low-tar, compared to 20 percent in 1975. The company's image and thematic approach to advertising, rather than focusing on tar figures, presents sophisticated or glamorous images with which consumers wish to identify, for example: the Max 120 mm advertisements present a chic and elegant image for women; Newport shows fun-filled people and events in its campaign, aimed chiefly at young, urbane smokers; and Kent III's campaign focuses on the names and landmarks of major cities. The advertising budget of the company increased from about 39 million dollars in 1975 to 108 million dollars in 1980. In early January 1980 the company underwent the most massive sales reorganization in its history; and its Greensboro processing and manufacturing plant, which already produces 60 percent of Lorillard's cigarettes, is undergoing a 25 million dollar expansion to be completed by Spring 1982. The expansion is expected to increase by 30 percent the plant's cigarette producing capacity. The remaining 40 percent of present cigarette production takes place at the Louisville manufacturing and processing plant. Lorillard's aim is to overtake Philip Morris and R. J. Reynolds for the number one position in the U.S. cigarette market.

82–0267. Neuber, D.; Rizio, D. **A Small But Growing Segment.** *Tabak Journal International* 4: 318-319, August 1981.

The sales of long, thin 120 mm cigarettes represent a small (only 2 percent) but growing share of the tobacco market. Of the approximately 105 billion units sold in 1980, over 80 percent were purchased by female smokers, who may feel that such cigarettes fit well in their hands and look attractive. In addition, the number of actual and potential women smokers is higher than ever, as the "baby boom" generation comes of age. Further, advertising campaigns of some companies promote female preference for 100 mm cigarettes. R. J. Reynolds Company's More brand enjoyed a 67 percent share of 120 mm sales, with a 1.1 percent share of domestic sales in 1981, relying on undertones and double meanings to associate More with sex. Philip Morris' Saratoga, promoted by humorous unisex ads, has a 0.4 percent overall market share, while Lorillard's Max seeks to present an image of sophistication and style. Max held a 0.2 percent market share in 1980. Other brands include American Brands' Tall and Liggett & Myers' Eve Lights. Indications suggest that the popularity of 120 mm cigarettes will continue. First, the popularity of 120 mm cigarettes has developed slowly and is thus less likely to fade. Second, a Lorillard study done on Max and 120 mm smokers in general described such smokers as loyal to their brand.

82–0268. Overton, J. **Diversification and International Expansion: The Future of the American Tobacco Manufacturing Industry With Corporate Profiles of the "Big Six."** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 159-195.

The history of the tobacco industry is a study of marketing and advertising wizardry. Six major companies have dominated the American cigarette market since the turn of the century: R. J. Reynolds, Philip Morris, Liggett and Meyers, American Tobacco, Brown and Williamson, and Lorillard, each promoting a few reliable brands. The formula for success was simple: marketing strategies including catchy slogans and baseball cards aimed at creating smokers out of nonsmokers. Nonfiltered cigarettes were the rage, and there was little need for product innovation. In the 1950s, however, health concerns were identified, the number of new smokers declined, and cigarette manufacturers scrambled to capture slices of the now-stagnant market with new low-tar and filtered brands. By the late 1970s, confronted with a nonexpanding market, large cash reserves, and minimal long-term debts, the industry was stagnating, and the message from Wall Street was clear: expand into other product lines. R. J. Reynolds, still the leader in cigarette sales, branched off into oil and shipping concerns, and also owns Del Monte foods. American Tobacco, with a declining share of the cigarette market, changed its name to American Brands and is prospering in nontobacco lines. Philip Morris has triumphed at home and in Europe with its Marlboro brand, and hopes to do the same with its Miller beer and Seven-Up subsidiaries. Brown and Williamson is now part of BAT Industries, the 49th largest company in the world and producer of 20 percent of all cigarettes sold in the free world. Lorillard, which did not respond to the changing market, was taken over by Laurence Tisch. Liggett and Meyers remained in third place for 40 years, but failed to respond to the changing market of the 1950s. The company diversified well, however, and tobacco is now of decreasing importance to overall operations.

82–0269. Pohl, H.-G. **West Germany—Now Biggest Cigarette Market in the EEC.** *Tabak Journal International* 4: 260-264, August 1981.

About 3 billion more cigarettes were manufactured in West Germany in 1980 than in 1979, thus making that country the largest single producer in the European Community, displacing Great Britain. German companies supplied 127.1 billion cigarettes for the domestic market in 1980, on which consumers spent about 18.5 billion DM. Exports in 1980 reached 31.6 billion and imports about 2 billion. The Federal Government obtains about 6.4 percent of its income from cigarette taxation. Philip Morris GmbH of Munich, R. J. Reynolds GmbH, and B.A.T. Cigaretten-Fabriken GmbH of Hamburg increased their deliveries to the domestic market in 1980, where German smokers had a choice of 245 brands of cigarettes, although the greatest demand was confined to a few brands. Just under half the total market, for example, was represented by four brands. Menthol cigarettes and 100 mm cigarettes showed improvement in sales, reaching 1.6 billion and 2.2 billion in 1980, respectively. In 1980, there was a slight increase in the sales of "mild" cigarettes, defined as cigarettes containing a maximum of 0.8 mg of nicotine and 10 mg of tar. Tobacco product wholesalers purchased about 68 percent of all the cigarettes supplied by manufacturers, of which about 60 percent were sold to the public through vending machines owned by the wholesalers. The amended tobacco tax law, which went into effect January 1, 1980, was not intended to produce more revenue but to be compatible with European Community directives on tobacco taxation. The manufacturers voluntarily agreed to print health warning labels in newspaper advertising starting November 1, 1980; in poster advertising from February 1, 1981; and on cigarette packets from October 1, 1981.

82–0270. Pugh, C. **The Federal Tobacco Program: How It Works and Alternatives for Change.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 13-25.

In recent years, groups within and without the tobacco industry have called for revisions in the Agricultural Adjustment Act of 1938, which restricts the supply of flue-cured and Burley tobacco to keep the average price above the open-market level without direct subsidies. Antitobacco advocates point out the inconsistency of the Federal Government supporting both the tobacco industry and antismoking campaigns. Leaf exporters wonder if American tobacco is priced out of the international market, where many comparable grades are cheaper. The current tobacco program restricts supply and stabilizes prices by use of a national marketing quota, individual farm quotas based on production history, price supports, and governmental funding of nonrecourse loans. The tobacco program could be changed in any of three ways: (1) alter particular features without changing the general approach, (2) substitute individual provisions, or (3) move to essentially an open market in producing and marketing tobacco. One feature that could be changed would be to have marketing quotas set by marketing orders or agreements or by a marketing board instead of by governmental action. The assigning of farm quotas could be tied to current acreage rather than to a historic base. The price support system could be changed by adjusting the price support formula or the distribution of price supports among grades. Modifying individual provisions could include a temporary support level freeze, replacing the current formula with a mandatory parity level, using a general economic indicator as the escalator rather than the Parity Index, moderating the pace of increases in support rates, tying support rates to tobacco production costs rather than to general farm costs, using a "two-price" plan rather than the single formula, or broadening administrative discretion to allow overall price supports to be within some legal range. An open market could mean a drop in prices and redistribution of income. Tobacco demand tends to be inelastic; and if quantity increases by a certain percentage, farm prices might decrease by a higher percentage. An open market could have a number of effects: (1) a moderate production increase over the long run; (2) a drop in leaf prices; (3) a drop in the resale value of farms with quotas; (4) a decrease in income for those getting rental income from tobacco quotas; (5) a geographical shift to more efficient areas; (6) a more rapid decline in the number of tobacco farms; (7) an increase in the volume of tobacco exports; and (8) small decreases in consumer prices. The government also would not have to advance loan funds or absorb losses on price support operations. There may, however, be no actual reduction in smoking. Proposals to modify or abolish the tobacco program could thus have a wide range of effects.

82–0271. Pugh, C. **Landmarks in the Tobacco Program.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 31-36.

Landmarks in the tobacco program, including the Agricultural Adjustment Act of 1933, which created the first Federal tobacco program and encouraged farmers to reduce 1934 plantings in return for payments to stabilize supplies and prices, are discussed. The program became known as the Agricultural Stabilization and Conservation Service. In 1938, legislation established tobacco marketing quotas, providing penalties for overproduction. In 1939, national and State quotas were converted from poundage to individual acreage allotments and changed the base period for flue-cured parity price from 1919-1929 to 1934-1939. In 1946 the Flue-Cured Tobacco Cooperative Stabilization Corporation was created to receive tobacco from farmers when prices were not above support level. The Agricultural Act of 1948 modernized parity to reflect trends in relative prices of all farm commodities during the preceding 10 years. In 1960 Congress froze price supports at the 1959 level and established a formula for future levels based on the moving average of the Parity Index in the preceding 3 years. The Index incorporates inflation rates in overall farm costs. In 1961-1962, existing allotment holders were allowed to lease allotments within the same county for production on their own farm, first on an annual basis and later for leases up to 5 years. In 1964, the Surgeon General's advisory committee released a report on the possible association between health problems and tobacco. In 1965, individual farm acreage allotments for flue-cured tobacco were replaced with acreage and poundage quotas for each farm. The "tying" provision of marketing was replaced by "loose-leaf" sales beginning in 1968. Television and radio cigarette advertising was banned in January 1971, and warning labels were added to cigarette packages. In 1971 the Burley tobacco program switched from acreage allotments to poundage quotas. In 1977-1978, the Secretary of Health, Education, and Welfare proposed several Federal antismoking efforts. The Commodity Credit Corporation (CCC) loan system was altered in 1981, so that the CCC now adjusts the interest rate to the prevailing market level (for loans to the cooperatives) twice a year instead of once.

82–0272. Seshagira Rao, P. **India's Tobacco Export Trade: Growth From 1970 to 1980.** *Tobacco International* 183(15): 105-106, 110, 114, 119, 122, 124, 126, July 24, 1981.

From 1970 to 1980 India produced 4.03 billion kilograms of all types of tobacco, of which 1.2 billion was flue-cured Virginia and 2.83 billion consisted of other varieties for use in cigars, cheroots, cigarettes, chewing tobacco, bidis, hookas, snuff, and other products. About 1.035 billion kg (green weight) were exported in unmanufactured form and 57 million kg (green weight) as manufactured products. Total exports in 1970-71 were 47.5 million kg, rising to 94.48 million kg in 1972-73 and fluctuating between 70 million and 80 million thereafter, until 1979-80, when exports dipped to 64.8 million kg. Earnings rose steeply from 1970 to 1980, due more to price increases than to export volume increases. The average unit price of exports rose from Rs 6.60 a kg in 1970-71 to Rs 15.35 a kg in 1979-80. Although the highest volume of 94.48 million kg occurred in 1972-73, the highest foreign exchange earning from exports was in 1977-78 at 110.7 crore rupees. India's main export customers for 1970 to 1980 were: Great Britain (about 33.7 percent of India's tobacco export volume and about 40.6 percent of its value); the U.S.S.R., (25.56 percent of volume and 28.76 percent of value); and Japan, Bangladesh, Bulgaria, Iraq, Italy, Portugal, and China. Repeated attempts to enter the West German market were relatively unsuccessful. Quality control of tobacco exports has been operated by the Tobacco Grading Scheme since May 1945. In 1963, minimum price control for different grades was introduced to curb unhealthy competition among merchant exporters and to prevent underinvoicing of export tobacco. The Minimum Price Control Scheme has been operated by the Tobacco Board since 1975, requiring a grading certificate and a price certificate for each export consignment. The fixing or revising of minimum export prices considered factors such as production cost at farm level; cost of handling, packing, and transport at export level; and trend of foreign demand. The Tobacco Board hopes to devise a long-term strategy for minimum export price revisions, and may carry prices and grading to the primary market, creating a minimum farm price.

82–0273. Rizio, D. **Philip Morris Fortifies Its Worldwide Stamina.** *Tabak Journal International* 4: 321-323, August 1981.

The continued success of Philip Morris Inc., reported for the year 1980, shows the company's operating revenues, net earnings, and earnings per share grew for the 27th consecutive year. It remains a leader in the cigarette, beer, and soft drink industries. Both Philip Morris U.S.A. and Philip Morris International strengthened the company's number two position in the domestic and overseas tobacco markets. Philip Morris U.S.A.'s share of the cigarette market rose from 29 percent in 1979 to 31 percent in 1980, due to the increasing popularity of Marlboro, which again became the best-selling brand in the United States and the world. Merit proved to be another popular domestic brand, growing by 14.6 percent in 1980. Philip Morris International's export sales increased 10.4 percent from 1979 to 1980, making it the largest exporter of cigarettes from the United States for the fourth consecutive year, with brands manufactured in 51 countries marketed in over 170 countries and territories. Construction of a new cigarette industrial plant in Carrabus County, North Carolina, continued on schedule, while more than 100 million dollars were spent to expand and modernize facilities in Bergen op Zoom, The Netherlands, and in West Berlin. The company's Argentine affiliate merged with two affiliates of Reemtsma GmbH. Philip Morris (Australia) Ltd. introduced Marlboro Red and Marlboro Special Mild, and a new agreement for the sale and distribution of Philip Morris products at hard currency outlets in major cities was signed with the People's Republic of China. Philip Morris continues to pride itself on employee relations and also continues to support and sponsor academic and artistic programs and public relief causes.

82–0274. Samfield, M. **Explosive Shift to Low Tar Cigarettes in the U.S.A.** *Tabak Journal International* 4: 314-316, August 1981.

Low-tar cigarettes, defined as those with a tar level below 15 mg, have increased their share of market sales in recent years from 1 percent in 1964 to an estimated 28 percent for 1981, with a possible 40 percent share by 1984. The median tar content of all cigarettes sold also has been declining and may approach 18 mg by the end of 1981. Since the 1964 U.S. Surgeon General's Report, the annual growth rate in the cigarette market has slowed from 3.5 percent to 1.5 percent. Changing public attitudes and a cigarette tax increase may also have contributed to slowed market growth. Cigarette companies have changed their market strategy, manufacturing practices, and related variables to cope with the new trends. Since the Federal Trade Commission (FTC) began publishing the tar and nicotine content of cigarettes, companies have introduced many new low-tar brands, with most aimed at the 5 to 12 mg market, since tobacco people feel that customers buying cigarettes with less than 3 mg are on the verge of quitting anyway. Advertising emphasis shifted from association of cigarette smoking with "health images" to tar and nicotine figures. At present there are three major commercially successful ways to control the tar content of cigarettes: (1) altering the cigarette to burn faster by methods such as decreased size, different blend, or faster burning cigarette paper; (2) diluting cigarette smoke to lower yields (porous cigarette paper, perforated paper, or a combination of perforated tipping paper and porous plug wrap); and (3) improving filtration efficiency, by increasing the length or diameter of the filter, increasing the filter pressure drop, decreasing the filter tow diameter, or using special filters. Future market trends are difficult to predict, but the low-tar cigarette seems to be here to stay. Smokers are now more accustomed to cigarettes with lower yields. Any significant increase in cigarette smoking does not appear likely in the present public climate.

82–0275. Shien, T. **JTS Sales Hurt by Tax Hike; Leaf Poor, Imports From N. America Up.** *Tobacco International* 183(18): 9-10, 12-13, September 4, 1981.

During the Japanese 1980 fiscal year (JFY, April 1980 to March 1981) the Japan Tobacco and Salt Public Corporation (JTS) sold a total of 304 billion cigarettes, a decrease of 2.9 billion from JFY 1979 sales, attributed chiefly to an average 21 percent increase in the retail price. The value of sales for JFY 1980, however, increased to 2,328 billion yen from the previous 1,966 billion yen, due mostly to the price rise. Filter cigarettes (36 of the 40 brands) accounted for 98.1 percent of the market in JFY 1980. There was a decrease in JTS imports of tobacco products of 330 million units, and an increase in exports of 3 percent. Imports (92 percent) were mainly from the United States, while most of the exports went to Southeast Asia; although the United States, Singapore, and Taiwan showed a significant increase. The JTS worked with national monopolies in France, Austria, and Italy to produce the new Champagne brand, introduced in all four cigarette markets simultaneously in March 1981. The complete 1980 tobacco crop (141,000 tons, down 8 percent from 1979), although affected by bad weather, producing an immature, thin-bodied crop, was bought by the JTS. Leaf imports by JTS in JFY 1980 were 71,418 tons, an increase of 6.3 percent over the previous year and a 5-year high. Over 300 billion cigarettes are manufactured yearly by JTS's 37 cigarette manufacturing plants, 12 leaf processing plants, and 5 sheet tobacco plants, which use modern high-speed machinery and have extensive computerization.

82–0276. Singh, A. N.; Rajendran, N.; Seethamma, M. **Tobacco Situation and Outlook in the Republic of Korea.** *Indian Tobacco Journal* 12(1): 6-9, April-June 1980.

A comparative newcomer to tobacco production and trade, Korea has emerged as an important source of leaf in the last decade, ranking ninth in total production, eighth in flue-cured, and fourth in Burley tobacco. Three types of leaf are cultivated: Virginia flue-cured, Burley, and native types (sun/air-cured). The Office of the Monopoly, Republic of Korea (OMROC) administers the industry, including marketing, trade, and manufacturing. The monopoly's goals are to: (1) raise the quality of Korean tobacco and products to international level, (2) elevate the technology for cigarette manufacture, (3) maximize the efficiency of management and productivity, and (4) expand exports. Seeds, equipment, and chemicals are supplied by the monopoly. Farmers have also received an average price increase of 20 percent over the past 6 years. South Korea currently has 7 tobacco processing plants and 11 drying plants (7 jointly owned with foreign investors). A new large research institute has been established to coordinate Korean research efforts, and it enjoys a mutual exchange of information with foreign countries. Production figures for 1960 to 1979 are given for the different varieties grown, and export statistics are provided for type of tobacco and country of destination.

82–0277. Sticht, J. P. **R. J. Reynolds Industries: A Hundred Years of Progress in North Carolina.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 197-198.

R. J. Reynolds Industries, a company started in 1875 to market chewing tobacco, is now a diversified worldwide business. Prince Albert smoking tobacco was introduced in 1907 and is still the best-selling smoking tobacco in the United States. By 1913, the company was the leader in the manufacturing and marketing of tobacco. Reynolds' first blended, nationally marketed cigarette, Camel, remained the company's flagship brand for almost 5 decades and still ranks seventh among all cigarettes sold. Winston, introduced in 1954, led the growth in filter cigarettes, and in 10 years was the best-selling cigarette in the United States; today Winston ranks second. Salem, the first filter-tipped menthol cigarette, appeared in 1956 and ranks third in the country today. It is felt that in the 1980s the domestic market will experience years of declining or no growth, with the smoking and health controversy a contributing factor. R. J. Reynolds Industries continues to support scientific research on the health hazards. Company diversification began in the 1950s when the Archer Aluminum Division began making packaging products for customers other than Reynolds Tobacco, and has continued into such areas as food, snacks, beverages, energy, and transportation. R. J. Reynolds affirms a continuing commitment to its workers and the support of educational and cultural institutions in North Carolina.

82–0278. Tornquist, E. **Labor Displacement in Tobacco Manufacturing: Some Policy Considerations.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 221-228.

The steady decline in tobacco manufacturing jobs over the past decade is especially evident in North Carolina, the nation's leading tobacco manufacturing State, where the number of workers fell from 31,850 in 1965 to 25,100 in 1979. The reductions, through attrition, gradual layoffs, and plant closings, may accelerate in the future, as companies that do well increase their mechanization, and those that do not, have to reduce their work force. Philip Morris and R. J. Reynolds, for example, although expanding, are relying on more sophisticated technology instead of a larger work force. Third-place Brown and Williamson closed its Louisville plant and reduced its Petersburg work force, but in a manner considered as a good model: the plant closure notice was given 18 months in advance, and the work force reduced over a 3-year period. The company also provided programs to assist workers in finding new jobs. Speculation is that Liggett and Myers, also reducing its work force, may even get out of the cigarette business. It is suggested that the tobacco industry be required to give advance notice of layoffs, and that an effort should be made to create new jobs in developing or emerging sections of the economy. To this end, the role of technical schools' training programs could be studied. State- and Federal-level policy planning might also be useful in helping displaced workers.

82–0279. Weissman, G. **A Future of Great Promise—for Tobacco and for Philip Morris.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 199-202.

The Philip Morris Company has undergone great changes in the previous two decades, becoming first an international company and then diversifying. The company, which now markets more than 160 different cigarette brands in more than 170 countries and territories, has 31 manufacturing and marketing affiliates, 34 licensees, and various regional export organizations. It is also the world's largest cigarette exporter with over 40 percent of the U.S. cigarette export market. In the 1970s, Philip Morris acquired the Miller Brewing Company and the Seven-Up Company; and is also active in packaging and paper, and land development. The company moved into fields where it could apply expertise learned in the tobacco industry. Cigarettes, beer, and soft drinks are large, profitable, recession-resistant industries; they are agriculturally based consumer products; and their sales are responsive to product innovation and to imaginative advertising. Tobacco remains the dominant business however, accounting for 64 percent of total revenues and 82 percent of operating profits in 1979. The U.S. market for cigarettes will continue to be large, but appears to be maturing. International sales (40 billion cigarette units in 1980) are expected to account for most future tobacco market growth, and it is felt that the smoking and health controversy will not adversely affect such prospects. In 1979, foreign sales of U.S.-grown tobacco and tobacco products generated 1.7 billion dollars in net trade surplus. Philip Morris continues to expand its facilities and to support research.

See also, **82–0061, 82–0235, 82–0282**

LEGISLATION

82-0280. Ehringhaus, J.C.B., Jr. **Some Legal Aspects of the Smoking-and-Health Controversy.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 279-285.

Legal aspects of the smoking and health controversy, including efforts to ban smoking in public places, government regulation of cigarette advertising, and attempts to regulate cigarettes under the Federal food and drug laws, are discussed. The greatest debate has centered around attempts to ban or restrict smoking in public places such as restaurants, offices, stores, or sports arenas. When such arguments have been brought to court, the antismokers' wishes have usually been rejected. Some State legislatures as well as county and city councils have adopted proposed statutes and ordinances, but many localities have rejected them. Antismokers sometimes claim a Constitutional right to a smoke-free environment; however, smokers may claim a Constitutional right to smoke, if they do so in a reasonable manner. It is not yet certain whether smoking causes harm to healthy nonsmokers. Smoking aboard airplanes has received particular attention: the Civil Aeronautics Board issued regulations in 1973 requiring that nonsmoking sections be designated on airliners, and antismoking advocates have continued to seek increased smoking restrictions aboard aircraft. Changes can be expected in the field of cigarette advertising as government agencies such as the Federal Trade Commission (FTC) attempt to further restrict cigarette advertisements or place new requirements on advertisers. Television advertisements for cigarettes are prohibited by law, and a consent agreement between manufacturers and the FTC resulted in cigarette warnings being included in newspaper and magazine advertisements, billboards, and other printed promotional materials. Manufacturers voluntarily included tar and nicotine information in advertisements. Some States and localities have also sought to regulate cigarette advertising and promotion. Some antismoking advocates recently asked that the Food and Drug Administration (FDA) regard cigarettes as drugs or medical devices, subjecting them to regulation. The FDA denied the request; a decision upheld in court.

82-0281. Finger, W. R. (Editor). **Appendix 27A. Major Actions Taken by Countries on Smoking and Health.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 304-309.

The major actions taken on smoking and health by representative developed and undeveloped countries are listed under: advertising, health warnings, restrictions, education, and other. In the United States, listed actions include: (1) advertising—the ban on cigarette advertisements on radio and television; (2) health warnings—the requirement for a warning label on cigarette packs and in advertisements; (3) restrictions—nonsmoking sections on aircraft and interstate buses, and the restriction of smoking in public places by more than 30 States; and (4) education—antismoking efforts, primarily by private organizations, as well as some State-supported school education programs. No "other" actions are listed. Many countries have stronger restrictions on advertising than the United States (Bulgaria, Canada, Finland, France, Iceland, Ireland, Italy, Norway, Netherlands, Spain, Sweden, United Kingdom, and the Soviet Union). In the areas of health warnings, restrictions, and education, there are wider variations reported. In the "other" category, the countries with additional areas of regulation are: Finland (government limits on tar, nicotine, and carbon monoxide content); Norway (comprehensive legislation passed in 1975); Sweden (1975 legislation considered the strongest in the world); and the United Kingdom (extra tax on high tar and nicotine cigarettes).

82-0282. Harden, B. **In the Public Interest . . . Not a Constitutional Birthright: An Interview With Former U.S. Secretary of Agriculture Robert Bergland.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 319-323.

Policies concerning the USDA price-support system for tobacco are discussed by former U.S. Secretary of Agriculture Robert Berglund. The USDA has stayed clear of the HEW enterprise to educate America on the pitfalls of smoking, and vice versa. USDA policy is that it is in the public interest to preserve the price-support program, as it maintains about 600,000 families. An objective of the program is saving the small family farm; however, price supports benefit large farms more than small ones. This issue is to be examined in 1981. The 1981 Omnibus Farm Bill will mean that the price-support program will be renewed periodically, like most farm-support programs. Plans are also underway to amend the price differentials so that lower grade leaf does not accumulate due to overpricing. As health concerns may eventually reduce tobacco demand, development plans are targeted for rural areas to provide economic alternatives for farmers forced to give up tobacco growing.

82-0283. Humphries, B. **Eagleton Tobacco Bill: Is It Thought Through Sufficiently?** *Tobacco International* 183(18): 24, 26, September 4, 1981.

Criticisms of the proposed Amendment No. 529 to the 1981 Farm Bill S.884 are presented. The amendment would prohibit nonproducers of tobacco from owning allotments. Its stated purpose is to assist tobacco producers by reducing the cost to those who have to lease allotments. Another proposed amendment would authorize the U.S. Secretary of Agriculture to adjust price supports for certain kinds of tobacco. The allotment amendment would separate the allotment or quota from the land and assign the allotment to those who have most recently leased it for actual production. Persons who received over half their income from nonfarming sources and who will not have planted their allotment in 2 out of 3 years from 1980 onwards would lose their allotments. Corporations would lose their allotments unless they were composed of one or more people engaged in farming and who produce tobacco on the corporation's allotment. A person could not lease or transfer his allotment, and would lose it unless he planted and produced it for 2 consecutive crop years. There are questions about what sources of income would be used to distinguish farmers and nonfarmers. Possible interpretations mentioned so far would include income from the sale of farm commodities and rentals received from farmland as farm income. Social Security and other retirement income might be considered as total income and the status of income from the lease and transfer of tobacco quota off the farm is unclear. There are also questions about the conditions under which an allotment holder might meet the planting requirement. One criticism is that an individual nonproducer could do little at present to change his status to producer, since 2 of the 3 years in which to achieve a 2-year planting history are already gone. No compensation is provided for those who lose allotments. A grower who leased and transferred tobacco quota the past 2 years would not be considered as "planted" and might lose his allotment. It is suggested that planting requirements to keep an allotment be based on 1982 and future years, giving more choice for people to resume tobacco planting if so desired.

82–0284. Joseph, J. D.; Kramer, M. S. **Legal and Policy Issues Concerning Smoking and Health To Be Faced in the 1980s.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 287-293.

Strong national trends toward restricting smoking in public places, including shopping areas, transportation vehicles, and government buildings, and efforts to regulate smoking in the workplace which have recently begun to gain momentum, are discussed. Also, expected efforts by Congress to strengthen its restrictions on cigarette advertising, require more stringent health warnings, and consider establishing safety standards for cigarettes are reviewed. Although nonsmokers have gained many new protections in the last decade—only some of which are catalogued here—many smoking and health concerns remain. Battles between tobacco advocates and nonsmokers' groups will continue throughout the 1980s on a wide range of legislative, judicial, and administrative fronts. (Auth. Abs.)

See also, **82–0251**

82–0285. Koplin, A. N. **Anti-Smoking Legislation: The New Jersey Experience.** *Journal of Public Health Policy* 2(3): 247-255, September 1981.

New Jersey's experience with antismoking legislation is an illustration of the nationwide campaign to prevent smoking. In 1974, the State's Public Health Association launched a program to reduce public smoking, and drafted a model code under which smoking would be restricted in certain places on a voluntary basis. By 1976, the provisions of the code were placed in regulatory language mandating limitations on public smoking, and in 1977 the Attorney General's office verified the authority of the Public Health Council to promulgate the regulations which had become chapters of the State Sanitary Code. The proposed regulation was published in the New Jersey Register of October 6, 1977, and a hearing scheduled. The debates and hearings continued for several years; the time required for passage of an antismoking regulation totaled 5 years. The major impediments to passage involved conflict between the authority of the regulatory body and the legislature, and included economic and enforcement considerations. The Department of Health staff carried out much of the analysis and prepared the case, countered by the New Jersey Tobacco Distributors, American Tobacco Institute, the Restaurant—Motel Association, and others. The opposition was based primarily on economic grounds.

82–0286. Warner, K. E. **State Legislation on Smoking and Health: A Comparison of Two Policies.** *Policy Sciences* 13: 139-152, 1981.

Two principal smoking-related State legislative activities stand in sharp contrast to one another. The one related to cigarette excise taxation was diffused among the States well before the connection between smoking and illness became a public issue, yet more recent tax increases appear to reflect a response to the national antismoking campaign. The growing disparity in cigarette prices between States has created a lucrative market in bootlegged cigarettes and has thereby brought new taxation to a virtual standstill for 6 years. By contrast, laws restricting smoking in public places represent a phenomenon of the 1970s and clearly bear the imprint of the antismoking campaign. From 1972 through 1978, the number of States with such laws in effect grew from 5 to 36, and the restrictiveness of the laws also increased. The dramatic correlation between the growth in smoking-restriction legislation and decreases in cigarette consumption rates seems best interpreted as a behavioral response to the entirety of the antismoking campaign. (Auth. Abs. Mod.)

GENERAL

82–0287. Buckley, W. F., Jr. **The Weed.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 325-326.

The government's responsibility toward smoking is examined. Three factors are identified as related to an early death—smoking, lack of exercise, and overweight. The feasibility of the government taking charge of our health by enforcing restrictions on any of these three parameters is quite unrealistic. In relation to smoking and while not endorsing a law requiring the cigarette companies to advertise the dangers of smoking, it is suggested that within the laws of a free society, any company that does not make known the dangers of the use of its product become liable to damage actions by victims. It is concluded that: "It pays to remind ourselves that the most important things of all are up to the individual, who can opt, after all, for heaven or hell."

82–0288. Carnegie-Mellon University **Fire Safety: A Comparative Risk Assessment.** Pittsburgh, Pennsylvania, Carnegie-Mellon University, May 1981, 146 pp.

The problems of upholstered furniture fires, fire safety devices, and the risk of fires following an earthquake are discussed with a view to formulating effective policies in these areas. In the case of upholstered furniture fires, attention is directed to alternative strategies to reduce risks. These strategies include cigarette modification, mandatory and voluntary furniture standards, and public education programs. Cigarette modification involves creating a cigarette less likely to cause such combustion, for which several patentable and "nonpatentable" processes exist. The Colite process, a self-extinguishing cigarette method, treats cigarette papers with sodium silicate, an inert substance which promotes slower burning rates and lower cigarette end (or "coal") temperatures. Such a cigarette will self-extinguish after several minutes. Alternative methods available include modifying tobacco composition to alter its burn rate, and creating lower porosity paper with or without the addition of a burn retardant. The possibility that cigarette modification could raise tar, nicotine, or toxic gas levels is not considered significant, while the self-extinguishing cigarette might significantly reduce the risks to nonsmokers. If the tobacco industry, which has successfully resisted governmental regulation, cannot demonstrate significant progress in the development of a modified cigarette, alternatives suggested are: (1) the adoption of the Cigarette Safety Act, which would allow the Consumer Product Safety Commission to set safety standards for all cigarettes manufactured in the United States; (2) a tax on brands that fail to meet a minimum safety level, with revenues used to finance educational programs; and (3) court cases against manufacturers for failure to produce a safer cigarette.

82–0289. Dixon, B. **Where There's Smoke.** *Sciences* 21(3): 29-30, March 1981.

A recent book by Professor Hans Eysenck (*The Causes and Effects of Smoking.* Beverly Hills, California, Sage Publications, 1980, 394 pp.), which argues that although smoking and lung cancer are suggestively associated they may not be causally linked, is reviewed. A propensity to develop lung cancer and to smoke might be the results of a third, inbred factor or endogenous cause. Criticisms of the book's interpretations are presented. Although the book mentions that the correlation between smoking and lung cancer rates in some countries is considered weak, and that the smoking—heart disease association can include inconsistent dose—response relationships and racial differences in cardiac response to smoking, such points merely confirm that smoking-associated diseases are influenced by several factors. Although agreeing with the concept of personality as a factor, criticisms are made of the reasoning concerning smoking as a cause of considerable mortality and morbidity: the existence of carcinogens in tobacco smoke, and the increase in lung cancer among women being cited as evidence of the causal hypothesis. See also 81-1014.

82–0290. Gloag, D. **How Much More Can We Do About Air Pollution?** *British Medical Journal* 282(6266): 805-808, March 7, 1981.

The most important air pollution components at present are carbon monoxide, nitrogen dioxide, ozone and other photochemical oxidants, and polycyclic aromatic hydrocarbons. The components may come from outdoor sources such as industry and traffic, or may be indoor pollutants such as smoking. Smoking is an important source of carbon monoxide, commonly giving the smoker a carboxyhemoglobin concentration of 5 to 15 percent or more, whereas nonsmokers even when exposed to street air rarely go above 3 percent. Smoking is also a source of nitrogen dioxide, with inhaled tobacco smoke containing as much as 226 mg/m^3. Smoking appears to be a much more important cause of lung cancer than city pollution and may also cause lung cancer in nonsmokers who are constantly exposed. A smoky atmosphere with 44 mgl carbon monoxide increases the mean carboxyhemoglobin concentration of nonsmokers from 1.6 percent to 2.6 percent (compared with 5.9 percent to 9.6 percent in the smokers). It is important to keep indoor pollutants such as tobacco smoke to a minimum.

82–0291. Gori, G. B. **Research Toward a Less Hazardous Cigarette.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 345-365.

Over 50 million Americans still choose to smoke in spite of many years of antismoking education. Public health efforts are needed to protect individuals who continue to smoke. Considerable evidence suggests that this goal can be accomplished in at least two ways. First, total intake of smoke should be reduced; and second, it may be possible to selectively remove toxic smoke components, thus reducing specific smoking hazards. Reducing intake and toxicity of smoke means reducing the total particulate matter in the smoke. This may be accomplished by: (1) increasing combustion efficiency, which would also increase water and carbon dioxide yield and lower output of tar and carbon monoxide; (2) filtration; (3) selection of certain tobacco varieties and cultivation techniques; (4) use of filler, reconstituted, expanded, or freeze-dried leaf; (5) increasing the porosity of cigarette paper; and (6) use of artificial tobacco substitutes.

82–0292. Guillory, F. **The Politics of Tobacco in North Carolina: "A Load Not Easy To Be Borne."** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 313-318.

North Carolina politicians are being forced to change their strategies as a result of growing antismoking pressures, which has made defending tobacco more difficult. The issues facing the politicians are: (1) domestic cigarette sales may decline if fewer people smoke due to antismoking efforts; (2) North Carolina growers must compete with less expensive high-quality foreign tobacco; and (3) Congress is less dominated by powerful southerners sympathetic to the tobacco industry. North Carolina leaders dispute less frequently than before the link between cigarette smoking and disease, and argue more for tobacco and its Federal price-support system as a means of preserving small family farms and rural culture. The tobacco price-support system is the only commodity program with permanent authorization in Federal law. As a result, tobacco-state congressmen do not have to work to renew the agricultural programs vital to their constituents, but only to defend the tobacco program against challenges. North Carolina congressmen can build coalitions of agricultural interests to back tobacco. North Carolina State leaders have sought to expand industrialization in rural areas and to promote agricultural diversification. These policies suggest a recognition that tobacco might play a diminished role in North Carolina's economy and politics in the future, though State politicians do not appear to be admitting it publicly. Further preparation may be needed if tobacco's importance in North Carolina does decline.

82–0293. Kestenbaum, H. **Resources on Smoking and Health.** In: Finger, W. R. (Editor). *The Tobacco Industry in Transition.* Lexington, Massachusetts, Lexington Books, 1981, pp. 295-302.

A selected bibliography on smoking and health is presented, containing an annotated listing of the primary references available on different facets of the smoking and health controversy. Topics under which references are listed include research methodologies, health studies, passive smoking, the less hazardous cigarette, legislation and public policy, State legislation, and international policy. Also included is a listing of selected organizations and agencies concerned with smoking and health. A chronology of major Federal actions and events on smoking and the public environment from the 1964 Surgeon General's Report through the March 1979 ruling by the General Services Administration prohibiting smoking in classrooms, auditoriums, and conference rooms is provided. A section of international regulations on smoking completes the unit.

82–0294. Sullivan, S. L. **University of Kentucky—Centre for Tobacco Research.** *Tabak Journal International* 4: 338-339, August 1981.

The Tobacco and Health Research Institute at the University of Kentucky was created by the State legislature in 1970. The Institute's purpose is "to prove or disprove the charges against tobacco products and to preserve and strengthen tobacco programs in this Commonwealth." The Institute's experimental cigarette laboratory formulates and standardizes Kentucky Reference Cigarettes, which serve as a universal reference tool and are available at reasonable cost to scientists upon request. There is also a Scientific Information Section where about 40,000 published articles relating to tobacco and health are available for those who wish to retrieve information. The Institute's laboratory equipment includes an electron microscope for examining the ultra-structure of animal tissues to determine the effects of tobacco smoking. Other departments are: the Hematology and Smoke and Leaf Chemistry Laboratories, Biostatistics, Computer Services, and Animal Care facilities. The present research program's goals are focused on smoking as it relates to cardiovascular and pulmonary diseases (chronic bronchitis and emphysema), passive smoking, the behavioral aspects of smoking, and product modification. The Tobacco and Health Research Institute has designed several unique devices for tobacco research, including smoking machines and smoke chemistry equipment. Its work has also resulted in modification of curing and cultivating practices, breeding techniques, and pesticide and sucker control levels. The diversity and scope of the Institute's work illustrate its continuing objectives, to work for the development of less hazardous smoking products and to ensure tobacco continues as an important part of Kentucky's economy.

82–0295. Tabak Journal International. **U.S.A.: Smokeless Tobacco Targeted.** *Tabak Journal International* 4: 350-351, August 1981.

Criticisms are presented of an earlier report (*New England Journal of Medicine* 304(13): 745-749, March 26, 1981) which concluded that habitual users of snuff and chewing tobacco are nearly 50 times more likely than nonusers to contract some form of oral cancer. The report was of a 2-year study ending in August 1978, in which the interview responses of 255 female oral cancer victims in the North Carolina area were compared with those of 502 healthy women from the same region. In that area, about 30 percent of white females use snuff. The Smokeless Tobacco Institute, New York, on behalf of the industry, questions the report's findings. The report stated only the area where the products are used, without describing the type of products or mentioning if they were used for a long period of time. Since few women in the United States use moist snuff or chewing tobacco, the report must be referring to dry snuff, which is taken orally instead of being sniffed as in some European countries. A 1970 study in the Archives of Otolaryngology examined the oral history of 16,000 snuff and chewing tobacco users and found no definite link between cancer and smokeless tobacco use. The Surgeon General's 1979 annual report also found no such link. The Tobacco Institute (Washington, D.C.) criticizes the report for not considering the dental care and diet of the subjects. See also 81-0788.

82-0296. Weis, W. L.; Fleenor, C. P. **Cold-Shouldering the Smoker.** *Supervisory Management* 26(9): 31-35, September 3, 1981.

The suggestion that smokers may be experiencing a growing negative bias in the job interview process and on the job itself is investigated. A survey of 223 Seattle-area managers found 119 would choose nonsmokers over smokers if they were equally qualified, 103 had no definite preference, and 1 preferred smokers. The managers were then asked to make two assumptions, that smokers have about a 50 percent higher absenteeism rate than nonsmokers, and that the health of nonsmokers who work around smokers is measurably impaired. The number choosing nonsmokers increased to 198, 23 were undecided, 1 had no response, and 1 still preferred smokers. Regarding the subject of a legal right to smoke on or off the job, the report draws a distinction between rights based on "ascribed" qualities such as race or national origin, which are protected by law, and rights based on "achieved" qualities, perhaps including smoking, which are not thus protected, in the report's opinion. The Seattle managers were asked to imagine themselves on the Supreme Court, deciding an appeal from an employer who was denied the right to ban smoking on his company premises, and from an employer who was denied the right to hire only nonsmokers. Without the assumptions of absenteeism or nonsmoker health effects, the managers ruled by 68 to 31 percent that an employer could ban smoking from company premises. They denied the employer's right to hire only nonsmokers by 62 to 37 percent. When the assumptions were added in, the managers voted 168 to 54 in favor of the employer's right to ban smoking on company premises and 118 to 105 against an employer's right to hire only nonsmokers. Finally, the managers were asked their preferences for one of three alternative policies regarding smoking at their respective organizations. Of the 223, 74 preferred a complete ban, 123 preferred separate areas for smokers and nonsmokers, and 26 preferred no restrictions.

AUTHOR AND ORGANIZATIONAL INDEX

Abelmann, W. H., 82–0125
Adams, F., 82–0241
Agnati, L. F., 82–0032
Agnew, T. M., 82–0127
Al-Mansour, A. H., 82–0068
Andersen, R. A., 82–0012
Anderson, A. E., Jr., 82–0060
Anderson, A. J., 82–0120
Anderson, M. J., 82–0111
Anderson, W. H., 82–0063
Andersson, K., 82–0032
Andrasik, F., 82–0128
Angevine, D. M., 82–0143
Angst, J., 82–0212
Arenella, L., 82–0125
Armaganidis, A., 82–0158
Aru, G., 82–0088
Asanuma, Y., 82–0099
Ashenberg, Z. S., 82–0216
Ashley, M. J., 82–0160
Ashton, H., 82–0033
Atkinson, W. O., 82–0004

Bachman, J. G., 82–0174
Bachner, I., 82–0034
Bachner, J., 82–0034
Back, D. J., 82–0036
Badger, A. J., 82–0242
Baettig, K., 82–0038
Baker, J. L., 82–0014
Barbee, R. A., 82–0151
Barbieri, M. T., 82–0088
Barboriak, J. J., 82–0120
Barboriak, P. N., 82–0120
Barford, M., 82–0001
Barratt-Boyes, B. G., 82–0127
Baskevitch, N., 82–0226
Bassfield, R. L., 82–0029
Bawol, R. D., 82–0112
†Bayer Aktiengesellschaft, 82–0230
Beaglehole, R , 82–0110
Beaver, C., 82–0210
Becker, J. J., 82–0227, 82–0228
Becker, M. H., 82–0183
Beleslin, D. B., 82–0035
Benfari, R. C., 82–0218
Bentler, P. M., 82–0173
Berardi, G., 82–0243
Berman, N. D., 82–0111
BfAI-Report., 82–0244
Bialer, M., 82–0013
Bielawiec, M., 82–0054
Bing, O.H.L., 82–0125
Black, A., 82–0107
Block, P. C., 82–0114
Bochenek, W., 82–0124
Bolin, J. F., 82–0091
Bonini, S., 82–0087
Bosl, G. J., 82–0136
Bosley, A.R.J., 82–0137
Boudene, C., 82–0140
Bousquet, J., 82–0096
Boyd, C. H., 82–0048
Boyko, E., 82–0129
Brandt, P.W.T., 82–0127
Breckenridge, A. M., 82–0036
Bresnick, E., 82–0067
Bright, M., 82–0063
Brille, D., 82–0157
†British-American Tobacco Co. Ltd., 82–0231
Broekelmann, T., 82–0059
Brooks, J., 82–0191
Brostoff, J., 82–0087
Brown, R. A., 82–0210
†Brown & Williamson Tobacco Corp., 82–0232
Bryant, L. R., 82–0092
Buckley, J. D., 82–0064
Buckley, W. F., Jr., 82–0287
Buda, A. J., 82–0111
Bulteau, V. G., 82–0152
Burch, P.R.J., 82–0112
Bureau of Drug Education, New York State Education Department., 82–0192
Burkhardt, A., 82–0104
Burrows, B., 82–0151
Burry, R. D., 82–0245
Burton, H. R., 82–0012

Caddy, G. R., 82–0178
Campbell, C. R., 82–0010
Campbell, J. S., 82–0246, 82–0247
Canadian Tobacco Grower., 82–0161
Carnegie-Mellon University, 82–0288
Carosone, G., 82–0211
Carr, D. B., 82–0083
Carson, R., 82–0089
Carta, P., 82–0088
Carter, N. L., 82–0152
Castelli, W. P., 82–0074
Castillo-Staab, M. I., 82–0133
Casula, D., 82–0088
Chalmers, J., 82–0094
Chamberlain, W. J., 82–0014
Chang, J., 82–0102
Chaplin, J. F., 82–0010, 82–0031
Chapman, S., 82–0162
Chavdarian, C. G., 82–0029
Choquet, M., 82–0165
Chortyk, O. T., 82–0014
Chowdhary, B., 82–0176
Christen, A. G., 82–0153
Chu, F. Z., 82–0193
Chwistecki, K., 82–0124
Clairmonte, F. F., 82–0248
Clement, F., 82–0154
Clot, J., 82–0096
Cocco, E., 82–0088
Cocco, P. L., 82–0088
Cogswell, J. J., 82–0138
Cole, P. V., 82–0118
Cole, R. J., 82–0198
Collins, G. B., 82–0002, 82–0005
Collins, W. K., 82–0003
Connellan, S. J., 82–0089
Connors, N. A., 82–0055, 82–0134
Conrad, L. J., 82–0229
Conti, M. C., 82–0070, 82–0082
Conway, T. L., 82–0163
Copeland, G., 82–0030
Corbett, L. O., 82–0223
Costas, R., Jr., 82–0133
Council on Scientific Affairs., 82–0155
Crawford, F. E., 82–0036
Crofton, J. W., 82–0094
Crooks, J., 82–0039
Crutchfield, R. D., 82–0164

Da Silva, A.M.T., 82–0090
Dagle, G. E., 82–0083
Dahms, T. E., 82–0091
Dalton, E. R., 82–0138

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Dalton, R., 82–0249, 82–0250
Dansie, D. R., 82–0048
Dato, A., 82–0087
David, T. E., 82–0111
Davidson, F., 82–0165
Davies, J. M., 82–0065
Davis, D. L., 82–0004, 82–0015
Davis, N. S., 82–0026
Dawber, T. R., 82–0113
Dawley, H. H., Jr., 82–0194
Day, R. G., 82–0193
Decker, J. R., 82–0083
De Jong, D. W., 82–0010
DeLucia, A. J., 82–0092
Denson, R., 82–0195
DePratti, V. J., 82–0115
Devitt, G., 82–0052
Diallo, A. D., 82–0184
Diamond, L., 82–0093
Dielman, T. E., 82–0183
Dill, C. A., 82–0196
Dimsdale, J. E., 82–0114
Dixon, B., 82–0289
Djordjevic, M., 82–0016, 82–0017
Dmitriev, M. T., 82–0037
Dobler-Mikola, A., 82–0212
Dobre, V., 82–0066
Doll, R., 82–0030, 82–0064
Dorough, W., 82–0004
Dorow, P., 82–0109
Douglas, D. R., 82–0025
Driscoll, P., 82–0038
Duncan, A., 82–0115
Duryea, E. J., 82–0179

Eastman, A., 82–0067
Ehringhaus, J.C.B., Jr., 82–0280
Eicher, T., 82–0230
Elliott, E. A., 82–0077
Ellis, D. A., 82–0094
Emanuel, D. A., 82–0097
Ennis, D. M., 82–0025
Epstein, L. H., 82–0128, 82–0181
Erikssen, J., 82–0116
Evans, D., 82–0213
Evans, R. I., 82–0196
Everette, G., 82–0015

Federal Trade Commission., 82–0251
Feely, J., 82–0039
Felix, R., 82–0109
Feyerabend, C., 82–0052
Fichera, A. T., 82–0214
Filipy, R. E., 82–0083
Finger, W. R., 82–0252, 82–0253, 82–0281
Finkel, C., 82–0194
†Firmenich, S. A., 82–0227, 82–0228
Fisher, D. A., 82–0166
Fisher, E. B., Jr., 82–0216
Flagle, J., 82–0197
Fleenor, C. P., 82–0296
Fontana, R. S., 82–0086
Foord, N., 82–0107
Foraker, A. G., 82–0060
Foreign Agriculture., 82–0254
Forfang, K., 82–0116
Forsberg, O., 82–0156
Foster, F. H., 82–0110
Franchebois, P., 82–0043
Frasinel, C., 82–0044, 82–0045
Freestone, S., 82–0117
Friedman, G. D., 82–0112
Friedman, L., 82–0040
Fu, M. C., 82–0018
Fuxe, K., 82–0032

Gaafar, H. A., 82–0068
Gairola, C., 82–0019
Galarraga, J., 82–0045
Gallup, G., 82–0167, 82–0168
Garcia-Palmieri, M. R., 82–0133
Garg, O. P., 82–0176
Geiszler, W. A., Jr., 82–0233
George, R. R., 82–0115
German, G. A., 82–0222
Gibinski, K., 82–0061, 82–0169
Gilman, M. J., 82–0095
Glasgow, R. E., 82–0215
Gloag, D., 82–0290
Gmur, M., 82–0212
Godard, P., 82–0096
Goeringer, G. C., 82–0044
Goldberg, D. M., 82–0170
Goldberg, S. R., 82–0170
Goldstein, A., 82–0050
Good, B. W., 82–0025
Gori, G. B., 82–0291
Graham, K. J., 82–0127
Green, L., 82–0216
Greenblatt, D. J., 82–0046
Greene, J. Y., 82–0189
Greenhalgh, R. M., 82–0118
Greenland, S., 82–0069
Griffiths, A.R.B., 82–0198
Griffiths, R. R., 82–0172
Gruchow, H. W., 82–0097
Guillory, F., 82–0292
Gurnani, M., 82–0176
Guthrie, T. J., 82–0196

Hackett, T. P., 82–0114
Hadergjonaj, Sh., 82–0100
Hakkinen, P. J., 82–0085
Hall, J., 82–0148, 82–0149
Hall, J. B., 82–0224
Halonen, M., 82–0151
Hamosh, P., 82–0090
Hansen, J. F., 82–0119
Harburg, E., 82–0171
Harden, B., 82–0282
Harris, R.W.C., 82–0064
Harrison, K. L., 82–0139
Hartz, A. J., 82–0120
Hasegawa, H., 82–0099
Hashimoto, Y., 82–0023
Hatch, F. W., 82–0025
Havlik, R., 82–0133
Haxhiu, M. A., 82–0100
Hay, D. R., 82–0110
Hayward, J. N., 82–0049
Hedin, C. A., 82–0156
Heffner, J. E., 82–0098
Henderson, A. H., 82–0196
Henningfield, J. E., 82–0172
Henry, C. J., 82–0048
Herget, J. B., 82–0255
Herzfeld, L., 82–0020
Heymach, G. J., III, 82–0059
Higginbotham, J. D., 82–0237
Hill, D., 82–0256
Hill, D. G., 82–0127
Hill, P. C., 82–0196
Ho-Yen, D. O., 82–0186
Hoffmann, D., 82–0209
Hoffmann, R. G., 82–0097, 82–0120
Holland, F., 82–0089
Hollenweger, V., 82–0070, 82–0082
Hopkins, P. N., 82–0121
Horsewell, H. G., 82–0231
Horvath, W. J., 82–0183
Houben, H., 82–0122
Hsu, F. S., 82–0025
Huba, G. J., 82–0173
Huber, G. L., 82–0125
Huebner, A., 82–0257
Huel, G., 82–0140
Hughes, C. A., 82–0138
Hume, A. S., 82–0026
Humphries, B., 82–0283
Hurwitz, I., 82–0218
Hutter, A. M., Jr., 82–0114

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Ibrahim, M. A., 82-0140
Infante, F., 82-0070, 82-0082
†International Flavors and Fragrances Inc., 82-0224, 82-0225, 82-0234
Irie, T., 82-0099
Isaac, R. S., 82-0076

Jacobsen, M., 82-0071
Jaffe, J. H., 82-0040
Jakins, P. R., 82-0103
Jarvis, M. J., 82-0052
Jenkins, J. M., 82-0232
Jerin, M. J., 82-0189
Jervell, J., 82-0116
Jewell, N. P., 82-0050
Johnson, K., 82-0136
Johnston, L. D., 82-0174
Jones, A., 82-0131
Jonquet, O., 82-0096
Joseph, J. D., 82-0284
Jost, S., 82-0198
Joyce, H., 82-0089

Kagedal, B., 82-0041
Kaliman, J., 82-0132
Kamishima, K., 82-0099
Kandaswami, C., 82-0072
Kannel, W. B., 82-0123
Kanzler, M., 82-0040
Kapsambelis, V., 82-0158
Kapuscinska, B., 82-0124
Karki, N. T., 82-0141
Kasperbauer, M. J., 82-0009, 82-0012
Kassman, A. J., 82-0233
Kauffmann, F., 82-0157
Kauppila, A., 82-0141
Kawakami, Y., 82-0099
Keen, K., 82-0152
Kehrer, J. P., 82-0085
Kelley, D., 82-0059
Kellner, H., 82-0175
Kennedy, B. J., 82-0136
Kennedy, T. P., 82-0095
Kephalas, T. A., 82-0158
Kerr, A. R., 82-0127
Kestenbaum, H., 82-0062, 82-0293
Kiburis, J., 82-0158
Kiger, H. C., 82-0258
Kim, S., 82-0199
Kinney, J. A., 82-0259
Kirk, I., 82-0177
Kishi, F., 82-0099
Kissinger, D. G., 82-0142
Kiwala, J., 82-0225, 82-0234
Klenke, B., 82-0104
Klisinski, J., 82-0061
Koivisto, M., 82-0141
Koplin, A. N., 82-0285
Korsgaard, R., 82-0073
Koskela, K., 82-0200
Kostenbauder, H. B., 82-0013
Kouri, R. E., 82-0048
Kovacs, S., 82-0108
Kramer, M. S., 82-0284
Krasniqi, A., 82-0100
Krstic, S. K., 82-0035
Kubler, W., 82-0126
Kujala, P., 82-0101
Kurtz, S.M.S., 82-0216
Kuwata, K., 82-0021
Kuzma, J. W., 82-0142

Laing, S. P., 82-0118
Lall, K. B., 82-0176
Lane, D. S., 82-0213
Langone, J. J., 82-0042
Laszlo, C. A., 82-0190
Laviolette, M., 82-0102
Lawson, C., 82-0134
Lazaratou, H., 82-0158
Lebowitz, M. D., 82-0151
Lee, P. N., 82-0074
Legg, P. D., 82-0002, 82-0005
Lelek, E., 82-0061
Lelkowa, E., 82-0169
Lemoine, J. M., 82-0075
Lenz, K., 82-0034
Leung, M., 82-0148, 82-0149
Lewis, D. J., 82-0103
Lichtenstein, E., 82-0210
Lillich, T. T., 82-0018
Liu, H. M., 82-0049
Lockard, J. M., 82-0053
Loizeau, E., 82-0070, 82-0082
Louis, A. M., 82-0260
Lowe, J. B., 82-0127
Lowe, M. R., 82-0216
Lubawy, W. C., 82-0076
Lynch, P., 82-0177

Maccoby, N., 82-0200
Macdonald, I. L., 82-0111
MacKenzie, K. M., 82-0143
Mackinnon, M. A., 82-0159
MacLean, A., 82-0177
Magnus, P., 82-0166
Maher, F. A., 82-0125
Maisto, S. A., 82-0178
Mamlin, J. J., 82-0189
Mandura, I., 82-0100
Mann, C. K., 82-0261
Manthey, J., 82-0126
Marr, B., 82-0131
Martensson, J., 82-0041
Martin, G. P., 82-0217
Martin, J. E., 82-0026
Marx, J. J., Jr., 82-0097
Mascres, C., 82-0043
Matanoski, G. M., 82-0077
Matsumoto, T., 82-0022
Matsuzaki, H., 82-0023
Maxwell, J. C., Jr., 82-0262, 82-0263
Maxwell, S. E., 82-0196
Mayrhofer, K., 82-0264
McAlister, A., 82-0200
McDonald, J. A., 82-0059
McGovern, J. P., 82-0106
McGuire, W. J., 82-0201
McKnight, D., 82-0181
McLeish, A. R., 82-0159
Meloni, M., 82-0088
Menkes, H. A., 82-0095
Meyer, M. B., 82-0147
Meyer, W., 82-0104
Michel, F. B., 82-0096
Micic, D., 82-0035
Miller, E. C., 82-0078
Miller, G. H., 82-0202
Miller, J. A., 82-0078
Miller, W. E., 82-0086
Milliman, E. M., 82-0083
Milmoe, S. R., 82-0265
Milsum, J. H., 82-0190
Milton, G. W., 82-0079
Mironenko, N. Ye., 82-0024
Mokhnachev, I. G., 82-0024
Mookherjee, B. D., 82-0225, 82-0234
Moolgavkar, S. H., 82-0065
Moore, L. D., 82-0008
Morgenstern, W., 82-0126
Morosco, G. J., 82-0044
Moschovakis, A., 82-0158
Mossman, B. T., 82-0067
Muhm, J. R., 82-0086
Muller, F., 82-0230
Murao, M., 82-0099
Murray, N., 82-0152
Mysliwiec, M., 82-0054

Neittaanmaki, L., 82-0204
Neuber, D., 82-0266, 82-0267
Newcombe, D. S., 82-0102

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Newcombe, R. G., 82–0137
Newman, I. M., 82–0179
Nightingale, T. E., 82–0044, 82–0045
Norris, R. M., 82–0127
Nussel, E., 82–0126
Nutall, R., 82–0218

Oalmann, M. C., 82–0133
O'Brien, P. J., 82–0072
Ochs, H. R., 82–0046
Ockene, I. S., 82–0218
Ockene, J. K., 82–0218
Office of Population Censuses and Surveys., 82–0180
Ogle, J., 82–0219
Ohloff, G., 82–0228
O'Malley, P. M., 82–0174
O'Neill, H. K., 82–0215
Opherk, D., 82–0126
Orme, M.L'E., 82–0036
Orzechowska, R., 82–0061, 82–0169
Ossip, D. J., 82–0128, 82–0181
Otten, H., 82–0046
Overton, J., 82–0268
Owens, M. V., 82–0220

Pachinger, O., 82–0132
Pallonen, U., 82–0200
Pandeya, R. S., 82–0006, 82–0028
Parrish, M. E., 82–0025
Patrick, J. M., 82–0105
Paxton, R., 82–0221
Pedder, C. T., 82–0232
Pelkonen, O., 82–0141
Pennell, M. D., 82–0148, 82–0149
Petitti, D. B., 82–0112
Peto, R., 82–0080
Phelps, D. W., 82–0083
Phelpstead, J.W.P., 82–0231
Philip, L., 82–0182
†Philip Morris Inc., 82–0233
Pinney, J. M., 82–0203
Pleho, A., 82–0047
Pohl, H.-G., 82–0269
Poole, A. D., 82–0222
Prevost, R. J., 82–0106
Pride, N. B., 82–0089
Pritchard, J. N., 82–0107
Probst, P., 82–0132
Prue, D. M., 82–0026
Pryor, W. A., 82–0027
Pugh, C., 82–0270, 82–0271
Puska, P., 82–0200, 82–0204

Qasim, M., 82–0049

†R. J. Reynolds Tobacco Co., 82–0229
Rabkin, S. W., 82–0129
Radius, S. M., 82–0183
Rahe, R. H., 82–0163
Raines, B. E., 82–0196
Rajendran, N., 82–0276
Ramsay, L. E., 82–0117
Rana, M. W., 82–0055, 82–0056, 82–0057, 82–0134
Rasmussen, R. E., 82–0048
Raymer, W. J., 82–0106
Raymond, L., 82–0070, 82–0082
Reaves, T. A., Jr., 82–0049
Reed, C. E., 82–0205
Reid, P., 82–0177
Reilley, C. N., 82–0025
Reinberg, A., 82–0106
Rimm, A. A., 82–0097
Rizio, D., 82–0266, 82–0267, 82–0273
†RJR Archer, Inc., 82–0240
Robertson, N.G.D., 82–0232
Robinson, A. G., 82–0139
Roche, A.H.G., 82–0127
Rockcliffe, W., 82–0177
Rogers, F., 82–0198
Romano, C., 82–0050
Rondahl, L., 82–0051
Ronquist, G., 82–0156
Rosa, N., 82–0028
Rosenstock, I. M., 82–0183
Roudaut, M., 82–0184
Rowell, P. P., 82–0144
Rozelle, R. M., 82–0196
Rush, D., 82–0145
Russell, M.A.H., 82–0052
Rys, M., 82–0061, 82–0169

Sabharwal, P. S., 82–0053
Sabo, J., 82–0093
Sahn, S. A., 82–0098
Samaras, J. T., 82–0220
Samfield, M., 82–0274
Sanders, E. B., 82–0029
Sanderson, D. R., 82–0086
Sanna-Randaccio, F., 82–0088
Sanson-Fisher, R. W., 82–0222
Sardi, F., 82–0108
Schafer, L., 82–0215
Schmidt, D., 82–0184
Schmitt, F. L., 82–0224, 82–0225, 82–0234
Schreiber, W. L., 82–0225
Scott, S., 82–0221
Secor, H. V., 82–0029
Seeman, J. I., 82–0029
Seethamma, M., 82–0276
Seltzer, C. C., 82–0112, 82–0130
Seshagiri Rao, P., 82–0272
Sharpe, D. N., 82–0110
Shaw, H. M., 82–0079
Shelton, J. H., 82–0025
Shida, A., 82–0099
Shien, T., 82–0275
Shor, M. B., 82–0185
Shor, R. E., 82–0185
Shubik, P., 82–0080
Sibert, J. R., 82–0137
Siceloff, B., 82–0235
Sidorski, S., 82–0054
Simons, J., 82–0131
Simons, L., 82–0131
Sims, J. L., 82–0007
Singh, A. N., 82–0276
Singhi, S., 82–0176
Sinzinger, H., 82–0132
Slavin, R. G., 82–0091
Sloneker, S. D., 82–0013
Smallwood, R. A., 82–0159
Smith, D., 82–0198
Smolensky, M. H., 82–0106
Snider, H. L., 82–0063
Snook, M. E., 82–0014
Song, B. H., 82–0015
Sorbo, B., 82–0041
Sorlie, P. D., 82–0133
Spealman, R. D., 82–0170
Spence, V., 82–0186
Spitz, J. B., 82–0206
Sprecker, M. A., 82–0224
Sram, R. J., 82–0146
Stefanovic-Denic, K., 82–0035
Stenback, F., 82–0080
Stencel, S., 82–0207
Stepney, R., 82–0033
Stevens, R. G., 82–0065
Stevenson, I. H., 82–0039
Sticht, J. P., 82–0277
Stock, S. L., 82–0187
Stoeppler, M., 82–0126
Stork, G., 82–0225
Strauss, H. D., 82–0111
Strbac, M., 82–0035
Streja, D. A., 82–0129
Stretch, S., 82–0195
Stunkard, A. J., 82–0040
Sullivan, J. M., 82–0055, 82–0056, 82–0057, 82–0134

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Sullivan, S. L., 82-0058, 82-0294
Sutton, G. C., 82-0187
Swanson, G. T., 82-0236
Swidersky, P., 82-0045
Sydnor, K. L., 82-0081
Sylvester, J. T., 82-0095

Tabak Journal International., 82-0295
†Talres Development (N.A.) N.V., 82-0237
Taylor, G. W., 82-0118
Taylor, W. F., 82-0086
†Teledyne Industries, Inc., 82-0236
Terrin, M., 82-0147
Thien, Th., 82-0122
Thomas, M., 82-0135
Thompson, J. W., 82-0033
Thornley, P. E., 82-0094
Tibbling, L., 82-0041
Tiendrebeogo, H., 82-0184
Toccaceli, F., 82-0087
Tomita, H., 82-0022
Tones, B. K., 82-0208
Tornquist, E., 82-0278
Traystman, R. J., 82-0095
Trenary, J., 82-0236
Trevathan, L. E., 82-0008
Tuimala, R., 82-0141
Tuomilehto, J., 82-0204

Uebori, M., 82-0021
Uhlenhopp, M. A., 82-0086

van der Loo, H. E., 82-0237
Van 'T Laar, A., 82-0122
Van Tilburg, J., 82-0238
Varga, L. E., 82-0190
Vaughn, T. H., 82-0012
Verebey, K., 82-0040
Vertes, L., 82-0188
Vessey, M. P., 82-0064
Vickers, R. R., Jr., 82-0163
Villiger, B., 82-0059
Vinals, J. F., 82-0225, 82-0234
Vock, M. H., 82-0225
Vogelzang, N. J., 82-0136
Vogts, H., 82-0104
Voirol, M., 82-0070, 82-0082
von Wichert, P., 82-0104
Vunakis, H. V., 82-0042

Waite, P.M.E., 82-0217
Wald, N., 82-0030
Walker, M., 82-0094
Walsh, M., 82-0107
Ward, H. W., 82-0163
Warner, K. E., 82-0286
Waters, M., 82-0177
Watson, D. C., 82-0025
Waugh, R. L., 82-0152
Wehner, A. P., 82-0083
Weinberger, M., 82-0189
Weintraut, A., 82-0126
Weis, W. L., 82-0296
Weiss, von Th., 82-0109
Weiss, W., 82-0084
Weissman, G., 82-0279
Wesch, H., 82-0126
Wheeler, J., 82-0197
Whidby, J. F., 82-0029
Whish, P., 82-0131
White, F. H., 82-0006
White, J. L., 82-0229
Whitlock, R.M.L., 82-0127
Widhalm, K., 82-0132
Wiener, C., 82-0237
Wightman, A. J., 82-0094
Wilcock, A. R., 82-0190
Wilke, A., 82-0104
Wilkinson, R. E., 82-0009
Williams, D. C., 82-0185
Williams, D. R., 82-0159
Williams, P. T., 82-0064
Williams, R. R., 82-0121
Williamson, R. E., 82-0031
Wilson, R. A., 82-0225, 82-0234
Wingard, J. A., 82-0173
Wistuba, J., 82-0061
Witschi, H. P., 82-0085
Woodlief, W. G., 82-0010
Woodward, D., 82-0177
Woolner, L. B., 82-0086
Wynder, E. L., 82-0209

Yamaguchi, T., 82-0239
Yamazaki, Y., 82-0021
Yeatts, R. E., 82-0240
Yeomans, N. D., 82-0159
Yeung, D. L., 82-0148, 82-0149
Yocum, J. O., 82-0011
Yoshida, D., 82-0022
Yoshikawa, T., 82-0099
Young, C. T., 82-0009

Zenk, K. E., 82-0150

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

SUBJECT INDEX

ACCIDENTS
upholstered furniture fires, cigarette modification proposals, 82–0288

ADOLESCENTS
actual and intended use of tobacco, 82–0173
deviant behavior, predictive value of smoking habits, 82–0165
Gallup poll smoking survey, 1981, 82–0168
health beliefs vs. smoking habits, 82–0183
Nebraska students' smoking habits, 82–0179
smoking prevention programs, 82–0197
Tasmanian high school students, smoking incidence, 82–0177

ADRENAL HORMONES
epinephrine-secreting cells of rat adrenal medulla, nicotine effects, 82–0055
nicotine arterial effects in rats, 82–0134

ADVERTISING
actions by developed and developing nations, 82–0281
antismoking messages financed by tobacco advertisement taxes, British proposal, 82–0198
Australian children's opinions, 82–0166
brand introduction difficulties, West German experiences, 82–0264
Brown and Williamson's new ultra-low-tar Barclay, 82–0260
demystified, Australian school program, 82–0162
Federal Trade Commission 1979 report on cigarettes, 82–0251
governmental restrictions, 82–0280
growth and expansion of the American tobacco industry, 82–0268
health warnings in West Germany, 82–0269
Lorillard's emphasis on sophisticated, stylized imagery, 82–0266
120 mm cigarettes, aimed at women, 82–0267
smokeless tobacco, male youth market, 82–0153
Third World countries, by transnational exporters, 82–0257
tobacco conglomerates, global promotion increases, 82–0248

AGE FACTORS
consumption patterns, United Kingdom 1972-1980, 82–0180
drug abuse as coping strategy, 82–0164
elderly persons, smoking incidence, 82–0188

AGRICULTURAL PRACTICES
alternative crops for tobacco farmers, prospects and problems, 82–0241
amino acid content in leaves, light and temperature effects, 82–0009
breeding of low-tar varieties, 82–0001
calcium fertilization effects on air pollution tolerance and leaf content, 82–0008
dark tobacco constituents, curing, and culturing effects, 82–0015
Delgold tobacco variety, commercial advantages, 82–0006
extractable protein yield from leaf varieties, harvesting effects, 82–0010
fertilizer and genotype influences on leaf and smoke constituents, 82–0007
Fifth Workshop Conference Proceedings, papers, 82–0058
genetic breeding, improved quality and reduced health risks, 82–0002
genetic breeding to alter tobacco chemical constituents, 82–0005
increased mechanization in harvesting, 82–0247
Korean tobacco industry, growth and outlook, 82–0276
less hazardous cigarettes relationship, 82–0291
maleic hydrazide residues in Kentucky tobaccos, 82–0004
maleic hydrazide, use restrictions, 82–0003
mechanization and bulk-barn curing, increased use, 82–0261
mechanization and chemicals for larger farms, 82–0250
mechanization, decreasing labor requirements, 82–0243
Tobacco and Health Research Institute programs, University of Kentucky, 82–0294
tobacco crops for protein in North Carolina, 82–0235
tobacco growing practices in Pennsylvania, 82–0011

AIR POLLUTION
calcium fertilization effects on tolerance of tobacco plants, 82–0008
indoor source from smoking, 82–0290
phenol content determination by liquid chromatography, 82–0021

ALCOHOL CONSUMPTION
behavioral coping strategy, 82–0164
handedness, effect on smoking and drinking habits, 82–0171

SUBJECT INDEX

impaired driving factor with smoking, 82–0190
liver neoplasms and cirrhosis, as factor with smoking, 82–0070
occupational stress responses, 82–0163
oral mucosa effects in smoking and nonsmoking cirrhotic patients, 82–0043
pancreatic carcinoma, tobacco, alcohol, and diet relationship, 82–0082
pregnancy effects, maternal smoking interrelationships, 82–0142

ALCOHOLS
derivatives, smoking tobacco organoleptic enhancement, 82–0225
plant growth and air curing effects on tobacco midveins, 82–0012

ALGERIA
tobacco goods production and consumption increases, 82–0244

ALIPHATIC HYDROCARBONS
tobacco expansion process with hot inert solid particles, 82–0229

ALKALOID CONTENT
dark tobacco concentrations, 82–0015
infrared reflectance spectroscopy for tobacco constituent analysis, 82–0028
stalk position and cultivar differences, 82–0031

ALKALOIDS, TOBACCO
cue receptors in male albino rats, 82–0050

ALLERGY
atopic predisposition for obstructive airways disease in male smokers, 82–0089

AMINO ACIDS
tobacco crops for protein in North Carolina, 82–0235

ANABASINE
nicotine cue in male albino rats, 82–0050

ANGINA PECTORIS
risk factor in diagnosis of coronary artery disease, 82–0119

ANTIBODIES
Farmer's Lung Disease in smokers vs. nonsmokers, 82–0097

ANTIGEN—ANTIBODY REACTIONS
Farmer's Lung Disease in smokers vs. nonsmokers, 82–0097

ANTISMOKING CAMPAIGNS
actions by developed and developing nations, 82–0281
adolescent and adult program approaches, 82–0209
advertising demystified, Australian school program, 82–0162
educational, preventive, radical, and self-empowerment methods of health education, 82–0208
films, junior high school program, 82–0196
financed by tobacco advertisement taxes, British proposal, 82–0198
Gallup poll smoking survey, increasing public support for tobacco restrictions, 82–0167
the Great American Smokeout, 82–0194
legal and historical aspects, 82–0207
legislated program in New Jersey, 82–0285
North Karelia Project for cardiovascular disease control in Finland, 82–0204
Ombudsman primary prevention program, effectiveness, 82–0199
public communications seven-step strategy, 82–0201
public smoking and advertising restrictions, 82–0280
school-based program, smoking as drug addiction, 82–0195
school education curriculum, New York State, 82–0192
school program proposals, 82–0205
State cigarette excise taxes and laws restricting public smoking, 82–0286
televised cessation program in Finland, community organization, 82–0200

ANTISMOKING MATERIALS
films, junior high school program, 82–0196
school education curriculum, New York State, 82–0192

ANTITRYPSIN DEFICIENCY
alpha-l-antitrypsis deficiency and chronic obstructive pulmonary disease, smoking relation, 82–0100

ANXIETY
cessation or reduction of smoking relationship, 82–0210

AORTA
atherosclerotic lesions and smoking, 82–0133
bypass operations, smoking as risk factor, 82–0111

AROMATIC HYDROCARBON CONTENT
moisture content relationship in cigarette smoke, 82–0017

AROMATIC HYDROCARBONS
benz(*a*)anthracene, carcinogenic skin treatment responses of mice, 82–0080
benzo(*a*)pyrene, formation and removal from hamster tracheal epithelial cells, 82–0067

ARTERIAL DISEASES
aortic atherosclerosis and smoking, 82–0133

coronary bypass operations, smoking as risk factor, 82–0111
Framingham study on risk factors in atherosclerosis, 82–0113
myocardial infarction, coronary occlusion as risk factor, 82–0120
Raynaud's phenomenon, promotion by smoking and chemotherapy, 82–0136
reconstructive surgery, smoking-related risk, 82–0118
risk factor of smoking, 82–0135
risk factors used in diagnosis, 82–0119

ARTERIES
pulmonary vascular responses to cigarette smoke in pig lung, 82–0095

ARTERIOSCLEROSIS
aortic lesions and smoking, 82–0133
coronary risk factors, smoking effects, 82–0121
Framingham study on risk factors, 82–0113
high-density lipoprotein cholesterol, smoking and weight loss effects, 82–0129
platelet sensitivity to antiaggregatory prostaglandins, 82–0132
quantification of smokers' characteristics, 82–0063
reconstructive arterial surgery, smoking-related risk, 82–0118
risk factors, smoking, 82–0114, 82–0135

ARYL HYDROCARBON HYDROXYLASE
asbestos in reduction of enzyme activity, 82–0072
induction in maternal and neonatal lymphocytes, 82–0141

ASBESTOS
benzo(*a*)pyrene effect in lung metabolism, 82–0072

ASTHMA
circadian assessment of respiratory function, 82–0087
passive smoking effects on pulmonary function, 82–0091
temporal pattern of respiratory distress, smoking effects, 82–0106

ATTITUDES
advertising demystified, Australian school program effects, 82–0162
Australian children's opinion of cigarette advertising, 82–0166
educational, preventive, radical, and self-empowerment methods of health education, 82–0208
Gallup poll smoking survey, 1981, 82–0167, 82–0168
health hazard perception by smokers and ex-smokers, 82–0189
impaired drivers, lifestyle hazards, 82–0190
managers' opinions toward smoking and nonsmoking employees, 82–0296
peer values and role models in adolescent smoking, 82–0173
physicians who smoke as inappropriate role models, 82–0175
schoolchildren's health beliefs vs. smoking habits, 82–0183
self-control treatment of addictive behavior, 82–0178
smoking as drug addiction, effect on smoking onset, 82–0195
university students, high school smoking education programs survey, 82–0205
university students reaction to tobacco smoke pollution, 82–0185

AUSTRALIA
advertising demystified, school program results, 82–0162
cigarette advertising, children's opinions, 82–0166
coronary risk factors, smoking among Aboriginal and non-Aboriginal subjects, 82–0131
Tasmanian high school students, smoking incidence, 82–0177

AVERSIVE THERAPY
rapid-smoking effectiveness, 82–0222
self-control and covert sensitization methods, symbolic vs. actual stimuli, 82–0216

BEHAVIOR
coffee drinking and smoker modeling vs. smoking habit, 82–0181
deviancy in adolescents, predictive value of smoking habits, 82–0165
educational, preventive, radical, and self-empowerment methods of health education, 82–0208
Gallup poll smoking survey, 1981, 82–0167, 82–0168
myocardial infarction patients, smoking cessation rates, 82–0127
physicians who smoke as inappropriate role models, 82–0175
psychosocial assets to handle stress without smoking, 82–0218
Tasmanian high school students, smoking incidence, 82–0177

BEHAVIOR, ANIMAL
cannabis disruption of sleep and nest-building behaviors in mice, 82–0158
nicotine inhibition of fighting patterns in rats, 82–0038
reinforcement in squirrel monkeys by nicotine and visual stimulus, 82–0170
vasopressin release in the cat, nicotine effect, 82–0049

BEHAVIOR, HUMAN
d-amphetamine-induced stimulation of tobacco consumption, 82–0172
advertising demystified, Australian school program effects, 82–0162

SUBJECT INDEX

elderly persons, smoking as negative role model image, 82–0188
frequency of maternal smoking vs. health knowledge, 82–0138
handedness, effect on smoking and drinking habits, 82–0171
health hazard perception in cessation of smoking, 82–0189
impaired drivers, lifestyle hazards including smoking, 82–0190
India school boys smoking habits, 82–0176
motivation modification, indications for success in cessation of smoking, 82–0169
occupational stress responses, 82–0163
peer values and role models in adolescent smoking, 82–0173
physician's recognition of patients' smoking habits, 82–0193
schoolchildren's health beliefs vs. smoking habits, 82–0183
self-control response to dependency, 82–0178
smoking and drug use as coping strategy, 82–0164
smoking cessation workshop followup survey, 82–0213
smoking prevention programs for adolescents, 82–0197

BENZ(*A*)ANTHRACENES
skin treatment of mice, carcinogenic responses, 82–0080

BENZO(*A*)PYRENE
asbestos effect in lung metabolism, 82–0072
formation and removal from hamster tracheal epithelial cells, 82–0067
infertility in exposed mice, 82–0143
metabolism in isolated rabbit lung, 82–0076
pulmonary biotransformations, inhibitor characteristics, 82–0013
tumorigenicity of tobacco smoke fractions, interrelationships, 82–0081

BIOASSAY
comparison of three tests to determine tobacco smoke condensate mutagenesis, 82–0019
smoke toxicity determination method, 82–0018
synthesis and bioactivity of four synthetic nicotine analogs, 82–0051

BIRTH WEIGHT
maternal smoking effects, 82–0137, 82–0147, 82–0148, 82–0150
maternal smoking effects on infant pO_2, pCO_2 blood levels, 82–0139

BLADDER NEOPLASMS
epidemiological studies, smoking as risk factor, 82–0077
mortality attributable to smoking, 82–0065
statistical analysis of incidence, 82–0069

BLOOD
clotting rate, smoking effects, 82–0054
nicotine and cotinine levels in body fluids, a radioimmunoassay technique, 82–0042
plasma propranolol, lower levels in smokers, 82–0039

BLOOD CHEMICAL ANALYSIS
dose markers in beagle dogs exposed to smoke, 82–0045
nicotine levels in snuff users vs. smokers, 82–0052
oral contraceptive steroids, smoking effects, 82–0036
plasma propranolol, lower levels in smokers, 82–0039
prolonged smoke exposure effects in primates, 82–0092

BLOOD CHOLESTEROL
high-density lipoprotein cholesterol, smoking and weight loss effects, 82–0129
myocardial infarction risk factor, 82–0120

BLOOD CIRCULATION
pulmonary vascular responses to cigarette smoke in pig lung, 82–0095

BLOOD LIPIDS
coronary disease risk reduction by cessation of smoking, 82–0124
high-density lipoprotein cholesterol, smoking and weight loss effects, 82–0129

BLOOD PRESSURE
β-adrenoceptor blocking drug treatment, smoking effects, 82–0122
coffee and smoking effects on pulse rate and hypertension, 82–0117

BLOOD VESSELS
coronary bypass operations, smoking as risk factor, 82–0111
pulmonary responses to cigarette smoke in pig lung, 82–0095
Raynaud's phenomenon, promotion by smoking and chemotherapy, 82–0136
response to varied doses of nicotine, 82–0128

BODY HEIGHT
elite class of Calcutta men, smoking relationship, 82–0182

BODY WEIGHT
base-forming diet for cessation of smoking, 82–0219
elite class of Calcutta men, smoking relationship, 82–0182
high-density lipoprotein cholesterol, smoking and weight loss effects, 82–0129

SUBJECT INDEX

BRAIN
- morphine, methadone, and pethidine inhibition of nicotine-induced emesis in cats, 82–0035
- nicotine inhibition of fighting behavior in rats, 82–0038
- rat adrenal medulla, nicotine effects, 82–0055

BREAST FEEDING
- duration by mothers who smoke, 82–0149

BRONCHIAL DISEASES
- bronchiectasis, smoking as factor, 82–0094
- lung function abnormalities in sons of pulmonary patients, 82–0099
- respiratory distress in asthmatics, bronchitics, emphysematics, smoking effects, 82–0106

BRONCHIAL EPITHELIUM
- chronic phlegm secretion in peptic ulcer individuals, 82–0157

BRONCHIAL MUCOSA
- chronic phlegm secretion in peptic ulcer individuals, 82–0157
- fibronectin concentrations and secretion, smoking effects, 82–0059

BRONCHIAL NEOPLASMS
- aversion to smoking as lung neoplasm symptom, 82–0066
- early detection by level of cigarette consumption, 82–0075
- Mayo Clinic screening of squamous and small cell carcinoma among male smokers, 82–0086

BRONCHITIS
- young males, smoking effects, 82–0101

BRONCHOPULMONARY DISEASE
- lymphocyte subpopulations in smokers vs. nonsmokers, 82–0096

CADMIUM
- cardiovascular risk factor from cigarette smoking, 82–0126
- maternal and newborn hair content, 82–0140

CANADA
- smoking survey, 1965-1979, 82–0161

CANNABIS
- marihuana smoke hazards compared to tobacco smoke, 82–0155
- national trends in drug abuse, high school seniors 1975-1979, 82–0174

CANNABIS DERIVATIVES
- mice sleeping time and nest building, cannabis and tobacco analogs effect, 82–0158

CARBOHYDRATE CONTENT
- infrared reflectance spectroscopy for tobacco constituent analysis, 82–0028
- stalk position and cultivar differences, 82–0031

CARBON DIOXIDE CONTENT
- maternal smoking effects on infant pCO_2 blood levels and birth weight, 82–0139

CARBON MONOXIDE
- levels in very low-yield cigarette smokers, 82–0040
- maternal smoking effects on infant pCO_2 blood levels and birth weight, 82–0139

CARBON MONOXIDE CONTENT
- chlorella green algae filter for yield reduction, 82–0239
- cigarette manufacturing techniques obtain lower yields, 82–0226
- decreasing yields in the United Kingdom, 82–0030
- filter ventilation for reduced yield, 82–0238
- indicator for nicotine exposure, 82–0033
- indoor air pollution from smoking, 82–0290
- noxious substance index, recommended values, 82–0020

CARBOXYHEMOGLOBIN LEVELS
- beagle dogs exposed to smoke, 82–0045
- indicator for nicotine exposure, 82–0033
- indoor air pollution effects, 82–0290
- oxygen transport reduction in polycythemias, 82–0154
- rat cardiac muscle responses, 82–0125
- reconstructive arterial surgery, smoking-related risk, 82–0118

CARCINOGENESIS
- benz(*a*)anthracene, skin treatment responses of mice, 82–0080
- benzo(*a*)pyrene and asbestos in lung metabolism, 82–0072
- chemical induction mechanisms, 82–0078
- cytogenetic analysis in diagnosis for malignancy, 82–0073
- dimethylnitrosamine, 3-methylcholanthrene, and urethan effects in mice, promotion by butylated hydroxytoluene, 82–0085
- Fifth Workshop Conference Proceedings, papers, 82–0058
- metastases facilitated by smoking, 82–0079
- tumorigenicity of smoke condensates, 82–0081
- tumors in rats exposed to tar and nicotine, 82–0083

CARCINOGENS
- benz(*a*)anthracene, skin treatment responses of mice, 82–0080
- benzo(*a*)pyrene removal from hamster tracheal epithelial cells, 82–0067
- chemical induction mechanisms, 82–0078

dimethylnitrosamine, 3-methylcholanthrene, and urethan effects in mice, promotion by butylated hydroxytoluene, 82–0085

CARCINOMA
carcinogenesis, chemical induction mechanisms, 82–0078
metastases facilitated by smoking, 82–0079

CARCINOMA, BRONCHOGENIC
early detection by level of cigarette consumption, 82–0075
incidence among smokers vs. nonsmokers, 82–0060
Mayo Clinic screening of squamous and small cell carcinoma among male smokers, 82–0086

CARDIOVASCULAR DISEASES
aortic atherosclerosis and smoking, 82–0133
causal relationship with smoking questioned, 82–0130
coronary mortality decline in New Zealand, reduced smoking as factor, 82–0110
coronary risk factors, smoking effects, 82–0121, 82–0131
Fifth Workshop Conference Proceedings, papers, 82–0058
mortality, cigarette smokers vs. quitters, 82–0112
myocardial infarction, coronary occlusion and cholesterol as risk factors, 82–0120
North Karelia Project for risk factor reduction, 82–0204
platelet sensitivity to antiaggregatory prostaglandins, 82–0132
reconstructive arterial surgery, smoking-related risk, 82–0118
risk factors, smoking, 82–0114, 82–0116, 82–0123, 82–0135
trace metals level as risk factor, 82–0126

CATECHOLAMINES
nicotine arterial effects in rats, 82–0134
nicotine effects on dopamine pathways in male rats, 82–0032
norepinephrine and epinephrine metabolism in female rats, nicotine effects, 82–0056
norepinephrine metabolism, nicotine effects in rats, 82–0057

CELLS
maleic hydrazide effects in rat lung, 82–0053
myeloid stem-cell proliferation in polycythemia, 82–0154
rat adrenal medulla, nicotine effects, 82–0055

CENTRAL NERVOUS SYSTEM
rat adrenal medulla, nicotine effects, 82–0055
vasopressin release in the cat, nicotine effect, 82–0049

CESSATION OF SMOKING
acupuncture treatment, psychological effect, 82–0217
adolescent and adult program approaches, 82–0209
antismoking programs for adolescents, 82–0197
cigarette holder for gradual phase-out of habit, 82–0211
coronary disease risk reduction, 82–0124, 82–0130
Gallup poll smoking survey, 1981, 82–0168
the Great American Smokeout campaign by the American Cancer Society, 82–0194
health care facility smoking prohibitions, lack of enforcement, 82–0175
health hazard perception as factor, 82–0189
legal and historical aspects of antismoking campaigns, 82–0207
lung neoplasm symptom in men over 40, 82–0066
maltol and ethyl maltol consumption, 82–0214
motivation modification, indications for success, 82–0169
myocardial infarction patients, habit modification, 82–0127
odoriferous substance injection into cigarettes, negative conditioning, 82–0223
physician's recognition of patients' smoking habits, 82–0193
psychosocial assets to handle stress without smoking, 82–0218
reinforcement by improvements in lung function, 82–0221
school program proposals, 82–0205
self-control and covert sensitization, 82–0216
stop smoking diet, 82–0219
televised national program in Finland, community organization, 82–0200
workshop followup survey, abstinence maintenance techniques, 82–0213

CESSATION OF SMOKING, METHODS
acupuncture, 82–0217
Hermano laying on of hands, evaluation, 82–0212
hypnotherapy, effectiveness of Damon Smoking Control program, 82–0220
nicotine fading and anxiety management program, 82–0210
rapid-smoking, results, 82–0222
self-administered and therapist-administered methods, 82–0215

CHILDREN
cigarette advertising in Australia, opinion of, 82–0166
health beliefs vs. smoking habits, 82–0183

CHROMOSOMES
cytogenetic analysis in diagnosis for malignancy, 82–0073
DNA replication and synthesis in smoke-exposed mice, 82–0048

maleic hydrazide effects in rat lung cells, 82–0053

CIGARETTE PAPER

fire safety modification recommendations, 82–0288
increasing porosity to reduce toxicity of cigarette smoke, 82–0291
perforated vs. unperforated, effects on smoke condensate, 82–0016
tar, nicotine, and carbon monoxide content reduction by smoke dilution, 82–0226

CIGARETTE SMOKE

aryl nitrile isolation and characterization from condensate, 82–0014
comparison of three bioassays to determine tobacco smoke condensate mutagenesis, 82–0019
gas chromatography analysis of constituents, 82–0025
moisture content and nicotine level relationship, 82–0017
nicotine retention of acetate and cellulose filters, 82–0024
nitric oxide and nitrogen dioxide, toxicological effects, 82–0093
toxicity of components, 82–0037

CIGARETTES

Algerian increase in production and consumption, 82–0244
consumption changes in United States, foreign markets, 82–0262
fire safety modification recommendations, 82–0288

CIGARETTES, HIGH-NICOTINE

pancreatic elastase activation in dogs, 82–0044

CIGARETTES, LESS HAZARDOUS

cellulose and combustion-modifying substances, 82–0230
chlorella green algae filter, physical and biological effects on smoke, 82–0239
composite tipping structure for air ventilation, milder cigarettes, 82–0240
consumer demand increase in United States, 82–0203
coronary heart disease reduction factor, 82–0074
deproteinized byproducts, economic factors, 82–0235
double-component filter, volatile aldehydes and hydrogen cyanide reduction, 82–0231
filter ventilation for tar, nicotine, and carbon monoxide reduction, 82–0238
manufacturing techniques, 82–0226
means of reducing toxicity of cigarette smoke, 82–0291
noxious substance index, recommended values, 82–0020
tar, nicotine, and carbon monoxide yields in United Kingdom cigarettes, 82–0030
Tobacco and Health Research Institute programs, University of Kentucky, 82–0294
tobacco plant modification vs. processing, 82–0001
toxic substances reduction, methods, 82–0209

CIGARETTES, LOW-NICOTINE

airway response to inhaled smoke, 82–0090
manufacturing techniques, 82–0226
puff volume relationship, 82–0186
reduced nicotine and anxiety management program, 82–0210
thiocyanate and carbon monoxide levels in smokers, 82–0040

CIGARETTES, LOW-TAR

Barclay promotion by Brown and Williamson, 82–0260
Canadian smoking trends, 1965-1979, 82–0161
Lorillard's emphasis, 82–0266
popularity in the United States, 82–0245, 82–0274
thiocyanate and carbon monoxide levels in smokers, 82–0040
tobacco plant modification vs. processing, 82–0001

CIGARS

Algerian increase in production and consumption, 82–0244

COCARCINOGENS

benzo(*a*)pyrene and asbestos in lung metabolism, 82–0072
tumorigenicity of smoke condensate, 82–0081

COFFEE CONSUMPTION

interaction with smoking, effect on pulse and blood pressure, 82–0117
occupational stress responses, 82–0163

CONTRACEPTIVES, ORAL

plasma levels of steroids, smoking effects, 82–0036
thrombosis risk factors in women, smoking effects, 82–0115

COPPER

nicotine effects on toxicity in small intestine, 82–0047

CORONARY DISEASE

aortic atherosclerosis and smoking, 82–0133
bypass operations, smoking as risk factor, 82–0111
filter cigarettes, disease risk reduction, 82–0074
mortality, cigarette smokers vs. quitters, 82–0112
mortality decline in New Zealand, reduced smoking as factor, 82–0110
multi-factor influences in smoking relationship, 82–0289
myocardial infarction, coronary occlusion and cholesterol as risk factors, 82–0120
myocardial infarction patients, smoking cessation rates, 82–0127
platelet sensitivity to antiaggregatory prostaglandins, 82–0132
preventable risk factors, smoking, 82–0123

- quantification of hospital patient smoking, 82–0063
- reconstructive arterial surgery, smoking-related risk, 82–0118
- risk factors, smoking effects, 82–0116, 82–0121, 82–0131, 82–0135
- risk factors used in diagnosis, 82–0119
- risk reduction by cessation of smoking, 82–0124
- trace metals level as risk factor, 82–0126

COST BENEFITS ANALYSIS
- economic and health consequences of smoking in Poland, 82–0061
- public communications antismoking campaign, seven-step strategy, 82–0201

COTININE
- levels in beagle dogs exposed to smoke, 82–0045
- radioimmunoassay technique, 82–0042

COUGH
- respiratory distress in asthmatics, bronchitics, emphysematics, smoking effects, 82–0106

CYANIDES
- thiocyanate serum levels in smokers and nonsmokers, 82–0041

DENTAL DISEASES
- cyclic nucleotide levels in gingival tissue, smokers vs. nonsmokers, 82–0156
- smokeless tobacco health hazards, 82–0153

DIET
- base-forming foods, in smoking cessation plan, 82–0219
- high-density lipoprotein cholesterol, smoking and weight loss effects, 82–0129
- tobacco, alcohol, and nutrition in pancreatic carcinoma etiology, 82–0082

DNA
- benzo(*a*)pyrene removal from hamster tracheal epithelial cells, 82–0067
- cytogenetic analysis in diagnosis for malignancy, 82–0073
- measurement of DNA synthesis as a bioassay of tobacco smoke mutagenesis, 82–0019
- replication and synthesis in smoke-exposed mice, 82–0048

DRUG ABUSE
- behavioral coping strategy, 82–0164
- national trends, high school seniors 1975-1979, 82–0174

DRUG METABOLISM
- β-adrenoceptor blocking drug treatment, smoking effects, 82–0122
- *d*-amphetamine-induced increases in tobacco consumption, 82–0172
- coffee drinking and smoker modeling vs. smoking habit, 82–0181
- nicotine emesis in cats, inhibited by morphine, methadone, and pethidine, 82–0035
- oxazepam clearance in human plasma, smoking, sex, and age relation, 82–0046
- plasma propranolol, age, smoking and hyperthyroidism effect, 82–0039
- Raynaud's phenomenon, promotion by smoking and chemotherapeutic drugs, 82–0136
- salicylate pulmonary edema, smoking effect, 82–0098

DUST
- coal miners lung disease mortality, smoking relationship, 82–0071
- coal miners lung function effects, smoking relationship, 82–0108
- lung impairment, additive effect of smoking, 82–0105
- metalliferous mine dust and smoking effects on lung function, 82–0088

EARS
- eye pigmentation and smoking association with hearing level, 82–0152

ECONOMICS
- Algerian expansion of tobacco goods production and consumption, 82–0244
- alternative crops for tobacco farmers, prospects and problems, 82–0241
- American Medical Association, tobacco stock sales, 82–0206
- British cigarette consumption decline, increased exports, 82–0256
- cigarette consumption trends in 1980, 82–0245
- company-sponsored withdrawal clinics, benefits, 82–0202
- corporate profiles of the "big six" American tobacco companies, 82–0268
- domestic tobacco industry, recent changes, 82–0252
- expanding tobacco exports by program changes and freer trade, 82–0258
- Federal tobacco program, 82–0253, 82–0271
- flue-cured tobacco farm consolidation and mechanization, 82–0250
- foreign market gains and diversification of U.S. tobacco companies, 82–0262
- India, tobacco exports from 1979 to 1980, 82–0272
- Japanese tobacco industry, 1980, 82–0275
- Lorillard's increasing market share, emphasis on low-tar cigarettes, 82–0266
- low-tar cigarettes' increasing popularity in the United States, 82–0274
- Maxwell 1981 Fact Book, tobacco industry status report, 82–0263

SUBJECT INDEX

- 120 mm cigarettes, increased sales, 82–0267
- North Carolina, tobacco crops for protein vs. smoking, 82–0235
- North Carolina's tobacco industry, 82–0255, 82–0292
- Philip Morris, domestic and foreign markets and diversification, 82–0273, 82–0279
- Poland, consequences of smoking, 82–0061
- R. J. Reynolds Industries, growth and diversification, 82–0277
- smokeless tobacco sales, 82–0295
- Third World countries as new markets, 82–0257
- tobacco acreage allotment system, 82–0261, 82–0283
- tobacco conglomerates, worldwide growth and diversification, 82–0248
- tobacco farms and farmers, decreasing numbers, 82–0243
- tobacco industry growth constraints, foreign and domestic, 82–0247
- tobacco industry growth, health education effects, 82–0203
- tobacco production and price stabilization attempts, 82–0242
- tobacco program modification or abolition, proposals, 82–0270
- tobacco program organizations, government and private, 82–0249
- U.S. flue-cured tobacco exports decline, 82–0246
- U.S. tobacco exports, 1976-1980, 82–0265
- United Kingdom tax increases on tobacco products, 82–0254
- USDA tobacco price-support program, 82–0282
- West German cigarette production, 82–0269
- West German tobacco market, 1979, 82–0264
- work force reductions in tobacco manufacturing, possible alternatives, 82–0278
- world market competition for U.S. growers, 82–0259

EMBRYO
- benzo(*a*)pyrene exposure in mice, 82–0143

EMPHYSEMA
- incidence among smokers vs. nonsmokers, 82–0060
- temporal pattern of respiratory distress, smoking effects, 82–0106

ENZYME ACTIVITY
- acid phosphodiesterases purification and characterization, 82–0023
- aryl hydrocarbon hydroxylase induction in maternal and neonatal lymphocytes, 82–0141
- pancreatic elastase activation in dogs exposed to cigarette smoke, 82–0044

ENZYMES
- acid phosphodiesterases, 82–0023

ERYTHROCYTES
- prolonged smoke exposure effects in primates, 82–0092

ESOPHAGEAL NEOPLASMS
- smokeless tobacco health hazards, 82–0153

EX-SMOKERS
- abstinence maintenance techniques, 82–0213
- attitude toward health hazards of smoking, 82–0189
- coronary heart disease, risk and mortality, 82–0112, 82–0130
- serum IgE levels, 82–0151

EYES
- pigmentation and smoking association with hearing, 82–0152

FEDERAL REPUBLIC OF GERMANY
- cigarette production increases, 82–0269
- tobacco market, 1979, 82–0264

FERTILITY
- benzo(*a*)pyrene exposure in mice, 82–0143

FERTILIZATION, TOBACCO
- increased leaf levels of nitrogenous constituents, 82–0015
- leaf and smoke constituent changes, 82–0007

FETAL GROWTH
- α-aminoisobutyric acid concentration in human placental villi, maternal smoking effect, 82–0144
- benzo(*a*)pyrene exposure in mice, 82–0143
- maternal smoking effects, 82–0137
- nutrition and maternal smoking relationship, 82–0145

FETAL MORTALITY
- stillbirth, increased incidence among smoking mothers, 82–0150

FIBRINOLYSIS
- fibrinogen levels, smoking effects, 82–0054

FILTERS
- acetate and cellulose, nicotine retention, 82–0024
- chlorella green algae, physical and biological effects on smoke, 82–0239
- composite tipping structure, air-ventilated cigarette, 82–0240
- dilution systems to reduce tar, nicotine, and carbon monoxide, 82–0226
- double-component, volatile aldehydes and hydrogen cyanide reduction, 82–0231
- elongated hollow sleeve, 82–0236
- inlet vents for increased draw resistance, 82–0233
- less hazardous cigarettes relationship, 82–0291
- ventilation for tar, nicotine, and carbon monoxide reduction, 82–0238

SUBJECT INDEX

FINLAND
- North Karelia Project for cardiovascular disease control, 82–0204
- televised nationwide smoking cessation program, community organization, 82–0200

GAS PHASE, CIGARETTE SMOKE
- free radicals, nature and concentration, 82–0027

GAS PHASE, TOBACCO SMOKE
- nitric oxide and nitrogen dioxide, toxicological effects, 82–0093

GASTRIC SECRETION
- duodenogastric reflux of bile acids, 82–0159

GASTROINTESTINAL SYSTEM
- nicotine absorption rate, 82–0034

GENETICS, PLANTS
- breeding to alter tobacco chemical constituents, 82–0005
- improved tobacco quality and reduced health risks, 82–0002

HEALTH EDUCATION
- actions by developed and developing nations, 82–0281
- advertising demystified, Australian school program, 82–0162
- informative, preventive, radical, and self-empowerment methods, 82–0208
- medical advice's effect on maternal smoking, 82–0138
- noxious substance index, recommended values, 82–0020
- Ombudsman primary prevention program, effectiveness, 82–0199
- public communications antismoking campaign, seven-step strategy, 82–0201
- Public Health Service 1980 recommendations, 82–0203
- research review, current concerns about smoking-related diseases, 82–0062
- school program proposals, 82–0205
- school program, smoking as drug addiction, 82–0195
- social and psychological approaches for adolescents, 82–0209
- televised smoking cessation program in Finland, community organization, 82–0200
- tobacco health education curriculum for elementary students, 82–0191
- university students in health professions, survey of knowledge of smoking consequences, 82–0160

HEALTH PRACTICES
- role model, inappropriateness of physicians who smoke, 82–0175

HEART
- nicotine arterial effects in rats, 82–0134
- rat cardiac muscle, tobacco smoke effects, 82–0125

HEART DISEASES
- decline in New Zealand, reduced smoking as factor, 82–0110
- filter cigarettes, possible rate reduction, 82–0074
- mortality, cigarette smokers vs. quitters, 82–0112
- multi-factor influences in smoking relationship, 82–0289
- platelet sensitivity to antiaggregatory prostaglandins, 82–0132
- risk factors, with smoking, 82–0116

HEART RATE
- β-adrenoceptor blocking drug treatment, smoking effects, 82–0122
- response to varied doses of nicotine, 82–0128

HEMATOCRIT
- determination before and after smoking, 82–0054

HEMODYNAMICS
- β-adrenoceptor blocking drug treatment, smoking effects, 82–0122

HEMOGLOBINS
- maternal smoking effects on infant pCOHb blood levels and birth weight, 82–0139
- prolonged smoke exposure effects in primates, 82–0092

HEREDITY
- mutagenic activity of cigarette smoke, 82–0146

HORMONES
- vasopressin release in the cat, nicotine effect, 82–0049

HUNGARY
- coal miners, smoking and dust exposure effects, 82–0108

HYPERLIPIDEMIA
- risk factor in diagnosis of coronary artery disease, 82–0119

HYPERTENSION
- β-adrenoceptor blocking drug treatment, smoking effects, 82–0122
- coffee and smoking effects on pulse rate and blood pressure, 82–0117

HYPNOSIS
- Damon Smoking Control program effectiveness, 82–0220

IMMUNE SYSTEM
- Farmer's Lung Disease, antibody prevalence among smokers vs. nonsmokers, 82–0097

serum IgE levels in smokers, ex-smokers, and nonsmokers, 82-0151

IMMUNOGLOBULINS
serum IgE levels in smokers, ex-smokers, and nonsmokers, 82-0151

INDIA
body measurement relationship to smoking in Calcutta's elite class, 82-0182
school boys smoking habit, 82-0176
tobacco exports, 1979 to 1980, 82-0272

INDOLES
amino-α-carbolines in cigarette smoke condensate, 82-0022

INFANT MORTALITY
maternal smoking effects, 82-0147
nutrition and maternal smoking relationship, 82-0145

INFANTS
maternal smoking, reduced weight and length, 82-0148

INTESTINES
copper and manganese toxicity, nicotine effect on, 82-0047

IVORY COAST
Abidjan residents smoking habits, 82-0184

JAPAN
tobacco production and sales, 1980, 82-0275

KENTUCKY
maleic hydrazide residues in tobacco, 82-0004

KETONES
tobacco expansion process with hot inert solid particles, 82-0229

KOREA
tobacco industry, growth and outlook, 82-0276

LABELING
noxious substance index, recommended values, 82-0020

LARYNGEAL NEOPLASMS
rabbit vocal cord mucosa, smoking effect, 82-0068
smokeless tobacco health hazards, 82-0153

LEAD
maternal and newborn hair content, 82-0140

LEGISLATION
Agricultural Adjustment Act of 1938, proposed modifications, 82-0270
antismoking actions by developed and developing nations, 82-0281
antismoking regulations in New Jersey, 82-0285
bibliography of smoking and health policies and actions, 82-0293
Cigarette Safety Act endorsement, 82-0288
Federal tobacco program landmarks, 82-0271
government control of smoking, comments on individual rights, 82-0287
legal and policy issues for the 1980s, 82-0284
proposed amendment to prohibit tobacco allotment ownership by nonproducers, 82-0283
public smoking and advertising restrictions, 82-0280
State laws restricting public smoking, trends, 82-0286
tobacco acreage allotment system, 82-0261
USDA tobacco price-support program, 82-0282
worldwide actions against smoking, 82-0062

LEUKOPLAKIA
smokeless tobacco health hazards, 82-0153

LIPOPROTEINS
coronary disease risk reduction by cessation of smoking, 82-0124

LIVER CIRRHOSIS
alcohol and smoking, oral mucosa effects, 82-0043
tobacco, alcohol, and nutrition as factors, 82-0070

LIVER NEOPLASMS
tobacco, alcohol, and nutrition as factors, 82-0070

LUNG DISEASES
coal miners' mortality, dust and smoking as factors, 82-0071
dust exposure and smoking effects, 82-0105
lymphocyte subpopulations in smokers vs. nonsmokers, 82-0096
radioaerosol lung patterns in asymptomatic smokers, 82-0109

LUNG FUNCTION
aerosol respiratory deposition in smokers and nonsmokers, 82-0107
airway response to inhaled cigarette smoke, 82-0090
arachidonic acid metabolism in smokers vs. nonsmokers, 82-0102
asthmatics, passive smoking effects, 82-0091
bronchial lability abnormalities in healthy smokers, 82-0087
cessation of smoking reinforced by improved exhalation, 82-0221
coal miners, smoking and dust exposure effects, 82-0108
Farmer's Lung Disease in smokers vs. nonsmokers, 82-0097
metalliferous mine dust exposure and smoking, 82-0088
phospholipid metabolism in rabbit lung, smoking effects, 82-0104

prolonged smoke exposure effects in primates, 82–0092
pulmonary vascular responses to cigarette smoke in pig lung, 82–0095
respiratory abnormalities in sons of pulmonary patients, 82–0099
respiratory distress in asthmatics, bronchitics, emphysematics, smoking effects, 82–0106
salicylate pulmonary edema, smoking effect, 82–0098
tracheal submucosal glands in rats, smoke exposure effects, 82–0103

LUNG NEOPLASMS
aversion to smoking as symptom, 82–0066
benzo(*a*)pyrene and asbestos in pulmonary metabolism, 82–0072
benzo(*a*)pyrene metabolism in etiology of, 82–0076
filter cigarettes, risk reduction, 82–0074
Mayo Clinic screening of squamous and small cell carcinoma among male smokers, 82–0086
mortality attributable to smoking, 82–0065
multi-factor influences in smoking relationship, 82–0289
occupational exposure and smoking as risk factors, 82–0084
passive smoking as risk factor, 82–0187
quantification of smokers' characteristics, 82–0063

LUNGS
DNA replication and synthesis in smoke-exposed mice, 82–0048
norepinephrine metabolism, nicotine effects on rats, 82–0057
tracheal submucosal glands in rats, smoke exposure effects, 82–0103

LYMPHOCYTES
aryl hydrocarbon hydroxylase induction from mothers and neonates, 82–0141
subpopulations in smokers vs. nonsmokers, 82–0096

MARIHUANA SMOKING
behavioral coping strategy, 82–0164
health hazards and therapeutic potential, 82–0155

MASS MEDIA
advertising and marketing in American tobacco industry, 82–0268
televised smoking cessation program in Finland, community organization, 82–0200

MATERNAL-FETAL EXCHANGE
benzo(*a*)pyrene exposure in mice, 82–0143
nicotine effects on pregnancy, 82–0150

MATERNAL SMOKING
α-aminoisobutyric acid concentration in human placental villi, 82–0144
aryl hydrocarbon hydroxylase induction, 82–0141
breast feeding duration effects, 82–0149
cadmium and lead content in hair of mothers and newborns, 82–0140
fetal growth reduction, 82–0137
infant pO_2, pCO_2, pCOHb blood levels and birth weight, effects of, 82–0139
medical advice's effect on frequency, 82–0138
neonatal and long-term effects, weight and length, 82–0148
nicotine effects on pregnancy, 82–0150
nutrition relationship in fetal growth effects, 82–0145
perinatal mortality and reduced birth weight, 82–0147
pregnancy effects, alcohol use interrelationships, 82–0142

MATHEMATICAL MODELS
Bayes' theorem, multivariate estimation methods for exposure-specific incidence, 82–0069

METALS, METAL ISOTOPES
copper and manganese toxicity in small intestine, nicotine effects on, 82–0047

MICHIGAN
Washtenaw County schoolchildren's smoking habits, 82–0183

MORBIDITY
bibliography of smoking and health topics, 82–0293
elderly persons, increased incidence in smokers, 82–0188
filter cigarettes, coronary heart disease risk reduction, 82–0074
Polish industrial workers, smokers vs. nonsmokers, 82–0061
research review, current concerns about smoking-related diseases, 82–0062
Tobacco and Health Research Institute programs, University of Kentucky, 82–0294

MORTALITY
coronary heart disease, smokers vs. quitters, 82–0112
lung and bladder cancer mortality attributable to smoking, 82–0065
Polish industrial workers, smokers vs. nonsmokers, 82–0061
polycythemias, smoking relationship, 82–0154
premature deaths among smokers vs. nonsmokers, 82–0060
research review, current concerns about smoking-related diseases, 82–0062
survival rates in rats exposed to tar and nicotine, 82–0083

MOTIVATION
coffee drinking and smoker modeling vs. smoking habit, 82–0181

health risk awareness, declining percentage of smokers, 82–0167
impaired drivers, lifestyle hazards, 82–0190
myocardial infarction patients, smoking cessation rates, 82–0127
peer values and role models in adolescent smoking, 82–0173
psychosocial assets to handle stress without smoking, 82–0218
reinforcement in squirrel monkeys by nicotine and visual stimulus, 82–0170
self-control treatment of addictive behavior, 82–0178

MOUTH NEOPLASMS
smokeless tobacco health hazards, 82–0153
snuff and chewing tobacco relationship, study criticisms, 82–0295

MUTAGENESIS
comparison of three bioassays to determine tobacco smoke condensate mutagenesis, 82–0019

MUTAGENS
amino-α-carbolines in cigarette smoke condensate, 82–0022
benzo(*a*)pyrene removal from hamster tracheal epithelial cells, 82–0067

MYOCARDIAL INFARCT
causal relationship with smoking questioned, 82–0130
coronary occlusion and cholesterol as risk factors, 82–0120
incidence among smokers vs. nonsmokers, 82–0060
platelet sensitivity to antiaggregatory prostaglandins, 82–0132
preventable risk factors, smoking, 82–0123
smoking cessation rates among patients, 82–0127

NEBRASKA
smoking behavior among adolescents, 82–0179

NEONATE
cadmium and lead content in hair of mothers and newborns, 82–0140
maternal drinking and smoking interrelationships, 82–0142
maternal smoking effects, 82–0150
maternal smoking effects on infant pO_2, pCO_2, pCOHb blood levels and birth weight, 82–0139
maternal smoking, reduced weight and length, 82–0148

NEOPLASMS
carcinogenesis, chemical induction mechanisms, 82–0078
metastases facilitated by smoking, 82–0079

NEURONS
nicotine effects on dopamine pathways in male rats, 82–0032

NEUROTICISM
treatment factor, Hermano laying on of hands smoking cessation method, 82–0212

NEW JERSEY
antismoking legislation, 82–0285

NEW YORK STATE
antismoking curriculum for schools, 82–0192

NEW ZEALAND
coronary heart disease mortality decline, reduced smoking as factor, 82–0110

NICOTINE
β-adrenoceptor blocking drug treatment, smoking effects, 82–0122
anatomical effects on rat adrenal medulla, 82–0055
arterial effects in rats, 82–0134
behavior reinforcement in squirrel monkeys, 82–0170
bioactivity of four synthetic analogs on animal tissues, 82–0051
carboxyhemoglobin trough concentrations as indicator of exposure, 82–0033
cardiovascular response to varied doses, 82–0128
dopamine pathways in male rats, effects on, 82–0032
eye pigmentation and smoking association with hearing level, 82–0152
gastrointestinal absorption rate, 82–0034
levels in beagle dogs exposed to smoke, 82–0045
plasma concentrations in snuff users vs. smokers, 82–0052
self-control treatment of addictive behavior, 82–0178
survival rates, weight gain, and carcinogenesis in exposed rats, 82–0083

NICOTINE CONTENT
acetate and cellulose filters, retention qualities, 82–0024
filter ventilation for reduced yield, 82–0238
moisture content relationship in cigarette smoke, 82–0017
nitrogen fertilizer influence, 82–0007
noxious substance index, recommended values, 82–0020
United Kingdom cigarettes, yields, 82–0030

NICOTINE METABOLISM
acid vs. base-forming diets for smokers, effects on, 82–0219
norepinephrine and epinephrine metabolism effects in female rats, 82–0056
norepinephrine metabolism in rats, 82–0057
pancreatic elastase activation in dogs, 82–0044

pulmonary inhibitor characteristics, 82–0013
radioimmunoassay technique, 82–0042

NICOTINE PHARMACOLOGY
arginine vasopressin release in the cat, 82–0049
copper and manganese toxicity effects in small intestine, 82–0047
cue receptors in male albino rats, 82–0050
emesis in cats, inhibited by morphine, methadone, and pethidine, 82–0035
fighting behavior inhibition in rats, 82–0038
neonatal effects from maternal smoking, 82–0150
nitrogen reactivity in nicotinoids, steric, and conformational effects, 82–0029

NITROGEN CONTENT
dark tobacco leaf levels, fertilization effect, 82–0015
fertilizer and genotype influences, 82–0007
infrared reflectance spectroscopy for tobacco constituent analysis, 82–0028

NITROGEN OXIDES
indoor air pollution from smoking, 82–0290
nitrogen fertilizer influence, 82–0007
noxious substance index, recommended values, 82–0020
pulmonary pathologies, 82–0093

NITROSAMINES
dimethylnitrosamine carcinogenic effect in mice, promotion by butylated hydroxytoluene, 82–0085

NONSMOKERS
serum IgE levels, 82–0151
student attitudes toward cigarette smoke pollution, 82–0185

NORTH CAROLINA
economic transitions and the tobacco industry, 82–0255
farm mechanization, adaptation case study, 82–0243
tobacco economy's changes and challenges, 82–0292
tobacco for protein vs. smoking, economic factors, 82–0235

NUCLEIC ACIDS
cyclic nucleotide levels in gingival tissue, smokers vs. nonsmokers, 82–0156

OBSTRUCTIVE AIRWAY DISEASES
alpha-l-antitrypsis deficiency and chronic obstructive pulmonary disease, smoking relation, 82–0100
atopic predisposition in male smokers, 82–0089
lung function abnormalities in sons of pulmonary patients, 82–0099

OCCUPATIONAL DISEASES
dust exposure and smoking in lung disease, 82–0105
Farmer's Lung Disease, 82–0097
green tobacco sickness among tobacco harvest workers, 82–0042
lung cancer and occupational lung disease risk factors, with smoking, 82–0084
lung disease mortality in coal miners, dust and smoking as factors, 82–0071
stress, behavioral responses, 82–0163

OCCUPATIONS
agricultural workers, 82–0097
coal miners, 82–0063, 82–0071, 82–0108
physicians, 82–0193

OVARIES
benzo(*a*)pyrene exposure, infertility in mice, 82–0143

OXYGEN TRANSPORT
bioassay determination of smoke toxicity, 82–0018
prolonged smoke exposure effects in primates, 82–0092
reduction by carboxyhemoglobinemia in polycythemias, 82–0154

PANCREAS
elastase activation in dogs exposed to cigarette smoke, 82–0044

PANCREATIC NEOPLASMS
tobacco, alcohol, and diet as factors, 82–0082

PARENTAL SMOKING
genetic risk, 82–0146
India school boys smoking influence, 82–0176

PASSIVE SMOKING
asthmatics, pulmonary function effect, 82–0091
bibliography of smoking and health topics, 82–0293
epidemiological studies criticized, 82–0130
legal and historical aspects of smoking control programs, 82–0207
legal and policy issues for the 1980s, 82–0284
lung cancer risks, 82–0187
student attitudes toward cigarette smoke pollution, 82–0185

PATENTS
alcohol derivatives, smoking tobacco organoleptic enhancement, 82–0225
benzodioxanones for organoleptic enhancement of tobacco, 82–0224
chlorella green algae filter, physical and biological effects on smoke, 82–0239
cigarette holder for gradual phase-out of smoking, 82–0211
composite tipping structure, air-ventilated cigarette, 82–0240
compound for enhancement of flavor and aroma, 82–0234

- double-component filter to reduce volatile aldehydes and hydrogen cyanide, 82–0231
- filter element with elongated hollow sleeve, 82–0236
- filter inlet vents for increased draw resistance, 82–0233
- filter ventilation for tar, nicotine, and carbon monoxide reduction, 82–0238
- maltol and ethyl maltol consumption for cessation of smoking, 82–0214
- odoriferous substance injection into cigarettes, negative conditioning, 82–0223
- reconstituted tobacco by papermaking process, 82–0232
- smokable product of cellulose and combustion-modifying substances, 82–0230
- sweet proteins as flavor enhancers, 82–0237
- tobacco expansion process with hot inert solid particles, 82–0229
- tricyclic lactones as organoleptic agents, 82–0227, 82–0228

PENNSYLVANIA
- tobacco growing practices and suitable varieties, 82–0011

PEPTIC ULCER
- duodenogastric reflux of bile acids, 82–0159
- incidence among smokers vs. nonsmokers, 82–0060
- increased levels for inhaling smokers, 82–0157

PERIODONTAL DISEASES
- cyclic nucleotide levels in gingival tissue, smokers vs. nonsmokers, 82–0156

PERSONALITY
- deviant behavior in adolescents, predictive value of smoking habits, 82–0165

PESTICIDES
- maleic hydrazide, 82–0003, 82–0053
- tobacco growing practices and guidelines in Pennsylvania, 82–0011

PHARYNGEAL NEOPLASMS
- rabbit vocal cord mucosa, smoking effect, 82–0068
- smokeless tobacco health hazards, 82–0153

PHENOL CONTENT
- liquid chromatographic determination in tobacco smoke, 82–0021
- smoke condensate components isolated and characterized, 82–0014
- stalk position and cultivar differences, 82–0031

PHYSICAL ACTIVITY
- young males, smoking effects, 82–0101

PHYSICAL FITNESS
- inverse relationship with cigarette consumption, 82–0116

PLACENTA
- α-aminoisobutyric acid concentration, maternal smoking effect, 82–0144

POLAND
- economic and health consequences of smoking, 82–0061
- Katowice District smoking survey, 82–0169

PREGNANCY
- α-aminoisobutyric acid concentration in human placental villi, maternal smoking effect, 82–0144
- benzo(*a*)pyrene exposure effect in mice, 82–0143
- genetic risk of smoking, 82–0146
- maternal drinking and smoking interrelationships, 82–0142
- maternal smoking, short- and long-term effects, 82–0148
- nutrition and maternal smoking, fetal growth effects, 82–0145

PREVENTION OF SMOKING
- advertising demystified, Australian school program, 82–0162
- educational, preventive, radical, and self-empowerment methods of health education, 82–0208
- films, junior high school program, 82–0196
- Gallup poll smoking survey, increasing public support for tobacco restrictions, 82–0167
- legal and historical aspects of antismoking campaigns, 82–0207
- Ombudsman primary prevention program, effectiveness, 82–0199
- programs for adolescents, 82–0197
- public communications seven-step strategy, 82–0201
- school program proposals, 82–0205
- school program, smoking as drug addiction, 82–0195
- youth antismoking programs, 82–0209

PROGRAMS AND POLICIES
- bibliography of smoking and health policies and actions, 82–0293

PROTEIN CONTENT
- extractable yield from leaf varieties, harvesting effects, 82–0010
- stalk position and cultivar differences, 82–0031
- tobacco crops for protein vs. smoking, economic factors, 82–0235

PROTEINS
- amino acid content in tobacco leaves, light and temperature effects, 82–0009
- cyclic nucleotide levels in gingival tissue, smokers vs. nonsmokers, 82–0156
- fibronectin concentrations and secretion, smoking effects, 82–0059

PULMONARY ALVEOLAR MACROPHAGES
- arachidonic acid metabolism in smokers vs. nonsmokers, 82–0102
- fibronectin secretion and concentration, smoking effects, 82–0059
- prolonged smoke exposure effects in primates, 82–0092

PULMONARY ALVEOLI
- salicylate edema, smoking effect, 82–0098

PULMONARY CLEARANCE
- bronchial lability abnormalities in healthy smokers, 82–0087
- inhibitor in tobacco smoke condensate, 82–0013
- metalliferous mine dust exposure and smoking, 82–0088
- phospholipid metabolism in rabbit lung, smoking effects, 82–0104

RECIDIVISM
- rapid-smoking cessation method, short- vs. long-term results, 82–0222

REDUCTION OF SMOKING
- acupuncture treatment, psychological effect, 82–0217
- cigarette holder for decreasing the amount of each cigarette consumed, 82–0211
- Hermano laying on of hands method, evaluation, 82–0212
- hypnotherapy, effectiveness of Damon Smoking Control program, 82–0220
- lung neoplasm symptom in men over 40, 82–0066
- maltol and ethyl maltol consumption, 82–0214
- myocardial infarction patients, habit modification, 82–0127
- nicotine fading and anxiety management program, 82–0210
- North Karelia Project for cardiovascular disease control, 82–0204
- physician's recognition of patients' smoking habits, 82–0193
- rapid-smoking method, results, 82–0222
- self-administered and therapist-administered methods, 82–0215
- SOS Stop Smoking Clinic, education and reinforcement, 82–0202

RESPIRATION
- phospholipid metabolism in rabbit lung, smoking effects, 82–0104

RESPIRATORY FUNCTION TESTS
- aerosol respiratory deposition in smokers and nonsmokers, 82–0107
- airway response to inhaled cigarette smoke, 82–0090
- arachidonic acid metabolism in smokers vs. nonsmokers, 82–0102
- asthmatics, passive smoking effects, 82–0091
- bronchial lability abnormalities in healthy smokers, 82–0087
- bronchiectasis, smoking relationship, 82–0094
- cessation of smoking reinforced by improved exhalation, 82–0221
- coal miners, smoking and dust exposure effects, 82–0108
- chronic airflow limitation in peptic ulcer individuals, 82–0157
- Farmer's Lung Disease in smokers vs. nonsmokers, 82–0097
- lung abnormalities in sons of pulmonary patients, 82–0099
- lymphocyte subpopulations in smokers vs. nonsmokers, 82–0096
- metalliferous mine dust exposure and smoking, 82–0088
- phospholipid metabolism in rabbit lung, smoking effects, 82–0104
- prolonged smoke exposure effects in primates, 82–0092
- tracheal submucosal glands in rats, smoke exposure effects, 82–0103
- young males, smoking effects, 82–0101

RESPIRATORY MUCOSA
- respiratory distress in asthmatics, bronchitics, emphysematics, smoking effects, 82–0106

RESPIRATORY SYMPTOMS
- young males, smoking effects, 82–0101

RESPIRATORY SYSTEM
- airway response to inhaled cigarette smoke, 82–0090
- tracheal submucosal glands in rats, smoke exposure effects, 82–0103

RESPIRATORY TRACT DISEASES
- lymphocyte subpopulations in smokers vs. nonsmokers, 82–0096

RESPIRATORY TRACT NEOPLASMS
- cytogenetic analysis in diagnosis for malignancy, 82–0073
- Mayo Clinic screening of squamous and small cell carcinoma among male smokers, 82–0086
- rabbit vocal cord mucosa, smoking effect, 82–0068

SALIVA
- reliability of thiocyanate in smoking exposure assessment, 82–0026

SEX BEHAVIOR
- activity of husbands and smoking as factors in cervical cancer, 82–0064

SEX RATIO
- blood clotting rates, smoking effects in males vs. females, 82–0054
- consumption patterns, United Kingdom 1972-1980, 82–0180
- lung and bladder mortality attributable to smoking, 82–0065
- mortality excesses among male hospital patients, 82–0060
- smoke exposure variations, male vs. female, 82–0063

SLEEP
- cannabis disruption of in mice, 82–0158

SMALL AIRWAYS DISEASE
- radioaerosol lung patterns in smokers vs. nonsmokers, 82–0109

SMOKE INHALATION, ANIMAL
- rat cardiac muscle responses, 82–0125

SMOKE STREAMS
- benzodioxanones for organoleptic enhancement, 82–0224
- filter inlet vents for increased draw resistance, 82–0233
- free radicals, nature and concentration, 82–0027
- nitric oxide and nitrogen dioxide, toxicological effects, 82–0093
- perforated and unperforated paper, effects on chemical composition of condensates, 82–0016

SMOKING ARTICLES
- cigarette holder for decreasing the amount of each cigarette consumed, 82–0211
- filter element with elongated hollow sleeve, 82–0236

SMOKING CHARACTERISTICS
- *d*-amphetamine effects, 82–0172
- carbon monoxide and thiocyanate levels in low-yield cigarette smokers, 82–0040
- coffee drinking and smoker modeling vs. smoking habit, 82–0181
- puff volume relationship to lower nicotine cigarettes, 82–0186
- quantification of smokers' exposure, 82–0063

SMOKING CONTROL PROGRAMS
- actions by developed and developing nations, 82–0281
- hospital rooms, proposed prohibition of smoking, 82–0206
- legal and policy issues for the 1980s, 82–0284
- managers' attitudes toward smoking and nonsmoking employees, 82–0296
- New Jersey's antismoking legislation, 82–0285
- Ombudsman primary prevention program, effectiveness, 82–0199
- public communications seven-step strategy, 82–0201
- public smoking and advertising restrictions, 82–0280
- school education curriculum, New York State, 82–0192
- self-control and covert sensitization, 82–0216
- tobacco health education curriculum for elementary students, 82–0191

SMOKING HABIT
- Abidjan, Ivory Coast residents, 82–0184
- body size relationship in elite class of Calcutta men, 82–0182
- bronchogenic carcinoma detection by consumption level, 82–0075
- coffee drinking and smoker modeling effects, 82–0181
- elderly persons, increasing incidence, 82–0188
- India school boys, 82–0176
- motivation modification, indications for success, 82–0169
- national trends, high school seniors 1975-1979, 82–0174
- New Zealand adults, 82–0110
- Tasmanian high school students, 82–0177
- United Kingdom consumption patterns, 1972-1980, 82–0180

SMOKING SURVEYS
- Abidjan, Ivory Coast residents, 82–0184
- adolescents, prediction of deviant behavior, 82–0165
- Canadian trends, 1965-1979, 82–0161
- Gallup poll, 1981, 82–0167, 82–0168
- India school boys. 82–0176
- Michigan schoolchildren, 82–0183
- Nebraska adolescents, 82–0179
- Poland's Katowice District, 82–0169
- SOS Stop Smoking Clinic attendees, 82–0202
- Tasmanian high school students, 82–0177
- university students, high school smoking education programs effect, 82–0205
- university students in health professions, knowledge of smoking consequences, 82–0160

SNUFF-DIPPING
- nicotine plasma concentrations in users, 82–0052
- oral cancer relationship, 82–0295
- oral health hazards, 82–0153

SOCIOECONOMIC STATUS
- New Zealand adults smoking habits, 82–0110
- students in India, smoking relationship, 82–0176
- United Kingdom consumption patterns, 1972-1980, 82–0180

STATISTICAL ANALYSIS
- Bayes' theorem, multivariate estimation methods for exposure-specific incidence, 82–0069
- cigarette smoke toxicity, 82–0037
- Federal Trade Commission 1979 report on cigarettes, 82–0251

Gallup poll 1981 smoking survey, comparison with earlier polls, 82–0167, 82–0168

STEROIDS
oral contraceptives, smoking effects, 82–0036

STOMACH
duodenogastric reflux of bile acids, 82–0159
gastrointestinal absorption rate of nicotine, 82–0034

STRESS
handedness, smoking and drinking response, 82–0171
nicotine inhibition of fighting behavior in rats, 82–0038
occupational, behavioral responses, 82–0163
psychosocial assets for coping without smoking, 82–0218

STUDENTS
attitudes toward cigarette smoke pollution, 82–0185
health consequences of smoking survey, health professions students, 82–0160
India school boys smoking habits, 82–0176
national trends in drug abuse, high school seniors 1975-1979, 82–0174
Nebraska adolescents' smoking survey, 82–0179
school antismoking curriculum, New York State, 82–0192
smoking prevention programs for adolescents, 82–0197
social modeling films effect on behavior, 82–0196
Tasmanian high school students, smoking incidence, 82–0177
tobacco health education curriculum for elementary students, 82–0191

SWITZERLAND
tobacco, alcohol, and diet in pancreatic carcinoma, 82–0082
tobacco, alcohol, and nutrition as factors in liver cancer and cirrhosis, 82–0070

TAR CONTENT
low-yield cigarettes' increasing popularity in the United States, 82–0274
noxious substance index, recommended values, 82–0020
United Kingdom cigarettes, 82–0030

TAR REDUCTION
cigarette manufacturing techniques, 82–0226
content controls, three commercially successful techniques, 82–0274
filter ventilation for reduced yield, 82–0238

TARS, CIGARETTE
amino-α-carbolines in smoke condensate, 82–0022
benzo (*a*)pyrene metabolism, effect on, 82–0076
nitrogen fertilizer influence, 82–0007
perforated and unperforated paper, effects on chemical composition of condensates, 82–0016
survival rates, weight gain, and carcinogenesis in exposed rats, 82–0083

TARS, TOBACCO
bioassay determination of toxicity, 82–0018
free radicals, nature and concentration in tobacco smoke, 82–0027

TASTE
ultra-low-tar cigarettes, Barclay's claim, 82–0260

TAXATION
antismoking messages financed by tobacco advertisement taxes, British proposal, 82–0198
British tobacco products increases, 82–0254, 82–0256
State and local increases, 82–0203
State cigarette excise taxes, trends, 82–0286

THIOCYANATES
levels in very low-yield cigarette smokers, 82–0040
reliability of smoking exposure assessment, 82–0026
serum levels in smokers and nonsmokers, 82–0041

THROMBOEMBOLISM
oral contraceptives and smoking as risk factors in women, 82–0115

TOBACCO
Fifth Workshop Conference Proceedings, papers, 82–0058

TOBACCO ADDITIVES
alcohol derivatives, organoleptic enhancement, 82–0225
benzodioxanones for organoleptic enhancement, 82–0224
means of reducing toxicity of cigarette smoke, 82–0291
odoriferous substance injection into cigarettes, negative conditioning, 82–0223
patented compound for enhancement of flavor and aroma, 82–0234
sweet proteins as flavor enhancers, 82–0237
tricyclic lactones as organoleptic agents, 82–0227, 82–0228

TOBACCO AROMA
alcohol derivatives, organoleptic enhancement, 82–0225
benzodioxanones for organoleptic enhancement, 82–0224
cellulose and combustion-modifying substances, 82–0230
odoriferous substance injection into cigarettes, negative conditioning, 82–0223
patented compound for enhancement of organoleptic qualities, 82–0234

- tricyclic lactones as organoleptic agents, 82–0227, 82–0228

TOBACCO CHEWING

- Nebraska adolescents, 82–0179
- oral cancer relationship, 82–0295
- oral health hazards, 82–0153

TOBACCO CONTAMINANTS

- maleic hydrazide, 82–0003, 82–0004, 82–0053

TOBACCO CURING

- bulk barns, 82–0243, 82–0261
- practices and varieties in Pennsylvania, 82–0011
- variation in constituent levels, 82–0015

TOBACCO EXTRACTS

- acid phosphodiesterases purification and characterization, 82–0023
- plant growth and air curing effects on midvein alcohols, 82–0012
- protein yield from leaf varieties, harvesting effects, 82–0010

TOBACCO FLAVOR

- alcohol derivatives, organoleptic enhancement, 82–0225
- benzodioxanones for organoleptic enhancement, 82–0224
- cellulose and combustion-modifying substances, 82–0230
- patented compound for enhancement of organoleptic qualities, 82–0234
- sweet proteins as enhancers, 82–0237
- tricyclic lactones as organoleptic agents, 82–0227, 82–0228

TOBACCO INDUSTRY

- Algerian expansion of production and consumption, 82–0244
- alternative crops for farmers, prospects and problems, 82–0241
- American Medical Association, stock liquidation, 82–0206
- British cigarette consumption decline, increased exports, 82–0256
- Brown and Williamson's promotion of ultra-low-tar Barclay, 82–0260
- cigarette consumption trends in 1980, 82–0245
- Delgold variety, agricultural advantages, 82–0006
- domestic tobacco economy, recent changes 82–0203, 82–0252
- expanding exports by program changes and freer trade, 82–0258
- farms and farmers, decreasing numbers, 82–0243
- Federal program, 82–0253, 82–0271
- Federal Trade Commission 1979 report on cigarettes, 82–0251
- flue-cured farm consolidation and mechanization, 82–0250
- foreign market gains and diversification of U.S. tobacco companies, 82–0262
- future of the "big six" American companies, diversification, 82–0268
- global conglomerates, growth and diversification, 82–0248
- government program modification or abolition, proposals, 82–0270
- growth constraints, foreign and domestic, 82–0247
- India, tobacco exports from 1979 to 1980, 82–0272
- Japanese production and sales, 1980, 82–0275
- Korea, growth and outlook, 82–0276
- legal and policy issues for the 1980s, 82–0284
- Lorillard's increasing market share, emphasis on low-tar cigarettes, 82–0266
- low-tar cigarettes' increasing popularity in the United States, 82–0274
- Maxwell 1981 Fact Book, tobacco industry status report, 82–0263
- 120 mm cigarettes, increased sales, 82–0267
- modified cigarettes, proposed regulations, 82–0288
- North Carolina, economic changes and challenges, 82–0292
- North Carolina's farmers, economic alternatives, 82–0255
- Philip Morris, domestic and foreign markets and diversification, 82–0273, 82–0279
- Poland, economic and health consequences, 82–0061
- production and price stabilization attempts, 82–0242
- program organizations, government and private, 82–0249
- R. J. Reynolds Industries, growth and diversification, 82–0277
- Third World countries as new markets, 82–0257
- tobacco acreage allotment system, 82–0261
- tobacco crops for protein vs. smoking, economic factors, 82–0235
- U.S. exports, 1976-1980, 82–0265
- U.S. flue-cured exports decline, 82–0246
- United States declining leaf exports to the United Kingdom, 82–0254
- USDA price-support program, 82–0282
- West German increased cigarette production, 82–0269
- West German tobacco market, 1979, 82–0264
- work force reductions, possible alternatives, 82–0278
- world market competition from developing countries, 82–0259

TOBACCO LEAF COMPONENTS

- acid phosphodiesterases purification and characterization, 82–0023
- amino acid content, light and temperature effects, 82–0009
- breeding of low-tar varieties, 82–0001
- calcium fertilization effects on content, 82–0008

  extractable protein yield from plant varieties, harvesting effects, 82–0010
  fertilizer and genotype influences, 82–0007
  Fifth Workshop Conference Proceedings, papers, 82–0058
  genetic breeding, improved quality and reduced health risks, 82–0002
  genetic breeding to alter chemical constituents, 82–0005
  infrared reflectance spectroscopy for constituent analysis, 82–0028
  maleic hydrazide residues in Kentucky crops, 82–0004
  plant growth and air curing effects on midvein alcohols, 82–0012
  stalk position and cultivar differences in chemical constituents, 82–0031
  Tobacco and Health Research Institute programs, University of Kentucky, 82–0294
  variation in constituent levels from curing and culturing, 82–0015

TOBACCO PROCESSING
  expansion process with hot inert solid particles, 82–0229
  means of reducing toxicity of cigarette smoke, 82–0291
  reconstituted tobacco by papermaking process, 82–0232

TOBACCO PYROLYSIS PRODUCTS
  toxicity, 82–0037

TOBACCO, RECONSTITUTED
  deproteinized byproducts to lower tar and nicotine content, economic effects, 82–0235
  lower tar and nicotine levels, 82–0226
  papermaking process for production, 82–0232

TOBACCO SMOKE
  aryl nitrile isolation and characterization from condensate, 82–0014
  Fifth Workshop Conference Proceedings, papers, 82–0058
  gas chromatography analysis of constituents, 82–0025
  genetic breeding to alter chemical constituents, 82–0005
  marihuana smoking, health hazard comparison, 82–0155
  phenol content determination by liquid chromatography, 82–0021
  pulmonary biotransformation inhibitor characteristics, 82–0013
  tumorigenicity of constituents of smoke condensate, 82–0081

TOBACCO SUBSTITUTES
  less hazardous cigarettes relationship, 82–0291
  smokable product of cellulose and combustion-modifying substances, 82–0230

TOBACCO VARIETIES
  Delgold, agricultural advantages, 82–0006
  extractable protein yield, harvesting effects, 82–0010
  genetic breeding for improved quality and reduced health risks, 82–0002
  genetic breeding to alter chemical constituents, 82–0005
  low-tar, plant development, 82–0001
  stalk position and cultivar differences in chemical constituents, 82–0031

TRACHEAL MUCOSA
  submucosal glands in rats, smoke exposure effects, 82–0103

UNITED KINGDOM
  American flue-cured tobacco, economic factors in reduced imports, 82–0246
  antismoking messages financed by tobacco advertisement taxes, proposal, 82–0198
  consumption patterns, 82–0180, 82–0256
  mortality in coal miners, dust and smoking as factors, 82–0071
  tar, nicotine, and carbon monoxide yields of cigarettes, 82–0030
  tax increases on tobacco products, 82–0254

UNITED STATES
  tobacco exports, 1976-1980, 82–0265

UROGENITAL NEOPLASMS
  epidemiological studies, smoking as risk factor, 82–0077
  Raynaud's phenomenon, promotion by smoking and chemotherapy, 82–0136

UTERINE NEOPLASMS
  sexual behavior of husbands and smoking as factors in cervical cancer, 82–0064

VASCULAR DISEASES
  aortic aneurysm rates among smokers vs. nonsmokers, 82–0060
  coronary risk factors, smoking effects, 82–0121
  Raynaud's phenomenon, promotion by smoking and chemotherapy, 82–0136
  reconstructive arterial surgery, smoking-related risk, 82–0118
  reduction, cessation, or brand switching as therapy, 82–0186
  risk factors, smoking, 82–0116, 82–0135
  trace metals level as risk factor, 82–0126

WITHDRAWAL CLINICS
  hypnotherapy, effectiveness of Damon Smoking Control program, 82–0220
  SOS Stop Smoking program, education and reinforcement, 82–0202

WOMEN
- lung and bladder cancer mortality attributable to smoking, 82–0065
- plasma levels of oral contraceptive steroids, smoking effects, 82–0036
- sales and popularity of 120 mm cigarettes, 82–0267
- sexual behavior of husbands and smoking as factors in cervical cancer, 82–0064
- thrombosis risk factors, oral contraceptives and smoking, 82–0115

*U.S. GOVERNMENT PRINTING OFFICE : 1982 0-361-166/6394

SMOKING AND HEALTH LITERATURE SEARCH REQUEST

Individual requesting search	Title or position
Individual other than requester completing form	Telephone number
Organization	Date of request
Address including zip code	**Purpose of search** ☐ Research ☐ Paper presentation ☐ Writing article ☐ Lecture ☐ Other ______

SEARCH REQUIREMENTS Give below a detailed statement of information requirements together with any necessary background data to meet your needs. Be as specific as possible to help formulate search strategy.

If topic has been reviewed or cited in past issues of the *Smoking and Health Bulletin* or *Bibliography on Smoking and Health* give pertinent accession number(s) or citation of articles.

Give below other articles which may serve as a guide on subject area (list full bibliographic citation if known)

List relevant key words or index terms appropriate to search.

If a search has been performed on subject utilizing other data sources, please list sources, results of search and search strategy if known.

SEARCH RESTRICTIONS Check appropriate search out-put desired.

☐ Full range (no restrictions)

☐ Year(s) ______________ only

☐ Citation only (no abstract)

☐ English language only

☐ Other language ______________________________

____

AGENCY USE ONLY. Do Not Write Below This Line.

Search statement or criteria	Date search received Date search released
	Number of documents retrieved

NOTICE

If you no longer need this publication, check here ____, return this sheet to the address below, and your name will be dropped from the mailing list.

If you have a change in name or address, please write the change on this sheet and return it to the address below.

WHEN REQUESTING A CHANGE PLEASE BE SURE YOUR FORMER LISTING ALSO APPEARS ON THIS SHEET.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health
Rockville, Maryland 20857

U.S. MAIL

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS
BOOK

MAY 7 1982
504-A
HE20.7211:982/2

COMPLETED
109

MARCH-APRIL 1982

Abstracts 82-0297-82-0606

AL + O
Cantin

Smoking and Health Bulletin

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

I

INTRODUCTION

All items appearing in the Smoking and Health Bulletin become part of the permanent document collection of the Technical Information Center (TIC) of the Office on Smoking and Health and can be referred to by their accession number.

In the past, at the end of each calender year, it has been customary to publish the Bibliography on Smoking and Health which contained a cumulative author and subject index based on the individual Bulletins issued that year. However, this year, due to fiscal and other constraints, it may not be possible to issue the index in the form of the Bibliography . All readers are therefore advised to keep the individual Bulletin issues for both 1981 and 1982 until further notice.

The TIC offers bibliographic search and retrieval services for information on smoking, tobacco, and tobacco use through its Automated Search and Retrieval System. Readers are encouraged to submit searches on the search request form found at the end of each Bulletin issue. Multiple copies of this form can be ordered from the TIC. Currently the automated file contains approximately 25,000 records, the majority of which were published and processed into the TIC collection since 1970. The TIC collection totals more than 35,000 items.

Bibliographic and reference services continue to be offered. Readers can take advantage of these services by writing or calling the TIC at the number listed below. Staff limitations currently exists for photocopying services, therefore your cooperation is sought when requesting such services. Until further notice no more than three items per Bulletin per request can be provided. Requests for services above this three document limit cannot be honored. In order that we may continue to provide photocopying services to the largest number of readers, we ask before requesting such items from the TIC collection, to first check your local resources regarding their availability. Visitors may use the TIC library during regular officer hours, 8:30 a.m. to 5:00 p.m. weekdays. Advance arrrangements of any planned visits are suggested.

We welcome suggestions or comments from our readership regarding our publications and services.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Telephone: 301-443-1690

ANNOUNCEMENT

The Office on Smoking and Health (OSH) has received numerous requests for the 1979 Report entitled *Smoking and Health - A Report of the Surgeon General*. Because of the great demand for this report, the OSH and Government Printing Office stock have been depleted.

The 1979 Report is now available from the U.S. Department of Commerce, National Technical Information Service, Springfield, Virginia 22161. It has been assigned NTIS Accession No. PB82-141300 and may be purchased in two forms: paper copy for $76.50 or microfiche for $11.50. When ordering, give accession number and price.

ISSN: 0081-0363
MARCH-APRIL 1982
Abstracts 82-0297-82-0606

Smoking and Health Bulletin

CONTENTS

AGRICULTURE 75
CHEMISTRY 75
PHARMACOLOGY AND TOXICOLOGY 81
MORTALITY AND MORBIDITY 90
NEOPLASTIC DISEASES 91
NON-NEOPLASTIC RESPIRATORY DISEASES 100
CARDIOVASCULAR DISEASES 108
PREGNANCY AND INFANT HEALTH 118
OTHER DISEASES AND CONDITIONS 121
BEHAVIORAL AND PSYCHOLOGICAL ASPECTS 124
SMOKING PREVENTION AND INTERVENTION 135
SMOKING CESSATION METHODS 139
TOBACCO PRODUCT ADDITIVES 141
TOBACCO MANUFACTURING AND PROCESSING 141
TOBACCO ECONOMICS 143
LEGISLATION 147
GENERAL 148
AUTHOR AND ORGANIZATIONAL INDEX i
SUBJECT INDEX vii

IV

AGRICULTURE

82-0297. Prasad Rao, J.A.V.; Gopalachari, N. C.; Satyanarayana Murty, B.V.V. **Effect of Crop Rotation on the Polyphenolic Constituents of Flue-Cured Tobacco.** *Tobacco Research* 7(2): 185-188, December 1981.

Since a positive correlation has been observed between smoke total particulate matter and levels of total polyphenols in pure, blended, and reconstituted cigarettes, attempts are being made to decrease these harmful leaf constituents by breeding techniques and cultivation practices. Results of a study on the effect of crop rotation on polyphenolic constituents of flue-cured tobacco show that fallow-tobacco grown after fallow-cotton exhibits higher levels of chlorogenic acid, rutin, and total polyphenols; however, tobacco grown after greengram resulted in lower levels. Representative leaf samples of all grades and primings were collected from a 2-year four-crop rotation experiment during the 1975-1976 and 1976-1977 seasons. The experiment was conducted with 10 crop sequences with twin plots for each sequence in a randomized block design with three replications. Cured samples were powdered and analyzed spectrophotometrically for levels of chlorogenic acid, rutin, and total polyphenols. Complete data for the two trials appear in tabular form.

CHEMISTRY

82-0298. Attiyat, A. S.; Christian, G. D.; Callis, J. B. **Recovery and Fluorometric Measurement of a Polynuclear Aromatic Hydrocarbon From Smoked Cigarettes.** *Microchemical Journal* 26(3): 344-353, September 1981.

To follow the metabolism of a certain polynuclear aromatic hydrocarbon (PAH) in tobacco smoke, a considerable quantity of this compound must be present. This study demonstrated the retention of one polynuclear aromatic hydrocarbon (perylene) in the smoke from a single cigarette spiked with that particular hydrocarbon. The ability of the cigarette filter to retain varying amounts of the hydrocarbon was also studied. The results show that cigarettes can be spiked with relatively large amounts of PAH, and sufficient quantities are retained in the cigarette smoke following pyrolysis (smoking) to be useful for following the fate of the compound in biological systems (e.g., in rats taught to smoke). No search was made for pyrolysis products at this stage, but it is likely that with adequate spiking, these will be produced in sufficient quantities to detect and monitor their biological fate. It was also demonstrated that cigarette filters retain a significant fraction of the polynuclear aromatic hydrocarbon, probably on particulates, depending on the amount present. The efficiency appears to increase with decreasing amounts of PAH. (Auth. Abs. Mod.)

82-0299. Baker, R. R. **Product Formation Mechanisms Inside a Burning Cigarette.** *Progress in Energy and Combustion Science* 7(2): 135-153, April 1981.

A burning cigarette is an oxygen-deficient, hydrogen-rich environment, in which hundreds of chemical constituents are exposed to temperatures of up to 950°C, and where thousands of products are formed, distributed between the gas phase and aerosol particles of the smoke. Mainstream smoke is formed during puffing, and sidestream smoke is formed in the interval between puffs. The location of the burning zones of a cigarette (a heat-producing combustion zone and a pyrolysis/distillation zone where most of the organic smoke products are formed) was established by mapping internal gas concentrations and by measuring changes in internal density and temperature. Carbon monoxide and carbon dioxide, formed by combustion and thermal decomposition, together account for almost 60 percent by weight of mainstream smoke products and may show a similar proportion in sidestream products. During a puff, the carbon oxides acquire over 50 percent of their oxygen from the atmosphere, being rapidly produced by thermal decomposition of tobacco, predominantly between 400°C to 800°C. Carbon monoxide is also apparently produced as a secondary product by the carbonaceous reduction of carbon dioxide. As the ventilation of a cigarette increases, the mainstream carbon monoxide/carbon dioxide delivery ratio decreases systematically, and the concentration of all the mainstream gas components is lowered. The relative proportion of substances which enter sidestream and mainstream smoke depends on the specific processes which help form them. Recent mathematical models of some of the processes contributing to smoke formation have been developed.

82-0300. Bevan, D. R.; Riemer, S. C.; Lakowicz, J. R. **Transfer of Polynuclear Aromatic Hydrocarbons From Particulate Matter to Membranes Measured by Fluorescence Spectroscopy.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 603-614.

Use of fluorescence methodology to quantify the membrane uptake rates of particle-adsorbed polynuclear aromatic hydrocarbons (PAHs) is demonstrated, and the results of the technique on five PAHs are presented. Coinstillation of particles and PAH results in increased tumorigenesis due to particle-enhanced cellular uptake of the PAH. Delivery rate of PAH is a function of both the particle and the PAH-to-particle weight ratio. It is possible to vary the carcinogen, the particle, and the effects of the particle on the rate of carcinogen uptake. In general, particles can bind about 1 percent PAH by weight. At higher levels, the PAH appears to be present in a nonabsorbed microcrystalline state.

82-0301. Bhide, S. V.; Pratap, A. I.; Shivapurkar, N. M.; Sipahimalani, A. T.; Chadha, M. S. **Detection of Nitrosamines in a Commonly Used Chewing Tobacco.** *Food and Cosmetics Toxicology* 19(4): 481-483, August 1981.

Analysis of a popular brand of Indian chewing tobacco (Pandharpuri) for *N*-nitroso compounds was carried out using high-pressure liquid chromatography and thin-layer chromatography techniques. It was demonstrated that this variety of tobacco contained nitrosodimethylamine, nitrosopyrrolidine, and nitrosohydroxyproline, each at levels of about 1-2 μg/g tobacco, as well as some nitrosonornicotine. Fractions containing the nitrosamines were subsequently subjected to analysis by gas-liquid chromatography using a thermal energy analyzer, to confirm the other findings and determine the concentrations involved. The results obtained, the large numbers of tobacco chewers in India, and the ability of these nitrosamines to induce tumors in animals point to the importance of studying nitrosamine levels in the saliva of tobacco chewers and of nonchewers. (Auth. Abs.)

82-0302. Brunnemann, K. D.; Hoffmann, D. **Assessment of the Carcinogenic N-nitrosodiethanolamine in Tobacco Products and Tobacco Smoke.** *Carcinogenesis* 2(11): 1123-1127, 1981.

A gas chromatography—thermal energy analyzer developed for analysis of the carcinogen N-nitrosodiethanolamine (NDELA) found NDELA in tobacco and smoke derived from tobacco treated with the sucker growth inhibitor maleic hydrazide-diethanolamine (MH-DELA). Experimental cigarettes made from tobaccos which were treated with MH-DELA contained 115-420 ppb of NDELA and their smoke contained 20-290 ng/cigarette, whereas hand-suckered tobacco and its smoke were free of NDELA. The tobacco of U.S. smoking products contained 115-420 ppb of NDELA and the mainstream smoke from such products yielded 10-68 ng/cigar or cigarette. NDELA levels in chewing tobacco ranged from 220-280 ppb and in two commercial snuff products were 3,200 and 6,800 ppb. Although the five analyzed MH-DELA preparations contained between 0.6-1.9 ppm NDELA it is evident that the major portion of NDELA in tobacco is formed from the DELA residue during the tobacco processing. Based on bioassay data from various laboratories which have shown that NDELA is a relatively strong carcinogen and based on the results of this study, the use of MH-DELA for the cultivation of tobacco is questioned. (Auth. Abs. Mod.)

82-0303. Bush, L. P. **Physiology and Biosynthesis of Tobacco Alkaloids.** In: *Tobacco Leaf Chemistry: Its Origin, Understanding and Current Trends.* Recent Advances in Tobacco Science, Volume 7. Proceedings of the 35th Tobacco Chemists Research Conference, Winston-Salem, North Carolina, October 6-9, 1981. 1981, pp. 75-106.

The physiologic and genetic control of nicotine levels in the leaf tissue of *Nicotiana tabacum* L. and on the formation of the tobacco alkaloids nicotine, nornicotine, anatabine, anabasine, and myosmine is reviewed. The precursors for the alkaloids have been determined, and biosynthetic schemes have been developed that are consistent with the data; but many of the intermediates remain unknown and many of the enzymes uncharacterized. Rates of growth and development of the plant alter nicotine accumulation and enzyme activities, but the regulation of alkaloid synthesis and/or accumulation is not understood. Analysis of the dominant and recessive alleles at the two principal loci controlling alkaloid accumulation indicates that the dominant alleles at both loci are associated with higher levels of total nitrogen and total volatile nitrogenous bases, but lower levels of protein nitrogen, than the recessive alleles. Nicotine accumulation in callus or suspension cultures of *Nicotiana* species seems to be variable and influenced by genetic predisposition, nature, and rate of growth, lighting, and growth regulator combinations. Cultures with either high or negligible nicotine concentrations are obtained by proper selection of cell strain and growth condition. In vitro cultures have been used to study nicotine biosynthesis and accumulation in tobacco because of the ease of manipulating growth conditions. However, not all enzymes detected in the plant have been found in cultures, and nicotine to nornicotine conversion does not occur readily. Consequently, conclusions on alkaloid biosynthesis based on results obtained with in vitro cultures must be accepted with caution. (Auth. Abs. Mod.)

82-0304. Croteau, R. (Editor). **Fragrance and Flavor Substances.** Proceedings of the Second International Haarmann and Reimer Symposium on Fragrance and Flavor Substances (New Products, Processes and Aspects of Product Safety). New York City, September 24-25, 1979. Pattensen, West Germany, D and PS Verlag, 1980, 200 pp.

The topic of product safety reflects a growing concern with new products and their applications. In this symposium, the latest advances in the preparative chemistry and the odor characteristics of a wide range of compounds are reviewed. Safety considerations in the broad sense are discussed, and recent work on the mechanisms of cutaneous toxicity is described. Included also is a review of terpene biosynthesis which complements synthetic studies in this area and underscores the larger point that fundamental biochemical processes are the basis of both odor and taste perception as well as toxicity considerations. Three final, nontechnical chapters cover the aspects of the U.S. Food and Drug Administration's regulatory activities.

82-0305. Cucco, J. A.; Brown, P. R. **Confirming the Presence of N-Nitrosamines in Ambient Air and Cigarette Smoke by Converting to and Photochemically Altering Their Corresponding N-Nitroamines.** *Journal of Chromatography* 213(2): 253-263, August 28, 1981.

One of the most sensitive analytical procedures available for the detection of N-nitrosamines is described, along with results from the analyses of ambient air and cigarette smoke samples. The procedure has been expanded to make the technique more selective. This procedure involves oxidizing the nitrosamines to their corresponding nitroamines, with subsequent detection of picogram quantities by gas chromatography with an electron-capture detector. The presence of the nitroamines is then confirmed by photochemically altering them using a UV lamp and observing if the resulting decrease in peak height with time of irradiation obeys rate expressions derived by using reference nitroamines. Ambient air samples were taken in Rhode Island, four at a site in Providence and four at a rural site in West Greenwich. Only one of the samples contained trace levels of both dimethyl- and diethylnitrosamine. Cigarette smoke samples were collected by smoking four cigarettes, two at a time. Each cigarette was puffed once every 30 seconds. Dimethylnitrosamine was found in the extract from the smoke of four American brands of cigarettes. The analytical procedure's lower detection limit for dimethyl- and diethylnitrosamine is about 0.05 ppb. (Auth. Abs. Mod.)

82-0306. Davis, D. L.; Litzinger, E. F.; Mattina, C. F. (Editors). **Tobacco Leaf Chemistry: Its Origin, Understanding and Current Trends.** Recent Advances in Tobacco Science, Volume 7. Proceedings of the 35th Tobacco Chemists Research Conference, Winston-Salem, North Carolina, October 6-9, 1981. 1981, 187 pp.

A brief introduction, six papers, and a symposium summary are included in this conference report dealing with tobacco leaf chemistry. Topics covered include: (1) the history of tobacco leaf, from its earliest use among Indian peoples in the New World; (2) the chemical and physical criteria of tobacco leaf in today's cigarettes and a review of trends in the usage of tobacco types; (3) the chemical changes that take place during senescence and curing; (4) tobacco alkaloids and their physiology and biosynthesis, including information about the genetic control of tobacco alkaloid levels; (5) the relationship of tobacco leaf precursors to smoke constituents and cigarette flavor; and (6) the molecular biology and genetic manipulation of tobacco leaf protein.

82-0307. Edreva, A.; Hadzhiyska, E. **Prilozhenie na Dvumernata Tunkosloyna Khromatografiya za Razdelyane na Polifenolnite Suedineniya pri Tyutyuna. [Application of Two-Dimensional Thin-Layer Chromatography for Separation of the Polyphenol Compounds From Tobacco.]** *Bulgarski Tyutyun* 25(11): 22-25, 1980, Bulgarian.

A thin-layer chromatographic (TLC) method is described for separation and identification of some 16 polyphenolic compounds contained in tobacco leaves. The method was developed using tobacco leaves of the 163 Kharmanliyska Basma variety and a synthetic mixture of the main polyphenolic components of tobacco. The best separation of these components was obtained with the MN 300 (10 μ) cellulose powder as sorbent and polar solvent mixtures of n-butanol-ethanol-water and acetic acid-water. Polyphenols are extracted from tobacco leaves with ethanol and their solution in ethanol deposited on chromatoplate in a 0.25 mm thick layer of sorbent. Chromatograms are examined in UV light and ammonia vapors under conditions of constant temperature and adequate atmospheric saturation. Three isomers of chlorogenic acid, rutin, scopolin, scopoletin, esculetin glucoside, and quercetin glucoside are some of the polyphenolic compounds identified by the described method. A perfect separation was achieved in a much shorter time than by paper chromatography.

82-0308. Ekstrom, G.; Gustavsson, H. **Nikotin i Modersmjolk Fran Rokande Modrar. [Nicotine in Mother's Milk From Mothers Who Smoke.]** *Var Foda* 32(9): 447-457, 1980, Swedish.

Twenty-five nursing mothers with different smoking habits (0-30 cigarettes/day) were chosen for the study. The mother's milk samples were taken in the homes, refrigerated, and transported to the National Food Administration for analysis. In no case could nicotine be detected in milk from nonsmoking mothers. The highest nicotine concentration found was 95 nanograms per ml milk (about 0.1 ppm) from a woman smoking 20-25 cigarettes/day. No clear-cut correlation between cigarette consumption and nicotine concentration in milk was demonstrable, probably because the sampling moments were chosen at random. Other factors, such as the way of smoking, cigarette brand, and time interval between smoking and sampling/nursing, also affect the nicotine concentration. (Auth. Abs.)

82-0309. Fairweather, F. A.; Carmichael, I. A.; Phillips, G. F.; Copeland, G.K.E. **Changes in the Tar, Nicotine, and Carbon Monoxide Yields of Cigarettes Sold in the United Kingdom.** *Health Trends* 13(3): 77-81, August 1981.

The tar, nicotine, and carbon monoxide yields of most cigarette brands on sale in the United Kingdom are regularly determined by the Government Chemist, and have declined progressively since the early 1960s—those of carbon monoxide more slowly than those of the other two. The incidence of lung cancer, bronchitis, and heart disease is strongly related to cigarette smoking. Evidence suggests that the recent decline in mortality rates from these diseases in men is related to the decline in the amounts of substances yielded by cigarettes. Greater experience of smoking low-tar cigarettes is needed to determine the extent to which the health risks are reduced. It is better not to smoke; but for those unable to stop, and particularly those at greater risk, it would be prudent to smoke only cigarettes in the low-tar band, those being generally lower in carbon monoxide as well. However, the mortality risks for lung cancer, bronchitis, and coronary heart disease, even for low-tar smokers, are greatly in excess of those found among lifelong nonsmokers. Reduction of this toll of sickness and death represents one of the greatest challenges to preventive medicine. (Auth. Abs.)

82–0310. Freeman, A. S.; Martin, B. R. **Quantification of Phencyclidine in Mainstream Smoke and Identification of Phenylcyclohex-1-ene as Pyrolysis Product.** *Journal of Pharmaceutical Sciences* 70(9): 1002-1004, September 1981.

Phencyclidine was recovered and quantified in mainstream smoke; phenylcyclohex-1-ene was identified as a pyrolysis product in prepared parsley cigarettes; tetrahydrocannabinol was recovered and measured from prepared placebo marihuana cigarettes; and nicotine was recovered and measured from prepared tobacco cigarettes. Parsley cigarettes containing (^{3}H)phencyclidine were machine smoked, and the mainstream smoke was trapped in glass wool filters. Radioactivity was extracted from these filters with chloroform. The average recoveries of radioactivity were 76, 85, 70, and 69 percent for cigarettes containing 3, 10, 30, and 50 mg of (^{3}H)phencyclidine hydrochloride, respectively. TLC and GLC-mass spectrometry were employed to identify and quantify compounds in the filter extracts. Approximately one-half of the recovered radioactivity represented a pyrolysis product, phenylcyclohex-1-ene. Formation of this product involved loss of piperidine from phencyclidine. Piperidine, which was not radiolabeled, also may appear in smoke intact. The remainder of the radiolabeled material represented unchanged phencyclidine. Therefore, the percentage of (^{3}H)phencyclidine delivered was about 40 percent of the amount smoked. This result was independent of puff frequency and quantity of phencyclidine hydrochloride smoked over the range tested. The (^{3}H)phencyclidine delivery was compared to the quantities of (^{3}H)-Δ^{9}-tetrahydrocannabinol and (^{3}H)nicotine delivered in mainstream smoke. The recovery of unchanged (^{3}H)-Δ^{9}-tetrahydrocannabinol from placebo marihuana cigarettes injected with a solution containing 3 mg of Δ^{9}-tetrahydrocannabinol was 60 percent. Tobacco cigarettes injected with (^{3}H)nicotine yielded 70 percent unchanged nicotine in mainstream smoke. (Auth. Abs. Mod.)

82–0311. Hecht, S. S.; Chen, C.-H.B.; Young, R.; Hoffmann, D. **Mass Spectra of Tobacco Alkaloid-Derived Nitrosamines, Their Metabolites, and Related Compounds.** *Beitraege zur Tabakforschung International* 11(2): 57-66, September 1981.

Mass spectra are presented for the tobacco alkaloid-derived nitrosamines *N'*-nitrosonornicotine (NNN), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), 4-(methylnitrosamino)-4-(3-pyridyl)butanal, *N'*-nitrosoanabasine, and *N*-nitrosoanatabine, as well as for known mammalian metabolites of NNN and NNK. Also included are the mass spectra of synthetic derivatives related to these nitrosamines and their metabolites. The mass spectral data are expected to be useful in further studies on the occurrence and metabolism of the tobacco alkaloid-derived nitrosamines and in studies of related derivatives of the tobacco alkaloids.

82–0312. Hecht, S. S.; LaVoie, E. J.; Amin, S.; Bedenko, V.; Hoffmann, D. **On the Metabolic Activation of the Benzofluoranthenes.** In: Bjorseth, A.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects.* Columbus, Ohio, Battelle Press, 1980, pp. 417-433.

The benzofluoranthenes, which occur in cigarette smoke, are carcinogenic in experimental animals. Benzo(j)fluoranthene, benzo(b)fluoranthene, and benzo(k)fluoranthene are mutagenic toward *S. typhimurium* TA 100, with activation. Benzo(j)fluoranthene and benzo(b)fluoranthene are carcinogenic when applied to mouse skin. This group was less powerful than benzo(a)pyrene as carcinogens on mouse skin, though levels in the environment are comparable to and sometimes exceed those of benzo(a)pyrene.

82–0313. Heckman, R. A.; Dube, M. F.; Lynn, D.; Rivers, J. M. **The Role of Tobacco Leaf Precursors in Cigarette Flavor.** In: *Tobacco Leaf Chemistry: Its Origin, Understanding and Current Trends.* Recent Advances in Tobacco Science, Volume 7. Proceedings of the 35th Tobacco Chemists Research Conference, Winston-Salem, North Carolina, October 6-9, 1981. 1981, pp. 107-153.

Tobacco smoke components which arise chiefly by pyrolysis of nonvolatile leaf precursors and which are likely to affect flavor are discussed. It is estimated that 3,675 constituents have been isolated from smoke, 2,481 from leaf, with 1,550 common to both. Chromatographic comparisons of leaf and smoke volatiles are given, providing visual evidence of the increased complexity of smoke composition over that of tobacco. The contribution of various tobacco leaf precursors to the number of volatile smoke components is discussed. Nitrogen and sulfur smoke components are mentioned, focusing on the probable origin and potential flavor effects of pyrazines, pyrroles, pyridines, and heterocyclic sulfur compounds. The oxygenated precursors in tobacco which influence the development of smoke flavor are examined, including nonvolatile hydrophilic precursors such as sugar esters, glycosides, and sugar-amine reaction products or Amadori compounds, as well as an insoluble oxygenated precursor, lignin. Although investigations have been useful in identifying probable flavor precursor situations, it is concluded that putting the information into practice appears to be at a less advanced stage.

82–0314. Hoffmann, D.; Adams, J. D.; Piade, J. J.; Hecht, S. S. **Chemical Studies on Tobacco Smoke LXVIII. Analysis of Volatile and Tobacco-Specific Nitrosamines in Tobacco Products.** *IARC Scientific Publication* 31: 507-516, 1980.

Recent progress in the analysis of *N*-nitrosamines in tobacco products is reviewed. Tobaccos which are low in protein and nitrate produce relatively low yields of volatile *N*-nitrosamines (VNA) when smoked as cigarettes. In cigars, the porosity of the cigar wrapper appears to be a more important factor influencing the smoke yield of VNA. Selective filtration and porous wrappers remain the major viable methods for selectively reducing VNA levels in mainstream smoke. Cellulose acetate filter tips can selectively remove 70 percent or more of the VNA from cigarette smoke. Three nonvolatile, tobacco-specific *N*-nitrosamines (NVNA) have been found in tobacco and tobacco smoke: *N*-nitrosonornicotine (NNN) formed by the nitrosation of nicotine and of nornicotine; 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK) formed from oxidative nitrosation of nicotine; and *N*-nitrosoanatabine (NAT) formed by the nitrosation of anatabine. Factors affecting the quantity of NVNA in smoke include the presence of nitrate, nitrite, tobacco alkaloids, NNN, NNK, and NAT in the tobacco, as well as the smoke pH. Cellulose acetate filter tips may selectively filter much of the NVNA from smoke.

82–0315. Iwainsky, H.; Winsel, K. **Schadstoffe im Tabak und Tabakrauch. [Damage Factors in Tobacco and Tobacco Smoke.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 157(1): 90-102, April 1981, German.

In the last 30 years the production and consumption of cigarettes have constantly increased, to the extent that more than 2,200 noxious substances have been detected in the smoke of cigarettes. The origin and the synthesis of selected groups of these compounds and their distribution in mainstream and sidestream smoke are described; and the possibilities for estimation and control of nicotine abuse are reviewed. The noxious substances of tobacco smoke result in damage of clearance mechanisms of the lung and a change of loading by drugs and other metabolizable noxious substances, because the biotransformation processes are influenced by tobacco smoke. In this connection, the importance of aryl-hydrocarbon-hydroxylase is also discussed. (Auth. Abs.)

82–0316. Kung, S. D.; Tso, T. C. **Tobacco Leaf Protein: Molecular Biology and Genetic Manipulation.** In: *Tobacco Leaf Chemistry: Its Origin, Understanding and Current Trends.* Recent Advances in Tobacco Science, Volume 7. Proceedings of the 35th Tobacco Chemists Research Conference, Winston-Salem, North Carolina, October 6-9, 1981. 1981, pp. 154-177.

Genetic engineering of tobacco fraction 1 (F1) protein for the purpose of obtaining better quality, higher yield, and even a safer tobacco product as smoking material, through recombinant DNA technology in which genes coding is used, is discussed. F1 protein is the most abundant soluble leaf protein in tobacco and in other higher green plants, and much is known about its properties. Little is known about the remaining smaller soluble proteins, the fraction II protein. In tobacco, F1 protein accounts for more than 50 percent of the soluble leaf protein. This protein is an important photosynthetic enzyme, ribulose 1,5-bisphosphate carboxylase-oxygenase (RuBPCase), and is very unique in many aspects: (1) it catalyzes two reactions, photosynthesis and photorespiration; (2) it consists of two types of subunits, large and small; and (3) it requires two separate genomes for its synthesis, chloroplast and nuclear. Tobacco F1 protein can be crystallized by a simple procedure with high yield. The procedure involves breaking chloroplasts in high salt concentration to release the protein, heating the crude filtrate to precipitate undesirable material, and removing the salt by gel filtration to yield crystalline protein. The pure enzyme consists of eight large and eight small subunits. Each large subunit (LS) contains separate sites for activation and catalysis, whereas the precise functions of the small subunit (SS) are still unknown. The LS is coded for by chloroplast genomes and synthesized on 70S ribosomes. SS is synthesized as a large precursor which is subsequently transported and processed before it is united with the LS to form a functional enzyme. Generally, the composition of large and small subunits can be genetically manipulated in such a way that enzymes consisting of identical LS and different SS, or vice versa, can be constructed. Thus, using the recombinant DNA technology, genes coding for the LS and SS can be isolated, cloned, and expressed in vivo. (Auth. Abs. Mod.)

82–0317. Kuwata, K.; Uebori, M.; Yamazaki, Y. **Reversed-Phase Liquid Chromatographic Determination of Phenols in Auto Exhaust and Tobacco Smoke as *p*-Nitrobenzeneazophenol Derivatives.** *Analytical Chemistry* 53: 1531-1534, August 1981.

Traces of phenols in auto exhaust and tobacco smoke were collected by using a fritted bubbler with 10 ml of 0.12 percent sodium hydroxide solution and determined by reversed-phase high-performance liquid chromatography via derivatization with *p*-nitrobenzenediazonium tetrafluoroborate in aqueous medium at pH 11.5. The analytical columns used (20 cm x 4.6 mm i.d.) were packed with LiChrosorb RP-18 (5 μm) and with Polygosil 60-5C_{18}, and the mobile phase was 85 percent methanol/15 percent water. Excellent separation of a number of the *p*-nitrobenzeneazo derivatives of water-soluble phenols was obtained. The detection limits of the phenols were 0.05-2.0 ng. Phenol vapors in synthetic air samples, except for *p*-chlorophenol, could be determined in the low parts-per-million or parts-per-billion levels with less than 6.5 percent of relative standard deviations. (Auth. Abs.)

82–0318. Long, R. C.; Weybrew, J. A. **Major Chemical Changes During Senescence and Curing.** In: *Tobacco Leaf Chemistry: Its Origin, Understanding and Current Trends.* Recent Advances in Tobacco Science, Volume 7. Proceedings of the 35th Tobacco Chemists Research Conference, Winston-Salem, North Carolina, October 6-9, 1981. 1981, pp. 40-74.

Many chemical transformations take place in tobacco during senescence and curing, and the complexity of the biological system makes it difficult to ascertain the onset of senescence in the tobacco plant. Unlike the climacteric in fruit, the shift in metabolic activity in tobacco leaves during growth, maturation, and senescence is gradual. Expansive growth ceases coincident with declining nitrogen uptake and reduction, and the onset of starch accumulation after about 7 to 9 weeks growth. The subsequent senescence of physiologically mature leaves is characterized visually by degradation of chlorophyll and carotenoid pigments, the hydrolytic products of which contribute to the flavor of the tobacco. Those pigment losses are accompanied by degradation of starch and chloroplastic proteins (principally ribulose bisphosphate carboxylase). Chemical transformations during curing are basically extensions of those occurring during senescence but proceed at accelerated rates during the yellowing phase. Certain amino acids accumulate and others diminish. Simple sugars accumulate but may be consumed by respiration depending upon the time of termination of biological activity. Leaf yellowing agents and accelerated curing schedules both influence these transformations. It is suggested that tobacco quality in the future will be described in terms of constituents in addition to the amounts of nicotine, sugar, and total nitrogen. (Auth. Abs. Mod.)

82-0319. Muramatsu, M.; Umemura, S. **Kinetics of Oxidation of Tobacco Char.** *Beitraege zur Tabakforschung International* 11(2): 79-86, September 1981.

The kinetic parameters for the oxidation of the tobacco char or carbonaceous residue left after the pyrolysis of tobacco shreds were studied, since such parameters, in addition to those for tobacco pyrolysis, might be useful in examining the smoldering mechanism of cigarettes. Kinetic parameters for tobacco char oxidation can include reaction order, pre-exponential factor, and activation energy. Analysis was carried out by thermogravimetry, derivative thermogravimetry, differential scanning calorimetry, and isothermal weight change measurement of the tobacco char. Data results indicated that tobacco char oxidation apparently consisted of two independent steps. In step I, a noticeable weight loss and an evolution of heat: began at about 200°C; greatly increased in the range of 320°C to 400°C; and peaked at 370°C. In step II, weight loss occurred rapidly in the range of 410°C to 460°C, with a peak at about 445°C. A relatively slow decrease in weight followed, finally leaving ash above 500°C.

82-0320. Pung, T. C.; Chou, C. C.; Tsai, H. T.; Wu, S. C. **An Internal Standard Method for the Determination of Trace Elements in Cigarette Tobacco by Neutron Activation Analysis.** *Journal of Radioanalytical Chemistry* 63(2): 253-262, 1981.

A neutron activation analysis procedure was developed which was able to measure simultaneously the trace elements lanthanum, antimony, scandium, chromium, iron, and cobalt in cigarette tobacco. Gold was used as an internal standard element. The procedure was tested on cigarette samples. The resulting data tended to be of the same magnitude as values found in other brands by other studies. The accuracy of the procedure's determinations was checked by analyzing the standard reference tobacco 1R1, the results showing fairly good agreement with literature values.

82-0321. Schoeny, R.; Warshawsky, D.; Hund, M.; Nagel, B.; Smiddy, B. A.; Warren, C. **Mutagenicity of Benzo(*a*)pyrene Metabolites Generated on the Isolated Perfused Lung.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 475-485.

Macrophage extracts from isolated perfused lungs treated with benzo(*a*)pyrene (BaP) were found to be mutagenic in salmonella. The extracts were more often mutagenic in strain TA 1538 than TA 100, and activity was largely dependent on additional metabolic activation by rat hepatic extracts. With minor exceptions, extracted material from blood perfusate aliquots was not mutagenic. The distribution of BaP metabolites differed according to administration: intratracheal routes produced both phenols and 9,10-diols, whereas intravenous routes resulted in decreasing diols and increasing phenols. Three particle types, fly ash, crude air particulate (CAP), and Fe_2O_3, were given in conjunction with BaP. Fly ash and CAP showed increased rates of metabolism over Fe_2O_3. Fly ash and Fe_2O_3 facilitated BaP accumulation and lack of metabolism in the macrophage.

82-0322. Severson, R. F.; McDuffie, K. L.; Arrendale, R. F.; Gwynn, G. R.; Chaplin, J. F.; Johnson, A. W. **Rapid Method for the Analysis of Tobacco Nicotine Alkaloids.** *Journal of Chromatography* 211(1): 111-121, June 1981.

A rapid glass capillary gas chromatographic (GC) method for the analysis of the nicotine alkaloids of tobacco, including nicotine, nornicotine, myosmine, anabasine, anatabine and 2,3'-dipyridyl, is described which may be useful for the evaluation of various tobaccos for quality and acceptability, as well as for the analysis of volatile alkaloids in other plant extracts or organic mixtures. The use of glass capillary GC methodology, coupled with nitrogen-specific detection, provided rapid, unambiguous alkaloid analysis. About 25 mg of cured tobacco and 1 ml of 0.05 *M* methanolic potassium hydroxide were placed in a standard 2-ml autosampler vial, and the vial was capped and placed in an ultrasonic bath for 15 minutes. A 1-µl volume was analyzed by glass capillary GC operating in the split-injection mode. The analysis procedure for nicotine alkaloids was considered simple and rapid, allowing a trained technician using one automated glass capillary GC system to analyze about 45 samples/day. (Auth. Abs. Mod.)

82-0323. Snook, M. E.; Fortson, P. J.; Chortyk, O. T. **Isolation and Identification of Aza-Arenes of Tobacco Smoke.** *Beitraege zur Tabakforschung International* 11(2): 67-78, September 1981.

The nitrogen analogs of polynuclear aromatic hydrocarbons (aza-arenes) were isolated and identified in a basic fraction of cigarette smoke condensate. Silicic acid chromatography removed the predominant nicotine alkaloids, while gel chromatography on Bio-Beads S-X12 in benzene effectively separated the aza-arenes from interfering aliphatic compounds. In addition, the gel columns separated the aza-arenes by ring number and degree of alkylation on the basis of an adsorption-type mechanism. These gel characteristics facilitated the identifications of a large number of isomeric aza-arenes. Compounds identified included 2-vinylpyridine, 3-vinylpyridine, and 2-phenylpyridine as well as quinoline, isoquinoline, 4-azafluorene, benzoquinolines, benzoisoquinolines, 1-azafluoranthene, 7-azafluoranthene, 4-azapyrene, 7-azaindole, pyrroloquinoline, and their mono-, di-, and trimethyl derivatives. All eight possible isomers of benzoquinoline and benzoisoquinoline were found, four of which are being reported for the first time. Evidence was also found for the probable presence of 5,6-benzo-7-azaindole. (Auth. Abs.)

82–0324. Spears, A. W.; Jones, S. T. **Chemical and Physical Criteria for Tobacco Leaf of Modern Day Cigarettes.** In: *Tobacco Leaf Chemistry: Its Origin, Understanding and Current Trends* , Recent Advances in Tobacco Science, Volume 7, Proceedings of the 35th Tobacco Chemists Research Conference, Winston-Salem, North Carolina, October 6-9, 1981. 1981, pp. 19-39.

Trends in the usage of tobacco types, including reconstituted sheet, are reviewed; and information on their chemical and physical properties is presented. Based on sales weighted nicotine percentages, it is suggested that the lowest tar brands (0-6 mg) indicate an increase in nicotine content over time. Friability of the leaf has become increasingly important as the less labor intensive methods of harvesting and curing produce more nonoriented leaf. Leaf density and burning properties of U.S. leaf are shown to be within satisfactory ranges, while chemical residues are within acceptable ranges with the exception of maleic hydrazide; however, these residues have no impact on organoleptic smoking quality. Individual tobacco constituents, including those components that are generally recognized as important to the organoleptic quality, are considered for their effect on the smoking characteristics of tobacco. Trace components, particularly residues, are identified with respect to their importance to the quality of modern day leaf. It is recommended that such characteristics be considered in future attempts to develop new varieties, cultivation, and curing methods.

82–0325. Young, J. C.; Robinson, J. C.; Rickert, W. S. **A Study of Chemical Deliveries as a Function of Cigarette Butt Length.** *Beitraege zur Tabakforschung International* 11(2): 87-95, September 1981.

The suitability of linear and log linear models to represent the relationship between chemical deliveries (condensate, nicotine, total hydrogen cyanide (HCN), and carbon monoxide (CO)) and butt length was investigated, utilizing two statistically designed experiments to explore these relationships for 10 major commercial brands of cigarettes. There are highly significant differences in slope, intercept, and perhaps even linearity between brand groups and brands within groups. Although several brands seem to be adequately linear on either arithmetic or logarithmic scales, they clearly behave differently from each other. There is also strong evidence that condensate and nicotine are not linear on the same scale, with condensate being approximately linear on the logarithmic scale and nicotine approximately linear on the arithmetic scale. Thus, it is clear that the ranking of brands on the basis of measured value of any parameter will depend on the butt length at which the parameter was measured. Therefore, international comparison of deliveries requires redetermination of deliveries at the new butt length standard rather than mathematical manipulation using a single functional relationship. The form of a suitable functional relationship for HCN cannot be ascertained from the present study and will require additional experimentation. The statistical design and analysis proved worthwhile in model discrimination for the other three parameters. (Auth. Abs. Mod.)

PHARMACOLOGY AND TOXICOLOGY

82–0326. Andersson, K.; Eneroth, P.; Agnati, L. F. **Nicotine-Induced Increases of Noradrenaline Turnover in Discrete Noradrenaline Nerve Terminal Systems of the Hypothalamus and the Median Eminence of the Rat and Their Relationship to Changes in the Secretion of Adenohypophyseal Hormones.** *Acta Physiologica Scandinavica* 113(2): 227-231, October 1981.

The effects of acute single doses (0.3 and 1 mg/kg) of nicotine on various hypothalamic catecholamine nerve terminal systems and on the secretion of adenohypophyseal hormones in the rat were studied. Nicotine, in a dose of 1.0 mg/kg, increased noradrenaline turnover in the median eminence and in the peri- and paraventricular hypothalamic regions. The dopamine and noradrenaline nerve terminal systems in the median eminence and the dorsomedial hypothalamic nucleus respectively were unaffected. Serum gonadotropic hormone (GH) levels were decreased and serum prolactin levels increased after a dose of 1 mg/kg. In the presence of tyrosine hydroxylase inhibition, nicotine in a dose of 1 mg/kg, instead increased GH and also luteinizing hormone (LH) secretion. It is suggested that the preferential increases of noradrenaline turnover in various hypothalamic noradrenaline nerve terminal systems by nicotine may be partly responsible for the nicotine induced increases of serum prolactin, GH, and LH levels observed. (Auth. Abs.)

82-0327. Andersson, K.; Fuxe, K.; Agnati, L. F.; Eneroth, P. **Effects of Acute Central and Peripheral Administration of Nicotine on Ascending Dopamine Pathways in the Male Rat Brain. Evidence for Nicotine Induced Increases of Dopamine Turnover in Various Telencephalic Dopamine Nerve Terminal Systems.** *Medical Biology* 59(3): 170-176, June 1981.

Intraventricular injections and intravenous infusions of nicotine appeared to enhance dopamine turnover and reduce dopamine stores in discrete areas of the forebrain of normal male rats, in this study which measured the decline of the dopamine stores after tyrosine hydroxylase inhibition using α-methyltyrosine methyl ester. The dopamine concentrations in the various telencephalic dopamine nerve terminal systems were measured using the Falck-Hillarp methodology involving quantitative microfluorimetry. The catecholamine concentrations in the anteromedial frontal cortex were measured biochemically using high pressure liquid chromatography combined with electrochemical detection. In the intraventricular experiments, the dopamine levels in discrete areas of the nucleus (nuc.) caudatus and nuc. accumbens were significantly reduced even with the lowest dose of nicotine (1 μg/rat). Intraventricular injections of nicotine in a dose of 100 μg/rat produced significant increases of dopamine turnover in various types of dopamine nerve terminal systems in the nuc. caudatus, nuc. accumbens, and tuberculum olfactorium; and following a dose of 10 μg/rat increases of dopamine turnover were observed in the medial part of the nuc. caudatus. Furthermore, nicotine (100 μg/rat) significantly increased noradrenaline but not dopamine turnover within the anterofrontal cortex. In the intravenous experiments, the dopamine levels were selectively reduced by nicotine (1,000 μg/kg) in the cholecytokinin positive and negative dopamine nerve terminal systems of the nuc. accumbens. On the other hand, dopamine levels in the anteromedial frontal cortex were increased after this dose of nicotine. Intravenous infusions of nicotine (10-1,000 μg/kg) produced dose-related increases of dopamine turnover in the various dopamine nerve terminal systems analyzed in the telencephalon. These effects became significant with a dose of 1,000 μg/kg/h. The dopamine terminals in the nuc. caudatus showed a higher sensitivity to intravenous infusions of nicotine, being affected by 10-100 μg/kg of nicotine. These findings suggest that relatively low doses of nicotine via an activation of central nicotine-like cholinergic receptors can reduce dopamine concentration and increase dopamine turnover in discrete limbic and striatal areas. These actions may in part represent the neurochemical basis for the rewarding actions of nicotine and for nicotine dependence in man. (Auth. Abs. Mod.)

82-0328. Bevan, D. R.; Riemer, S. C.; Lakowicz, J. R. **Effects of Particulate Matter on Rates of Membrane Uptake of Polynuclear Aromatic Hydrocarbons.** *Journal of Toxicology and Environmental Health* 8(1/2): 241-250, July-August 1981.

Investigations of the mechanism of particle-enhanced uptake of benzo(*a*)pyrene into model membranes and microsomes have been extended by studying the effects of particulates on the rates of uptake of four additional polynuclear aromatic hydrocarbons (PAH) into model membranes. Adsorption of dibenzo(*a,h*)anthracene, benzo(*g,h,i*)perylene, and 3-methylcholanthrene to the surface of particles resulted in enhanced rates of membrane uptake of these PAHs. Adsorption to the fibrous asbestos minerals (chrysotile and anthophyllite) produced the greatest enhancement in membrane uptake rates compared to uptake from microcrystalline dispersions of the PAH. Adsorption of these PAHs to nonfibrous minerals resulted in uptake rates intermediate between those of the asbestos-adsorbed and microcrystalline states. In contrast, adsorption of dibenzo(*c,g*)carbazole to particles did not result in enhanced uptake. In this case, the rates of uptake in the absence and presence of particles were all extremely high, presumably as a result of the high water solubility of this PAH. The methods described permit characterization of particle-PAH mixtures in terms of the rate of PAH uptake into membranes. (Auth. Abs.)

82-0329. Breckenridge, A. M.; Back, D. J.; Cross, K.; Crawford, F.; MacIver, M.; Orme, M. L'E.; Rowe, P. H.; Smith, E. **Influence of Environmental Chemicals on Drug Therapy in Humans: Studies With Contraceptive Steroids.** In: *Environmental Chemicals, Enzyme Function and Human Disease.* Ciba Foundation Symposium 76 (New Series), 1980, pp. 289-306.

Results of an examination of various environmental factors on the variability in response to oral contraceptive steroid therapy in women showed 10- to 30-fold variations in plasma concentrations of norethisterone, L-norgestrel, and ethinyloestradiol in samples taken 12 hours after administration of oral contraceptives in mid-menstrual cycle. Factors shown to be responsible for this variation include passage into the enterohepatic circulation, a variable first-pass effect, and changes in metabolism in the gut wall or liver due to diet, disease, smoking, or administration of drugs. Smoking was shown to increase the rate of metabolism of drugs such as phenacetin, antipyrine, and theophylline, but not diazepam, phenytoin, or warfarin. Preliminary data suggest that plasma concentrations of ethinyloestradiol are lower among cigarette smokers than in nonsmoking controls. This may be important in the toxicity of contraceptive steroids, since these smokers may have higher plasma concentrations of reactive steroid metabolites. These metabolites have been postulated to be the cause of the excess vascular disease found in women who smoke and take contraceptive steroids.

82-0330. Chakarvarti, S. K.; Lal, N.; Nagpaul, K. K. **Content of Uranium Trace in Urine Determined From Fission Track Etch Technique.** *International Journal of Applied Radiation & Isotopes* 31(12): 793-795, 1980.

Trace concentrations of uranium measured in normal human urine samples from smokers and nonsmokers appeared to indicate that the uranium excretion rate was not dependent on the smoking or nonsmoking habits of the individual. Urine analysis is a common method for measuring internal contamination by radionuclides from natural or man-made sources. The study also evaluated the possibility of examining the intake of uranium from cigarette smoking. Urine samples of male smokers and nonsmokers aged 25-30 years, subjected to the fission track etch technique, showed a heterogeneous distribution of uranium concentration values. Uranium excretion rates did not appear to be determined by smoking or nonsmoking habits, although it was suggested that the data were too meager yet to reach a definite conclusion. It was also considered possible that uranium intake from cigarette smoking was negligible since it is nonvolatile at the 600-800°C temperature of a burning cigarette.

82–0331. Cohen, A. J.; Roe, F.J.C. **Monograph on the Pharmacology and Toxicology of Nicotine.** London, Tobacco Advisory Council Occasional Paper 4, 1981, 45 pp.

Pharmacological and toxicological data concerning nicotine are summarized, concentrating on data from studies on nicotine or its salts rather than studies on tobacco smoke or tobacco-smoke condensate where it is difficult to distinguish the effects of nicotine from those of other constituents. The approach has been to regard nicotine as a "drug" to which man is exposed in various "vehicles" and by various routes, and to delineate the effects of the "drug." Specific topics addressed include chemistry, metabolism, pharmacology, toxicity, carcinogenicity, mutagenicity, reproduction and teratogenicity, the etiological role of nicotine in smoking-associated diseases, and the role of nicotine in the smoking habit. (Auth. Abs. Mod.)

82–0332. Digenis, G. A.; Cheng, Y. C.; McQuinn, R. L.; Freed, B. R.; Tilbury, R. S. **In Vitro and in Vivo Disposition of ^{13}N-Labeled Nitrosoureas and Nitrosocarbamates.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, November 1-2, 1979. 1980, pp. 91-123.

Ureas and carbamates can each react with nitrite during N-nitrosation reactions to form nitrosoureas and nitrosocarbamate derivatives. Nitrosoureas exhibit carcinostatic properties, while nitrosocarbamates have been shown to be mutagenic. This study investigated the in vivo and in vitro disposition of the nitroso function of nitrosoureas and nitrosocarbamates. Millicurie quantities of 1,3-bis(2-chloroethyl)-1-nitrosourea (BCNU) and the nitrosocarbamates nitrosocarbaryl and N-nitroso-N-chloroethyl-1-chloroethylcarbamate (BCNC) were prepared and labeled in the nitroso group with nitrogen-13. Whole body retention of ^{13}N radioactivity in the rat, following IV injection of ^{13}NO-BCNU, was measured by external scintigraphy. Biphasic loss of ^{13}N with a prolonged terminal phase was revealed. In contrast to the retention, loss of ^{13}N from the rat's body was very rapid (more than 90 percent in 10 minutes) after IV injection of ^{13}NO-nitrosocarbaryl and ^{13}NO-BCNC. The rate of elimination of ^{13}N-radioactivity from rat blood in vivo was found to be at least five times slower than the elimination of intact BCNU ($t_{1/2}$ = 21.7 minutes). A pronounced parallelism between the levels of ^{14}C and ^{13}N-radioactivities (obtained after IV administration of ^{14}C and ^{13}N-BCNU) in the vital organs of the rat was observed during 1-hour postinjection period. Approximately 10 percent of the ^{13}N dose appeared in the urine probably as $^{13}NO_3$. The above data suggest that BCNU is metabolized in vivo very rapidly and that its nitroso function may remain covalently linked with its alleged chloroethyl alkylating moiety. Intragastric administration of ^{13}NO-nitrosocarbaryl showed a long retention of ^{13}N-activity in the stomach. Since this nitrosocarbamate was found to be stable at pH 7.4, it is suggested that the compound remains intact in the stomach of the rat. The present work tends to indicate that there might exist important differences in the in vivo metabolic pathways between carcinostatic nitrosoureas and the corresponding nitrosocarbamates which are carcinogenic. (Auth. Abs. Mod.)

82–0333. Dmitriev, M. T. **Kosvennaya Toksichnost' Tabachnogo Dyma pri Passivnom Kurenii. [Indirect Toxicity of Tobacco Smoke in Passive Smoking.]** *Zdravookhranenie Belorussii* (12): 28-31, 1980, Russian.

Both direct and indirect effects of air pollution with tobacco smoke are evaluated in relation to the passive smoker. The total index of air pollution (toxicity) with tobacco smoke is calculated to be 384,000 times the maximum permissible concentration (MPC); that is, 4.25 times the toxicity of car exhaust gases. The indirect effect of tobacco smoke is shown to be the result of a reduction in the index of air freshness, caused by the elimination (discharge) of atmospheric light ions, decomposition of ozone, and, consequently, a decrease in air luminescence intensity. Evolution of the air pollution index, indirect toxicity, and the ratio of indirect to direct toxicity is shown versus the degree of air pollution with tobacco smoke (the number of smoked cigarettes/m+U3 of air). Indirect toxicity accounts for 40 percent of the total effect of tobacco smoke toxicity on the passive smoker. According to Soviet authors, indirect toxicity of tobacco smoke causes ill feeling, asphyxia, fainting spells, and increases the incidence of many diseases in both smokers and passive smokers.

82–0334. Dolara, P.; Mazzoli, S.; Rosi, D.; Buiatti, E.; Baccetti, S.; Turchi, A.; Vannucci, V. **Exposure to Carcinogenic Chemicals and Smoking Increases Urinary Excretion of Mutagens in Humans.** *Journal of Toxicology and Environmental Health* 8(1/2): 95-103, July-August 1981.

Urine samples from a control population and from a population of chemical workers from two chemical plants near Florence, Italy, were analyzed for the presence of mutagenic chemicals by the Salmonella/microsome test. When tested with strain TA 1538, the urine of nonsmoking chemical workers showed higher mutagenic activity than that of controls in the presence of in vitro metabolic activation; but no difference was found between controls and chemical workers who both smoked. Increased mutagenic activity was observed in the group of control smokers compared to control nonsmokers, but the same effect was not observed for chemical workers. When

TA 100 was used as the tester strain, the chemical workers, both smoking and nonsmoking, had significantly higher mutagenic activity than controls. Although some perturbing effects of smoking habits were observed, the results seemed to indicate the usefulness of the Salmonella/microsome test for detection of mutagens in human urine. The results also suggest that people exposed to potentially carcinogenic chemicals may show high enough traces of those chemicals and/or their metabolites in their body fluids to be detected with current mutagenesis techniques. (Auth. Abs.)

82–0335. Eikmann, Th.; Lenaerts, H.; Prajsnar, D.; Einbrodt, H. J. **Phenolausscheidung und Rauchverhalten. [Phenol Excretion and Smoking Habit.]** *Oeffentliche Gesundheitswesen* 43(5): 228-231, 1981, German.

Smoking habits of a representative sample of 254 men and 248 women, aged 65 to 67, were compared with their carboxyhemoglobin (COHb) blood levels and the 24 hr phenol excretion in their urine. The phenol concentration in urine is considered as a factor of the smokers' contamination with benzene from tobacco smoke. Blood was analyzed for COHb by an unpublished gas chromatographic method, using catalytic conversion of CO to CH_4. The average COHb values were found to be closely correlated with the smoking habits of both male and female subjects. The average COHb was 2.24 percent for men vs. 1.15 percent for women, which was in good agreement with the 29 percent vs. about 5 percent of moderate and heavy smokers among men and women respectively. Also, the average COHb value was 0.81-0.82 percent for nonsmokers and ex-smokers, 2.60 percent for light smokers (1 to 10 cigarettes/day), and significantly higher for moderate and heavy smokers of both sexes. In contrast, variations in the average phenol value were not statistically significant either by sex or in relation to the smoking habit of the subjects. Various hypotheses are suggested to explain the discrepancy between the phenol excretion data and benzene concentrations in cigarette smoke.

82–0336. Falkeborn, Y.; Larsson, C.; Nordberg, A. **Chronic Nicotine Exposure in Rat: A Behavioural and Biochemical Study of Tolerance.** *Drug and Alcohol Dependence* 8(1): 51-60, August 1981.

After 31 days of nicotine withdrawal, no tolerance to nicotine was found in a study group of male Sprague Dawley rats. Rats received nicotine (50 mg nicotine base/l) in their drinking fluid for 28 days. The daily consumption of nicotine was about 4 mg/kg per day. Controls received plain water. Body weight, food consumption, and fluid intake were registered during the whole treatment period and up to 31 days after withdrawal of nicotine. A significant lower body weight was noted in rats treated with nicotine, compared with control rats during the whole treatment period. After withdrawal of nicotine the body weight returned to normal. Only slight effects were seen on the food intake (at the beginning of the nicotine treatment). The fluid intake, on the other hand, was significantly lower in the nicotine group during the whole period of treatment and a reduced intake persisted 28 days after withdrawal of nicotine. Tolerance to nicotine was measured in rats 24 hours after withdrawal of nicotine, using two behavioral tests. No tolerance to nicotine was found after 31 days of withdrawal of nicotine. The number of nicotine-like binding sites was found to be reduced in the midbrain 24 hours after withdrawal of nicotine, while no significant change was found in the cortex and hippocampus. (Auth. Abs. Mod.)

82–0337. Gilli, G.; Corrao, G.; Scursatone, E. **In Tema di Prevenzione Dell'Ossicarbonismo: Da una Indagine Epidemiologica ad un Modello Matematico. [Prevention of Carbon Monoxide Poisoning: From Epidemiological Research to Mathematical Model.]** *Giornale Italiano di Medicina de Lavoro* 2(3-4): 139-148, 1980, Italian.

A mathematical model was designed to determine carboxyhemoglobin ratios secondary to cigarette smoke. Subjective parameters (number of cigarettes smoked/day and the time passed from the last cigarette) and objective variables (thiocyanate concentration in blood serum) were considered. Some possible application for diagnostic and preventive medicine is indicated.

82–0338. Grandjean, P.; Olsen, N. B.; Hollnagel, H. **Influence of Smoking and Alcohol Consumption on Blood Lead Levels.** *International Archives of Occupational and Environmental Health* 48(4): 391-397, August 1981.

Blood lead levels were determined in 88 percent of all men and women born in 1936 and residing in four suburbs of Copenhagen, Denmark (504 men and 548 women). Smoking habits and alcohol consumption were assessed by interview and were found to be independent of other indicators of lead exposure. Increased blood lead levels of smokers could, for the most part, be explained by augmented alcohol intake as indicated by the partial correlation coefficient and by the relationship between alcohol consumption and lead levels in nonsmokers. Further, smoking contributed little to the blood lead levels of individuals who did not consume any alcohol. A multivariate analysis indicated that one unit of alcohol (1.35 cl pure ethanol) per day might contribute 0.5-1.0 μg lead/100 ml blood. No significant difference was found between the influence of beer, wine, and hard liquor. (Auth. Abs.)

82–0339. Gregson, R. L.; Prentice, D. E. **Aspects of the Immunotoxicity of Chronic Tobacco Smoke Exposure of the Rat.** *Toxicology* 22: 23-31, 1981.

Extrapolation of results of an immunotoxicity study in rats suggests that cigarette smoke induced immunosuppression may be involved in the etiology and pathogenesis of a number of respiratory diseases. Wistar-strain rats (180) were exposed to differing concentrations of tobacco smoke, for periods of up to 20 months, to examine the response of the pulmonary immune system. The amount of bronchus-associated lymphoid tissue in the lungs of exposed rats increased initially over the first 5 weeks of exposure, subsequently falling to below control levels by the 14th week, and eventually increasing again to a level slightly higher than that of the controls by the 12th month, at which level it was maintained until the 20th month. Quantitative immunohistochemical assay of bronchial immunoglobulin levels (only assessed over the initial 14 weeks of exposure) revealed a transitory enhancement of levels followed by a depression, the speed of response being apparently dose-related. Alveolar macrophage activity, indicated by lysosomal enzyme activity, increased relative to the control animals over the same 14-week exposure period. The significance of these observations is discussed and a tentative explanatory hypothesis is advanced. (Auth. Abs. Mod.)

82-0340. Grygiel, J. J.; Birkett, D. J. **Cigarette Smoking and Theophylline Clearance and Metabolism.** *Clinical Pharmacology and Therapeutics* 30(4): 491-496, October 1981.

Plasma theophylline clearance (Cl_T) was found to be greater in smokers than in nonsmokers. Most theophylline is metabolized in humans, with only 10 percent excreted unchanged in urine. The metabolites include 1-methyluric acid (1MU), 3-methylxanthine (3MX), and 1.3-dimethyluric acid (DMU). An analysis was conducted of plasma and urinary theophylline concentrations and of urinary metabolite concentrations among eight nonsmokers and nine smokers under steady-state oral dosing conditions. The mean Cl_T was 78 percent greater in smokers compared to nonsmokers. Analyses of urinary metabolites revealed that clearance of 1-methyluric acid (Cl_{1MU}) and clearance to 3-methylxanthine (Cl_{3MX}) were increased in smokers 1.99-fold and 2.10-fold over nonsmoking controls ($P<0.005$). Clearance to 1.3-dimethyluric acid (Cl_{DMU}) was also enhanced in smokers 1.68-fold compared to controls ($P<0.01$). The positive relationship between Cl_{1MU} and Cl_{3MX} in both smokers and nonsmokers supports the concept that the N-demethylation pathways for producing 1MU and 3MX are under common regulatory control and involve a form of cytochrome P_{450}distinct from that mediating 8-hydroxylation of theophylline to DMU. These results suggest that cigarette smoking induces both of the cytochrome P_{450} -mediated pathways of theophylline metabolism but that N-demethylation may be increased to a greater extent than 8-hydroxylation. (Auth. Abs. Mod.)

82-0341. Hecht, S. S.; LaVoie, E. J.; Bedenko, V.; Hoffmann, D.; Sardella, D. J.; Boger, E.; Lehr, R. E. **On the Metabolic Activation of Dibenzo(*a,i*)pyrene and Dibenzo(*a,h*)pyrene.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 43-54.

The tumorigenic activity of dibenzo(*a,i*)pyrene (DB(*a,i*)P), and dibenzo(*a,h*)pyrene (DB(*a,h*)P), detected in the neutral fraction of cigarette smoke, is somewhat less than that of benzo(*a*)pyrene on mouse skin. Data on the fluorinated compounds suggest that 3,4-dihydro-3,4-dihydroxy DB(*a,i*)P and 1,2-dihydro-1,2-dihydroxy-DB(*a,h*)P, which can form bay region dihydrodiol epoxides, are proximate carcinogens of DB(*a,i*)P and DB(*a,h*)P. Two dose levels were tested: 100 µg and 500 µg. After 17 weeks of promotion at the higher dose, 2,10-difluoroDB(*a,i*)P was inactive. When judged by the number of tumors per mouse at the higher dose, 2-fluoroDB(*a,i*)P was somewhat less active than DB(*a,i*)P, whereas 3-fluoroDB(*a,i*)P was as tumorigenic as the parent compound. At the lower dose, however, only DB(*a,i*)P showed significant tumorigenic activity. The inactivity of 2,10-difluoroDB(*a,i*)P was also seen in tests using subcutaneous injection in mice, and indicates the involvement of the angular rings in tumorigenesis by DB(*a,i*)P.

82-0342. Hicks, R.; Dysken, M. W.; Davis, J. M.; Lesser, J.; Ripeckyj, A.; Lazarus, L. **The Pharmacokinetics of Psychotropic Medication in the Elderly: A Review.** *Journal of Clinical Psychiatry* 42(10): 374-385, October 1981.

The use of psychotropic medications for the elderly is discussed, including general pharmacokinetic principles and terminology, general principles of geriatric pharmacokinetics, descriptions of specific agents, important drug interactions, and the effects of possible drugs of abuse such as tobacco. Such information is considered important for elderly patients because they frequently use drugs in combinations and are especially sensitive to the side effects of multiple drug usage. Several possible effects of smoking are mentioned. Smoking may antagonize analgesics, especially propoxyphene. It may diminish the pain threshold. There may also be a correlation between genetic disposition and induction by tobacco smoke of certain enzyme systems thought to predispose to cancer. Smoking appears generally to increase the rate of biotransformation. It may also reduce diazepam and chlordiazepoxide drowsiness. It is suggested that geriatric patients be advised not to smoke.

82-0343. Kaszubowski, P.; Wysocki, J.; Machalski, M. **Zachowanie Sie Limfocytow T I ich Subpopulacji Tγ i Tµ U Palaczy Papierosow. [Lymphocytes T and Their Subpopulations Tγ and Tµ in Cigarette Smokers.]** *Polskie Archiwum Medycyny Wewnetrznej* 65(2): 101-105, 1981, Polish.

The percent proportions of lymphocytes T and their subpopulations Tγ and Tµ were compared in cigarette smokers and nonsmokers (27 and 31 subjects respectively, matched for age and of either sex). A statistically significant rise in the number of lymphocytes Tγ ($p<0.01$) was shown in cigarette smokers with a history of smoking exceeding 10 years. On the other hand, no difference was observed in the percent proportions of lymphocytes T and their subpopulation Tµ. The study gives no answer to the question of whether the observed rise in the number of potentially suppressor cells (Tγ) is a primary or secondary phenomenon. (Auth. Abs.)

82-0344. Korte, A.; Wagner, H. M.; Obe, G. **Simultaneous Exposure of Chinese Hamsters to Ethanol and Cigarette Smoke: Cytogenetic Aspects.** *Toxicology* 20(2-3): 237-246, 1981.

High mitotic activity was found in the bone marrow cells of Chinese hamsters exposed to cigarette smoke. Four groups of hamsters were treated with ethanol, cigarette smoke, or both, during the experimental period of 12 weeks: (1) a control group (C) which received water for drinking; (2) a water drinking group (S) which received cigarette smoke treatment during the last 4 weeks for 1 hour per day; (3) a group (E) which received 20 percent (v/v) ethanol for the 12-week period as the only liquid; and (4) a group (ES) with the same ethanol treatment, simultaneously treated with cigarette smoke during the last 4 weeks of the experiment for 1 hour per day. Bone marrow cells showed no effects with regard to chromosomal aberrations and sister chromatid exchanges, but smoke treatment may have been too short to establish a level of mutagens which could have induced such changes. The mitotic indices, however, were significantly larger in groups E, S, and ES compared to group C ($P < 0.001$), with the highest readings among the smoke-exposed groups: 2.7 percent for group C, 4.2 percent for group E, 7.1 percent for group S, and 6.0 percent for group ES.

82-0345. Langenbach, R.; Nesnow, S.; Malick, L.; Gingell, R.; Tompa, A.; Kuszynski, C.; Leavitt, S.; Sasseville, K.; Hyatt, B.; Cudak, C.; Montgomery, L. **Organ Specific Activation of Carcinogenic Polynuclear Aromatic Hydrocarbons in Cell Culture.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 75-84.

Cell-mediated mutagenesis was used to study organ specificity in response to chemical carcinogens. Liver, lung, kidney, and bladder cells from rats were used to metabolically activate benzo(*a*)pyrene (BaP) and 7,12-dimethylbenz(*a*)anthracene (DMBA), both carcinogenic, and anthracene, a noncarcinogen. The relative mutagenicities of BaP and DMBA are expressed as factors of increase over background per 10^6 activating cells, with anthracene assigned a value of one. The mutagenicity of BaP was 2, 56, 5, and 6, in liver, lung, kidney, and bladder cell systems, respectively. DMBA was mutagenic at factors of 14, 154, 12, and 21, respectively. The order of formation of total BaP metabolites produced by the four cell types after 48 hours of incubation was lung >liver>bladder>kidney. It is concluded that the cell-mediated mutagenesis approach can be used to study organotropic effects of certain chemical carcinogens under controlled conditions.

82-0346. LaVoie, E. J.; Tulley-Freiler, L.; Bedenko, V.; Girach, Z.; Hoffmann, D. **Comparative Studies on the Tumor Initiating Activity and Metabolism of Methylfluorenes and Methylbenzofluorenes.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 417-427.

The molecular basis for the mutagenic activity of 9-alkylfluorenes toward *S. typhimurium* was explored. Comparative studies of several 9-alkyl fluorenes and 9-phenylfluorene showed that mutagenic potential is inhibited by increasing the alkyl chain length beyond two carbon units, or by phenyl and secondary alkyl substituents. These studies suggest the possible formation of a spiroepoxide as the ultimate mutagen of 9-methylfluorene. In evaluating the tumor-initiating activity, it was found that 11-methylbenzo(*b*)fluorene is active as a tumor initiator on mouse skin; but the other fluorenes, benzofluorenes, and their mutagenic methylated derivatives assayed were inactive.

82-0347. Lubawy, W. C.; Isaac, R. S. **Inhibition of Pulmonary Metabolism of Benzo(*a*)pyrene Produced by Acute Tobacco Smoke Exposure.** In: Bjorseth, A.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects.* Columbus, Ohio, Battelle Press, 1980, pp. 243-257.

Cigarette smoke exposure in rabbits was found to have an initial inhibitory effect on the pulmonary metabolism of benzo(*a*)pyrene (BaP), in contrast to a delayed induction effect. The carcinogenic potential of BaP can be developed by cytochrome P-450 mediated aryl hydrocarbon hydroxylase (AHH) metabolizing BaP to reactive intermediates. Chronic smoke exposure may increase the activity of pulmonary AHH. Following administration of 7,10-^{14}C-labeled BaP to isolated perfused rabbit lungs (pretreated 24 hours earlier with 3-methylcholanthrene (3mc), tobacco smoke condensate (TSC), or corn oil), BaP in rabbit lungs exposed to 3mc or TSC disappeared more rapidly, and more metabolites were produced than in lungs treated with corn oil. Labeled BaP was also added to rabbit lungs immediately after exposure to cigarette smoke or to the gas phase (obtained by filtering particulate matter from smoke). The lungs exposed to whole smoke showed an increase in unchanged BaP recovered, while gas phase exposure results were not significantly different from those of the controls. Rabbit lungs were also exposed to whole smoke 30 minutes after labeled BaP was administered, by which time most of the BaP had been metabolized and metabolite levels in the perfusate had reached their peak. The metabolites disappeared more slowly in lungs exposed to whole smoke than in controls. Study results indicated that cigarette smoke initially inhibited the pulmonary metabolism of BaP, and decreased the bioconversion of the primary metabolites of BaP, probably due to the particulate matter of the smoke, since the gas phase alone produced little effect, although the mechanism for the initial inhibitory effect is unknown. The delayed effect of smoke exposure was to induce or promote the pulmonary metabolism of BaP.

82-0348. Marshall, M. V.; McLemore, T. L.; Martin, R. R.; Marshall, M. H.; Wray, N. P.; Busbee, D. L.; Cantrell, E. T.; Arnott, M. S.; Griffin, A. C. **Benzo(*a*)pyrene Activation and Detoxification by Human Pulmonary Alveolar Macrophages and Lymphocytes.** In: Bjorseth, A.; Dennis, A. J. (Editors).

Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects. Columbus, Ohio, Battelle Press, 1980, pp. 299-318.

The activity of pulmonary alveolar macrophages (PAM) in the metabolism of benzo(*a*)pyrene (BaP) may be a predisposing factor in lung cancer. One-hour incubation of PAM with BaP resulted in metabolites of phenols and quinones. During 24 hours of incubation, major metabolites produced were 7,8-dihydrodiol and 9,10-dihydrodiol. PAMs that had been induced with benz(*a*)anthracene (BaA) prior to incubation with BaP produced more metabolites than noninduced macrophages. Lymphocytes displayed less metabolic activity of BaP than PAMs, but showed greater inducibility with BaA. Monocyte precursors of PAMs also demonstrated a twofold to fivefold greater metabolism of BaP than lymphocytes. PAMs of smokers metabolized more BaP and produced more 7,8-dihydrodiol than nonsmokers, possibly due to prior induction of aryl hydrocarbon hydroxylase by cigarette smoke.

82-0349. McManus, I. C.; Herning, R. S.; Jones, R. T.; Mines, A. H.; Bachman, J. **Puff Volume Increases When Low-Nicotine Cigarettes Are Smoked. (Letters).** *British Medical Journal* 283(6300): 1186, October 31, 1981.

The conclusion of a previous article (*British Medical Journal* 283(6285): 187-189, July 18, 1981) that cigarette smokers took a greater puff volume with low-nicotine than with high-nicotine cigarettes is criticized. Figures from the article indicate that the greater puff volume is present at the very first puff, and since nicotine from this first puff does not reach the central nervous system in less than eight seconds, smokers inhale more deeply for purely sensory factors. Also, the differences in puff volume of cigarettes of varying strengths decrease as more puffs are taken. Therefore, it is felt that there is no substantial evidence for nicotine addiction in tobacco dependence. The authors reply that nicotine content was the only variable in the experimental cigarettes; thus different puffing behavior was in response to the nicotine content of the cigarettes. The very first puff was excluded from the data because it has an undetermined volume of air in addition to smoke. In addition, they agree that the sensory effects of nicotine may have influenced initial puffing behavior; but after enough time had passed for nicotine to reach the central nervous system, puffing behavior reflected the nicotine content of the cigarette. Thus the results supported the role of nicotine in tobacco dependence. See also 81-1932.

82-0350. Meltzer, L. T.; Rosecrans, J. A. **Investigations on the CNS Sites of Action of the Discriminative Stimulus Effects of Arecoline and Nicotine.** *Pharmacology Biochemistry and Behavior* 15(1): 21-26, July 1981.

The dorsal hippocampus (DH) and mesencephalic reticular formation (MRF) were found to have an important role in mediating the discriminative stimulus (DS) effects of nicotine but not of arecoline in rats. The rats were trained to discriminate nicotine (1.14 mg/kg) from saline. Unlike arecoline, peripherally administered nicotine generalized to injection of nicotine directly into the DH and MRF. The stimulus effect of centrally administered nicotine was antagonized by peripherally administered mecamylamine, but not atropine. Response rate decreases were also observed after nicotine injection into either central site. In rats trained to discriminate arecoline (1.74 mg/kg) from saline, peripherally administered arecoline did not generalize to the direct injection of arecoline into the DH and MRF. However, a decrease in response rates was observed after arecoline injection into either site. Thus, the DH and MRF are important in mediating the DS effects of nicotine but not arecoline. (Auth. Abs. Mod.)

82-0351. Michiels, F.; Duverger, A.; Chouroulinkov, I. **Correlation Between Preneoplastic Lesions in Rat Embryo Lung Treated With B(*a*)P or CSC in Organ Culture and Tumour Production *In Vivo*.** *Carcinogenesis* 2(9): 885-896, 1981.

Stages in the development of preneoplastic alterations were established in vitro which are significantly parallel to those observed in rat lung tumorigenesis in vivo. This study followed the evolution of normal and treated rat embryo lungs in organ culture and classified the induced lesions in relation to the in vivo tumor histogenesis. Lungs from 15-day-old Wistar rat embryos were maintained in organ culture in M 199 medium and 20 percent horse serum and treated with benzo(*a*)pyrene (BaP) or cigarette smoke condensate (CSC). Explants were studied during 30 days of culture. The control explants display a prevalence of epithelial tissue along with a reduction in connective tissue, whereas treated explants show similar morphological alterations after both BaP and CSC. The percentage of the observed lesions depends on the strain of rats used. Alterations are classified into preneoplastic stages according to Shabad's pattern developed for rat lung tumors in vivo. This classification permits the assessment and quantitation of the carcinogenic effect of a substance. To prove that lung explants may undergo neoplastic transformation after treatment in vitro, normal and carcinogen-treated lung organ explants were dispersed and, after cell subculture, injected intraperitoneally into isogenic rats. Tumors were obtained after only 5 subcultures with treated cells, while with the controls, tumors were obtained after 21 subcultures. (Auth. Abs.)

82-0352. Orzechowska-Juzwenko, K.; Polarz, H. **Wplyw Palenia Tytoniu na Farmakokinetyke Fenazonu. [Effect of Smoking on Phenazone Pharmacokinetics.]** *Polski Tygodnik Lekarski* 36(11): 413-415, March 16, 1981, Polish.

The coefficient of elimination, K; the period of retention, $t_{0.5}$ = ln 2/K; the volume distribution, V_d = D/C_0 (D-dose, C_0-initial concentration); and clearance Cl = Vol x K of phenazone were determined in tests designed to evaluate the extent of smoking interference with biotransformation of phenazone as a model drug in research of metabolic activity of liver microsomal enzymes. Phenazone was administered orally on an empty stomach at a dose of 18 mg/kg of body weight to a group of 19 male and female smokers of 10 to 40 cigarettes/day, aged 20 to 59, and to 20 controls (nonsmokers) of similar sex and age distribution. Phenazone level in blood plasma of the subjects was determined before drug administration and 3, 6, 9, 12, and 24 hours after. The average K and Cl values in the smokers were found to be statistically significantly higher and the average $t_{0.5}$ values shorter than in nonsmokers. These data indicated a significantly accelerated phenazone elimination in smokers as compared with the controls. The results suggest that metabolism of other drugs in smokers may also be accelerated, a fact to be taken into account in selecting drug dose.

82-0353. Putzrath, R. M.; Langley, D.; Eisenstadt, E. **Analysis of Mutagenic Activity in Cigarette Smokers' Urine by High Performance Liquid Chromatography.** *Mutation Research* 85(3): 97-108, June 1981.

The chemical nature and biological activity of the mutagens in cigarette smokers' urine is characterized as part of an investigation to define the possible relationship between cancer risk and mutagens in body fluids. Mutagenic activity in smokers' urine which had been concentrated by XAD-2 resin can be separated from approximately 90 percent of the nonmutagenic material by CH_2Cl_2 extraction. The extract appears to be stable for 3 months at -20°C. High performance liquid chromatography analysis of the CH_2Cl_2 extract showed multiple mutagenic nonpolar fractions which were better activated by rat liver homogenates prepared from 3-methylcholanthrene treated rats than from rats treated with phenobarbital. Mutagenic activity in smokers' urine was extractable by acid, but not by base. The mutagens in smokers' urine appear to be a complex mixture of relatively nonpolar chemicals. (Auth. Abs. Mod.)

82-0354. Romano, C. **Nicotine Receptors in the Rat: Behavioral, Pharmacological, and Biochemical Studies.** Doctoral Dissertation, Stanford University, Department of Pharmacology, Stanford, California, University Microfilms International 81-08986, November 1980, 164 pp.

Nicotine as a discriminative cue was studied in rats trained to differentially respond to nicotine and saline injections. Once trained, their response accuracy was over 90 percent. Using a new statistical procedure developed to analyze these experiments, the nicotine cue was shown to be stereospecific, and therefore a receptor-mediated phenomenon. Another new system was utilized to study nicotine's ganglionic actions in the rat, using the oxotremorine-contracted longitudinal muscle of the rat terminal colon, which transiently relaxes in the presence of nicotine. This effect was shown to be a typical nicotinic ganglionic action, since it was blocked by tetrodotoxin and hexamethonium. Nicotine's effect was mimicked by classical nicotinic agonists, and their relative potencies were quantified. A binding assay was developed to directly study the chemical interaction of nicotine with its receptor on rat membranes. More displaceable binding is seen in the presence of rat membranes than in their absence; this binding is time and temperature dependent, and is linearly related to the amount of brain membranes present. This demonstrates the presence of a specific binding site for nicotine on rat membranes.

82-0355. Santodonato, J.; Basu, D.; Howard, P. H. **Multimedia Human Exposure and Carcinogenic Risk Assessment for Environmental PAH.** In: Bjorseth, A.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects.* Columbus, Ohio, Battelle Press, 1980, pp. 435-454.

Smoking a pack of cigarettes could result in less exposure to benzo(*a*)pyrene than consuming smoked or charbroiled food. The average human diet results in exposure to polynuclear aromatic hydrocarbons (PAH) which may surpass tobacco smoking as a major contributor to human PAH body burden, and is the most significant source for nonsmokers. Contamination of ambient air and water varies, and is usually a small fraction of the intake from food. The quantitative aspects of exposure from these sources are unknown, but the impact of PAH in humans may be underestimated by traditional theories which considered inhalation as the primary route of exposure.

82-0356. Sarantos-Laska, C.; McCulloch, M. W.; Rand, M. J.; Story, D. F. **Nicotinic Release of Noradrenaline in the Presence of Tetrodotoxin From Sympathetic Nerve Terminals in Rabbit Isolated Atria.** *Journal of Pharmacy and Pharmacology* 33: 315-316, 1981.

An investigation of rabbit isolated atria found that tetrodotoxin blocked the noradrenaline-releasing action of low but not of high concentrations of nicotine. Noradrenaline release in the atria was elicited by exposure to nicotine at 10, 50, or 100 μM for 3 minutes or by electrical field stimulation, in the absence or presence of tetrodotoxin at 0.9 μM. The amounts of noradrenaline released by field stimulation and by 10 μM nicotine were similar, and were reduced by tetrodotoxin to 10 percent and 39 percent, respectively, of control values. The 50 μM and 100 μM concentrations of nicotine released larger amounts of noradrenaline, and the tetrodotoxin did not reduce the effects. It may be that the mechanism of action of nicotine depends on its concentration.

82-0357. Seidrup, J. **Relating Plasma Levels of Clomipramine and Clinical Response.** *Journal of International Medical Research* 8(Supplement 3): 96-110, 1980.

In a study relating plasma concentrations of clomipramine and clinical response, it was found that smoking did not affect either the clinical response or side effects to the drug. Other factors examined for interaction with clomipramine were the contraceptive pill, and age. No relationship was evident between plasma concentrations of clomipramine, clinical response of the patients, and cigarette smoking.

82-0358. Smith, L. C.; Doody, M. C. **Kinetics of Benzo(a)pyrene Transfer Between Human Plasma Lipoproteins.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 615-624.

The kinetics of benzo(*a*)pyrene (BaP) transfer between human plasma lipoproteins are presented. The halftimes for the transfer between high-density lipoproteins, between low-density lipoproteins and between very low-density lipoproteins were 40 msec, 180 msec, and 390 msec, respectively. The increase in monomer fluorescence was first order, in agreement with the results for transfer of pyrene and other fluorescent lipids between lipoproteins. The transfer of BaP between lipoproteins of the same density class was about 20-fold slower than that of pyrene under the same experimental conditions. The rate of BaP transfer between lipoproteins also decreased with increasing size of lipoproteins, in agreement with the behavior of pyrene and other fluorescent lipids in these experimental systems. (Auth. Abs. Mod.)

82-0359. Sonin, B. V. **Vliyanie Etilovogo Alkogolya, Nikotina i Ikh Sochetaniya na Nekotorye Pokazateli Razvitiya Kurinogo Embriona. [Effect of Ethanol, Nicotine and Their Combination on Characteristics of Chick Embryo Development.]** *Farmakologiya i Toksikologiya* 44(3): 347-353, 1981, Russian.

Egg white, egg yolk, or air chamber of 220 chick embryos were inoculated with 0.25 ml of buffered aqueous solutions of nicotine, or ethanol, or both at three different concentrations (0.1, 0.03, and 0.015 percent for nicotine). A high percentage of the embryos died at an early stage of incubation if egg white or yolk were inoculated. Hence, in the subsequent experiments the solutions of nicotine and alcohol were introduced into air chamber only. The embryos were taken out 1 day before the end of natural incubation period and were examined. The median lethal dose LD_{50} of nicotine was found to be 4.4 mg/kg, and its absolute embryo specific lethal dose ELD_{50} was 2,400 times that of alcohol. The dose to effect dependence was linear for all preparations tested at any dose. The toxic effect of nicotine manifests primarily at the cellular and subcellular levels and affects mainly cardiovascular system and liver. At a high dose a single combined alcohol-nicotine inoculation resulted in an increase in LD_{50} compared with separate inoculation. At intermediate or small doses, single or repeated combined inoculation did not change appreciably LD_{50}.

82-0360. Statland, B. E.; Winkel, P. **Response of Clinical Chemistry Quantity Values to Selected Physical, Dietary, and Smoking Activities.** In: Stofanini, M.; Benson, E. S. (Editors). *Progress in Clinical Pathology.* The University of Minnesota Issue. Volume 8, New York, Grune and Stratton, 1981, pp. 25-44.

Recent literature, published in selected clinical chemistry and human physiology journals, relating to the general problem of the preparation of a patient prior to specimen collection, is critically reviewed. Included are investigations of hormones, metabolites, and various enzymes for which new assays are now available. Factors responsible for the perturbations occurring prior to specimen collection are grouped under the following headings: (1) posture, (2) exercise, (3) fasting and malnutrition, (4) ethanol ingestion, (5) diet, and (6) tobacco smoking. Smoking has been demonstrated to cause higher hemoglobin concentration, higher erythrocyte counts, larger erythrocytes, increased concentration of norepinephrine and epinephrine, as well as serum gastrin increases. It is concluded that since healthy subjects engage in a number of activities which affect the concentrations of certain clinical quantities, the noted effects should be exploited by designing new, dynamic types of laboratory tests rather than being suppressed by demanding rigorous and often ill-founded regulations to be fulfilled prior to specimen collection.

82-0361. Sullivan, J. M.; Rana, M. W. **Effects of Nicotine on Norepinephrine Metabolism in Female Rat Heart.** *IRCS Medical Science: Biochemistry* 9: 1014-1015, 1981.

Chronic administration of nicotine in young female Sprague-Dawley rats led to a slight change in norepinephrine (NE) levels in the heart, although the change was not considered significant. Twenty-four rats with an initial weight of 60-65 g were divided into an experimental group and a control group. The experimental animals were administered 1.0 mg/kg nicotine in one injection per day for 60 days, while the control animals received 1.0 ml of 0.9 percent sterile saline injection per day for the same period. Overt behavioral responses to nicotine injections included circling, tremor, increased breathing, and sympathetic stimulation. After sacrifice of the animals at 60 days, the average weight of the saline-injected rats was 230±2.5 g compared to 210±3.5 g for the nicotine-injected rats. The average level of NE in the heart of nicotine-treated rats was 592±60 ng/g, compared to 458±74 ng/g for the saline-injected rats. The slight change in NE in the female rat heart was not statistically significant.

82-0362. Woebkenberg, N. R.; Mostardi, R. A.; Ely, D. L.; Worstell, D. **Carboxyhemoglobin and Methemoglobin Levels in Residents Living in Industrial and Nonindustrial Communities.** *Environmental Research* 26(2): 347-352, December 1981.

Measurements of carboxyhemoglobin (COHb) and methemoglobin (METHb) among residents living in an industrial (Cleveland, Ohio) and a nonindustrial (Elyria, Ohio) community found that COHb levels were significantly higher among both male and female smokers and nonsmokers in Cleveland compared with their counterparts in Elyria. METHb levels were significantly higher in Elyria even though ambient NO, and NO_2 levels were higher in Cleveland. Subjects smoking 10 or more cigarettes, cigars, or pipes per day were designated smokers. Those who did not smoke and never smoked were designated nonsmokers. Male smokers at both sites had significantly higher COHb values than did females, possibly attributable to higher consumption among the male smokers:

male smokers in Cleveland smoked an average of 26.7 pack years compared with 24.6 pack years for females. In Elyria, the respective figures were males, 29.4; females, 26.5. The higher METHb levels in Elyria were attributed to higher nitrate levels in the drinking water, possibly due to inorganic fertilizer runoff from surrounding farmland. In Cleveland, both male and female smokers had significantly higher METHb values than nonsmokers, differences not found in Elyria. It is suggested that METHb has physiological importance and, in combination with elevated COHb values, could be a health factor. (Auth. Abs. Mod.)

82–0363. Wyatt, R. J.; Bridges, R. B.; Halatek, D. G. **Complement Levels in Cigarette Smokers: Elevation of Serum Concentrations of C5, C9, and C1-Inhibitor.** *Journal of Clinical Laboratory Immunology* 6(2): 131-135, September 1981.

Serum level measurements of 13 individual complement component and control proteins conducted among 25 male cigarette smokers and 25 male nonsmokers showed a significant elevation of the mean levels of C5, C9, and C1-inhibitor for the smokers. The levels of C1q, C2, C4, C3, C6, C8, Factor B, properdin, C3b inactivator and βlH did not differ significantly between the two groups. Serum level of C9 in individual smokers correlated significantly with both the present amount of consumption and cumulative consumption, while C5 level correlated with present consumption. The complement profile in the cigarette smoker is similar to that observed in certain inflammatory conditions. The elevation of C5, C9, and C1-inhibitor levels may reflect the presence of a low grade inflammatory process in the cigarette smoker. (Auth. Abs. Mod.)

MORTALITY AND MORBIDITY

82–0364. Goldman, R. H.; Peters, J. M. **The Occupational and Environmental Health History.** *Journal of the American Medical Association* 246(24): 2831-2836, December 18, 1981.

A 4-step procedure for detecting occupational or environmental diseases promoted by exposure to a variety of agents, including cigarette smoke, is described. First, a physical examination should be made and a routine patient history prepared including a list of current and past longest held jobs; the patient should be questioned about exposure to fumes, chemicals, dusts, loud noise, or radiation; and an examination should be conducted of the complaint for any temporal relationship to work or home activities, relationship to job titles and history of exposure, and contributing factors such as cigarette smoking or medications. Smoking as an agent is associated with chronic bronchitis and emphysema, and may induce other illnesses if contaminated by chemical agents at the workplace. Second, sources of exposure in the workplace should include a list of all jobs, and note areas of employment and items manufactured, a description of the work operation, and illnesses in other workers; while the assessment of home surroundings should consider possible contamination from neighborhood external pollution or household contamination. The third step, to identify and characterize the hazardous agent, includes finding the generic name if the agent is a chemical, describing the physical form if it is a dust, or determining the form if it is radiation. It is also necessary to describe how the agent is handled and its mode of entry. Smoking at work or home is one way of entry for an agent. The fourth step involves followup, sources of consultation, and resolution of the problem. Using such methods, a practitioner can help in the detection and prevention of occupational and environmental diseases.

82–0365. Hamman, R. F.; Barancik, J. I.; Lilienfeld, A. M. **Patterns of Mortality in the Old Order Amish. I. Background and Major Causes of Death.** *American Journal of Epidemiology* 114(6): 845-861, December 1981.

Lifestyle may be the primary determinant of overall mortality patterns in the Old Order Amish people who exercise vigorously and avoid cigarettes and alcohol (although pipe and cigar smoking and tobacco chewing are condoned). Three settlements in Indiana, Ohio, and Pennsylvania were compared with neighboring non-Amish communities utilizing death certificates and Amish censuses to determine mortality risks which were summarized using age-adjusted mortality ratios (MRs). Amish mortality patterns were not systematically higher or lower than those of the non-Amish, but differed by age, sex, and cause. Amish males had slightly higher all-cause MRs as children and significantly lower MRs over the age of 40, due primarily to lower rates of cancer (MR = 0.44, age 40-69), and cardiovascular diseases (MR = 0.65, age 40-69). Amish female MRs for all causes of death were lower from age 10 to 39, not different from 40 to 69, and higher over age 69. MRs were not significantly different for all cancer sites combined in Amish women, and they had higher cardiovascular mortality ratios at age 70 and over (MR = 1.34). Mortality ratios from all respiratory diseases were significantly lower by over 80 percent in Amish males age 40-69 and by 50 percent in those 70 and over. In the chronic pulmonary diseases category, including emphysema, bronchitis, and asthma, the lower number of deaths in both males and females was highly significant and may reflect the relative absence of cigarettes and urban and occupational air pollutants.

82–0366. Philips, B. U., Jr.; Bruhn, J. G. **Smoking Habits and Reported Illness in Two Communities With Different Systems**

of Social Support. *Social Service and Medicine. Part A—Medical Sociology* 15(5): 625-631, September 1981.

A retrospective comparison of smoking habits and reported illness in Roseto, Pennsylvania, and Tecumseh, Michigan, was undertaken to determine the role of different systems of social support in abating the consequences of risk behaviors such as smoking on chronic diseases. In this study of 898 adult inhabitants of Roseto, a demographically homogeneous Italian-American community, and 4,590 adult inhabitants of Tecumseh, a heterogeneous geographically defined community, smoking habits were related to health and social characteristics. In both communities smokers of both sexes had less education than nonsmokers, although the reverse was true among women with 13 years or more of education. The smoking habits of husbands and wives were also similar. No significant relationship was found in either community between smoking (or the amount smoked in Roseto) and coronary heart disease, hypertension, or somatic complaints. Cigarette smoking was more common, however, among respondents with a history of peptic ulcers; and in Roseto these persons with ulcers also tended to be heavy smokers. A greater prevalence of hypertension was observed in Tecumseh among males and females than in Roseto. These results are interpreted in light of differing systems of social support in the two communities. (Auth. Abs.)

82-0367. Vogt, T. M. **Risk Assessment and Health Hazard Appraisal.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 2. Palo Alto, California, Annual Reviews Inc., 1981, pp. 31-47.

Three methods for assessing health risks, such as smoking, are screening for a single or specific disease, Multiphasic Screening (MPS), and Health Hazard Appraisal. Screening for a single disease is intended to cull from a large population those subjects most likely to have a disease or condition under study. The sensitivity of a screening test is the proportion of all persons with disease who have a positive test. Specificity is the proportion of all persons without a condition who have a negative test. The predictive value is the fraction of all positive tests that actually represent disease cases. It is dependent on disease prevalence and, with sensitivity and specificity, determines a screening test's viability. Intense screening activities have been conducted in the past decade in the field of cardiovascular diseases. Multiphasic Screening involves the use of multiple examinations and tests to identify potential health problems at as early a stage as possible. The costs of MPS may be larger than those of single disease screening. It may be possible to identify combinations of risk factors that could have different clinical implications than would any one of the factors alone. Evaluating MPS accurately can be difficult. Health Hazard Appraisal is designed to estimate the risk of illness and death in individual patients. It involves the use of chemical, actuarial, and epidemiologic findings to create estimates of risk of death based on known risk factors. The value of assessing health risks varies, and is related to the characteristics measured and to the effort given to necessary changes and therapies.

82-0368. Wingerson, L. **Executive Women—Healthier Than Thou?** *New Scientist* 91(1271): 718-721, September 17, 1981.

In recent years the rate and pattern of disease in women has become more like that of men, a change that may be partly associated with increased risk behaviors among women, such as smoking. Other behavioral factors that may be influencing this trend include alcohol consumption and various types of stress. Coronary heart disease and lung cancer rates have been rising among women in the United States and the United Kingdom in recent decades. Ulcers have also increased among women in both countries, and since smoking irritates the digestive tract, many doctors suspect a link between ulcers and smoking. Although men and women in general may be less likely to smoke than they were a decade ago, the decline appears more pronounced among professional women than among women in the lower social classes.

See also, 82-0527

NEOPLASTIC DISEASES

82-0369. American Cancer Society. **1982 Cancer Facts and Figures.** New York, American Cancer Society, 1981, 32 pp.

Cigarette smoking is responsible for 83 percent of lung cancer cases among men, and 43 percent among women, and has been implicated in cancer of the mouth, pharynx, larynx, esophagus, pancreas, and bladder, as well as ulcers, emphysema, chronic bronchitis, and heart disease. Smoking-related disorders are estimated to cause some 325,000 premature deaths each year, and cost the nation about 27 billion dollars in medical care. Besides tar and nicotine, cigarette smoke contains poisonous gases—hydrogen cyanide, volatile aromatic hydrocarbons, and especially carbon monoxide—which has been cited as a possible critical factor in coronary disease. In addition, a number of flavoring additives or cellulose-based tobacco substitutes may be included in manufactured cigarettes; some of these available for use are either known or suspected carcinogens, or give rise to carcinogenic substances when burned. The Surgeon General has linked maternal smoking directly with a slowing of fetal growth and an increase in risk of infant death at or shortly after birth. There is evidence that children of smoking mothers may have deficiencies in physical growth, intellectual development, and/or emotional development. Industrial workers are especially susceptible to lung diseases due to the combined effects of cigarette smoking and exposure to toxic industrial substances. Exposure to asbestos in combination with cigarette smoking increases an individual's cancer risk about 60 times.

82-0370. Angeloff, L. G. **Cigarette Smoking Is Not the Cause of Cancer: Effect of Nicotine and Tar on Nucleic Acids of Rat Lung and Spleen.** *Nucleus* 23(1/2): 56-59, April and August 1980.

Data are presented from a study of the effect of nicotine on rat lung and spleen slices, and tumor cells, and of observations of humans in relation to rats, which appear to confirm that cigarette smoking is not the cause of cancer. Results showed that (1) nicotine and tar in vitro did not show any effect on normal tissues, (2) cells of Novikoff (Hepatoma) tumor were more sensitive to tar and nicotine than normal cells, and (3) cancer as a primary growth appears on soft tissues such as the lung and the breast. It is concluded that the high mortality from lung cancer in males (18.22 percent) and from breast cancer in females (14.42 percent) can only be explained by assuming that cancer is capable of choosing its own sites and occurs in the softest tissues of the host (human or rat).

82-0371. British Medical Journal. **Coffee Drinking and Cancer of the Pancreas.** *British Medical Journal* 283(6292): 628, September 5, 1981.

Comments and reactions to a study (*New England Journal of Medicine* 304(11): 630-633, March 12, 1981) which had found pancreatic cancer to be associated with cigarette smoking and coffee drinking, are reported. In interviews with 369 pancreatic cancer patients and 644 controls, the relative risk of the disease in smokers was 1.4 times that in nonsmokers; 1.8 for those drinking up to two cups of coffee daily, compared to those who weren't coffee drinkers; and 2.7 for those drinking three or more cups. The risks were calculated after being adjusted for the smoking by coffee drinkers. The dose-response relationship of increasing risk associated with increasing consumption was found only in women. No association was found for the use of cigars, pipe tobacco, alcohol, or tea. However, such a case-control study can have numerous sources of bias and several sources are suggested, including the selection of controls, since potential control patients with diseases associated with smoking and alcohol (cardiovascular disease, diabetes mellitus, respiratory or bladder cancer, and peptic ulcers) were not included. Smoking and coffee drinking are correlated, and the exclusion may have removed heavy coffee drinkers from the control group. It is agreed that further studies are needed to confirm the reported association. See also 81-0775.

82-0372. Burch, J. D.; Howe, G. R.; Miller, A. B.; Semenciw, R. **Tobacco, Alcohol, Asbestos, and Nickel in the Etiology of Cancer of the Larynx: A Case-Control Study.** *Journal of the National Cancer Institute* 67(6): 1219-1224, December 1981.

A causal role for asbestos exposure and cancer of the larynx was supported in a case-control study of laryngeal cancer conducted in southern Ontario between 1977 and 1979 of 204 subjects with newly diagnosed cancer and 204 controls, individually matched by sex, age, and residence. Tobacco products and alcohol showed strong associations with cancer of the larynx for males, with relative risks (RR) for users of cigarettes, cigars or cigarillos, pipes, and alcohol of 6.1, 2.9, 1.6, and 5.2, respectively. The population attributable risk percent for males using tobacco products and alcohol together was estimated to be 94 percent. Cigarette smoking was also an important risk factor for females, although the small number of female pairs (20) precluded any meaningful detailed analysis of other possible risk factors. The RR for males for exposure to asbestos after the effects of cigarette smoking were controlled was 2.3, and the effects seemed restricted to cigarette smokers. The findings on asbestos were based on small numbers of cases and controls exposed and consequently were subject to large sampling errors. The estimate was consistent, however, with that from other studies. (Auth. Abs.)

82-0373. Busbee, D. L.; McLemore, T. L.; Martin, R. R.; Wray, N. P.; Marshall, M.; Cantrell, E. T. **High Aryl Hydrocarbon Hydroxylase Inducibility Is Positively Correlated With Occurrence of Lung Cancer.** In: Bjorseth, A.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects.* Columbus, Ohio, Battelle Press, 1980, pp. 917-934.

Pulmonary alveolar macrophages (PAMs) from lungs of healthy cigarette smokers had significantly higher levels of aryl hydrocarbon hydroxylase (AHH) than a similar group of nonsmokers. AHH levels in PAMs increased significantly over a 30-day period after initiation of smoking, and decreased to normal levels 3 months after cessation of smoking. AHH was inducible in vitro in PAMs by cigarette tar extracts. Comparisons of PAM AHH levels and in vitro benz(*a*)anthracene (BaA)-induced PAM AHH levels showed correlations for both lung cancer and noncancer patients, all of whom were smokers. In noncancerous patients, a correlation existed between AHH levels in PAMs and lymphocytes, which did not exist in lung cancer patients. A significantly large percentage of those highly inducible for AHH are found in the lung cancer population, but none of these patients were highly inducible for AHH in both PAMs and lymphocytes.

82-0374. Casey, K. R.; Rom, W. N.; Moatamed, F. **Asbestos-Related Diseases.** *Clinics in Chest Medicine* 2(2): 179-202, May 1981.

Diseases associated with asbestos include benign pleural disease such as pleural plaques and benign asbestos pleurisy, pulmonary asbestosis, diffuse malignant mesothelioma, bronchogenic carcinoma, and neoplasms in other sites, including laryngeal carcinoma, gastrointestinal cancer, and other cancer. Smoking is common among asbestos workers and may increase mortality from asbestosis. Exposure to asbestos may take place during the mining, milling, and preparation of the mineral; during its incorporation into products; by sawing, cutting, or breaking of finished asbestos products; or at a worksite where asbestos is used, in the homes of asbestos workers, or in the community at large. Much of the airways obstruction found in many asbestos workers is the result of cigarette smoking, although some individuals may respond to asbestos exposure. Cigarette smoking and asbestos exposure do not appear to interact in producing diffuse malignant mesothelioma. Lung cancer death rates appear to be higher among smoking asbestos workers than for smokers not exposed to asbestos, nonsmoking asbestos workers, or nonsmokers unexposed to asbestos. The two etiologic factors of asbestos exposure and smoking seem to interact in a multiplicative manner, as in pulmonary cancer, where asbestos may act as a cocarcinogen to accentuate the carcinogenic process initiated by cigarette smoke. Asbestos workers should be advised to quit smoking, to decrease the high risk of cancer observed when the two carcinogenic influences are combined.

82-0375. Clemmesen, J. **Uses of Cancer Registration in the Study of Carcinogenesis.** *Journal of the National Cancer Institute* 67(1): 5-13, July 1981.

The clinical usefulness of cancer registration and advances made in cancer research as a result of registration are outlined, including the role of smoking data in cancer studies. At the outset of cancer registry in 1942, 5-year retroactive studies led to postulation of a 2-decade lag time between onset of smoking and appearance of lung cancer. After 10 years of cancer registry, one study showed an association between bladder cancer and smoking. Later studies confirmed a correlation between cigarettes and cancer of the pancreas. Smoking was also found to add to certain occupational factors in carcinogenesis, as in asbestos workers. Methods and a comparison of international cancer registers are discussed.

82-0376. Fortic, B. **Karcinom Pluca u Sloveniji i Njegovi Epidemioloski Trendovi (Studija u Vremenu od 1953 do 1976 Godine). [Lung Cancer in Slovenia and Its Epidemiological Trends (Study of the 1953 to 1976 Period).]** *Plucne Bolesti i Tuberkuloza* 33(2/3): 89-108, April-September 1981, Serbo-Croatian.

The incidence of lung cancer and its etiology among the population of Slovenia, a republic of Yugoslavia, was studied in relation to sex, age, smoking habit, and geographical location. Results are based on 24-year records of 2,906 patients with lung cancer, histomorphologically diagnosed at the Golnik Institute for pulmonary diseases. The number of the recorded patients with lung cancer per 100,000 population had increased fourfold over the period (total for men and women). A significant correlation was observed between lung cancer incidence and smoking: 80 percent of the patients were cigarette smokers, 15 percent nonsmokers, and 5 percent ex-smokers. Among the 2,516 male patients, 89 percent were smokers compared with only 28 percent smokers among the 390 female patients. The ratio of men smokers to nonsmokers and ex-smokers becomes even higher if only patients with epidermoid carcinoma are considered. Also, micro- and macrocellular carcinoma types were significantly correlated with smoking. Other possible causes of lung cancer epidemic are discussed. It is concluded that the risk of lung cancer is very high for male smokers and its incidence in Slovenia has not yet reached its peak.

82-0377. Grossarth-Maticek, R. **Synergetic Effects of Cigarette Smoking, Systolic Blood Pressure, and Psychosocial Risk Factors for Lung Cancer, Cardiac Infarct and Apoplexy Cerebri.** *Psychotherapy and Psychosomatics* 34(4): 267-272, 1980.

Smoking was found to be a physical risk factor for lung cancer and cardiac infarct in a 10-year study (1965-1975) of 1,353 inhabitants of a Yugoslavian town, especially if combined with a psychosocial risk factor. The disease rates for lung cancer, cardiac infarct, and apoplexy were examined, and an analysis made of the interaction between the strongest physical risk factors (including smoking) and psychosocial risk factors for the diseases. The physical risk factor of systolic blood pressure and the psychosocial risk factor of "rationality and anti-emotionality" appeared to interact in apoplexy and infarct. Smoking and rationality and anti-emotionality also showed an interaction for infarct. For lung cancer, the most prominent factors were smoking and "life events that caused lasting depression and hopelessness." An interaction was also measured for lung cancer between smoking and a less prominent psychosocial risk factor, rationality and anti-emotionality. The interactions were significant in all of the analyses conducted. The effectiveness of the physical risk factors was increased by the psychosocial risk factors.

82-0378. Hirayama, T. **Operational Epidemiology of Cancer.** *Journal of Cancer Research and Clinical Oncology* 99(1/2): 15-28, 1981.

Operational epidemiology is an action-oriented epidemiology directly aimed at cancer control. This approach has been elucidated by illustrating examples pertaining to cancer of the esophagus, lung, and breast. Using data collected by retrospective and prospective studies on these types of cancer, it was observed that relative risk increases substantially when the various contributory factors are taken into account. Thus, the relative risk of esophageal cancer increases when combined with the intake of hot tea-gruel. Relative risk increases further when other factors, such as nutritional deficiencies and cigarette smoking, are considered. Cigarette smoking is by far the most important cancer risk factor. If cigarette smoking disappears, cancer in men will be reduced by one-third. These studies support the well-known hypothesis of initiator and promotor mechanism of carcinogenesis. A few other modifying factors are also illustrated. Based on these studies, strategies for intervention are suggested. (Auth. Abs. Mod.)

82-0379. Hoffmann, D.; Castonguay, A.; Rivenson, A.; Hecht, S. S. **Comparative Carcinogenicity and Metabolism of 4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone and *N'*-Nitrosonornicotine in Syrian Golden Hamsters.** *Cancer Research* 41(6): 2386-2393, June 1981.

The tobacco-specific nitrosamines 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and *N'*-nitrosonornicotine (NNN) were tested for carcinogenic activity in Syrian golden hamsters, the NNK appearing to be the more powerful carcinogen. In Assay A, 30 hamsters were each given 19 injections of 0.048 mmol of NNK or NNN. In Assay B, 20 hamsters each received 75 injections of 0.012 mmol of NNK or NNN. Among the NNK-treated hamsters in Assay A, three developed carcinomas of the nasal cavity, and 19 had adenomas and/or adenocarcinomas of the lung. In the NNN group, one hamster developed a lung adenoma, and five had tracheal papillomas. In Assay B, 11 of the NNK-treated hamsters developed carcinomas of the nasal cavity, 16 had lung adenomas and/or adenocarcinomas, and 7 had tracheal papillomas; in the NNN group, there was only one hamster with a lung adenoma and one with a tracheal papilloma. These findings confirm that NNK is a more powerful carcinogen than NNN. In metabolism studies, 96 to 98 percent of the radioactivity of the injected (1-^{14}C)NNK was recovered in the urine, 4 percent was recovered in the feces, and less than 0.5 percent was recovered as exhaled $^{14}CO_2$. The corresponding distribution of (2'-^{14}C)NNN was 62 to 78 percent in urine, 10 percent in feces, and less than 0.5 percent in respiratory $^{14}CO_2$. The levels of binding of (1-^{14}C)NNK and (2'-^{14}C)NNN to the trichloroacetic acid-insoluble fractions were highest in liver, lung, kidney, and adrenals. The urinary metabolites of NNK and NNN resulted from α-hydroxylation, from *N*-oxidation of NNN to *N'*-nitrosonornicotine-1-*N*-oxide, and from reduction of NNK to 4-(methylnitrosamino)-1-(3-pyridyl)butan-1-ol. (Auth. Abs. Mod.)

82-0380. Holck, S. E.; Warren, C. W.; Rochat, R. W.; Smith, J. C. **Lung Cancer Mortality and Smoking Habits: Mexican-American Women.** *American Journal of Public Health* 72(1): 38-42, January 1982.

Lower smoking levels and lower lung cancer mortality rates were found among survey groups of Mexican-American women compared with Anglo women, in this update of lung cancer mortality data in Texas. Smoking patterns of 1,255 Mexican-American women from a household survey in the four States bordering Mexico are compared with findings for a reference group of Anglo women residing in the same area. In 1970, lung cancer mortality rates were similar for Mexican-American and Anglo women in Texas; however, by 1974-1976 Mexican-American women in Texas had a 40 percent lower rate than Anglo women, and by 1979, a 46 percent lower rate. In the 1979 survey results, Mexican-American women reported lower levels of smoking, both in prevalence and amount smoked, compared with Anglo women. The lower prevalence was reported for all social and demographic categories examined. The relatively low lung cancer mortality rate was most likely due to relatively low levels of cigarette smoking among Mexican-American women. Based on the trend in lung cancer deaths and the survey findings, a continuing low level of lung cancer mortality among Mexican-American women would be anticipated. (Auth. Abs. Mod.)

82-0381. Howe, G. R. **An Epidemiologic Study of Bladder Cancer.** In: Connolly, J. G. (Editor). *Carcinoma of the Bladder.* Progress in Cancer Research and Therapy, Volume 18. New York, Raven Press, 1981, pp. 55-60.

A study of the risk factors in 480 male and 152 female bladder cancer patients interviewed at home, found that smoking was an important risk factor. Each case had an age and sex-matched control. The factors examined were smoking, occupation, possible sources of nitrosamines in the diet, bladder and kidney infections, analgesic consumption, coffee consumption, and artificial sweetener use. Compared with lifetime nonsmokers, the relative risk for cigarette smokers was 3.9 for males and 2.4 for females, both of which were statistically highly significant. Duration and average frequency of use showed highly significant dose-response relationships. Ex-smokers showed a significant decrease in risk relative to current smokers for both sexes. An increased risk was associated with pipe smoking. Pipe smoking also showed a dose-response relationship. In terms of the population attributable risk percent (PARP), cigarette smoking was the most important single risk factor observed, accounting for 61 percent of bladder cancer in males and 26 percent in females. The figures assumed that the association with cigarette smoking was causal, as seemed most likely from the accumulated evidence. Occupational exposure and the use of a nonpublic water supply may have been important risk factors also.

82-0382. Huhti, E. **Smoking and Lung Cancer—Now.** *European Journal of Respiratory Diseases* 62(3): 147-149, June 1981.

Smoking is generally accepted as the main cause of lung cancer, with the cigarette tar appearing to be the most harmful agent present. In developed countries the tar yield of cigarettes has decreased since the 1950s, but in developing countries the greater part of cigarettes sold still have high-tar levels. Polycyclic aromatic hydrocarbons, such as benzo(*a*)pyrene and its metabolites, appear to be the most dangerous carcinogens in cigarette tar. There is some evidence that lung cancer mortality may be less among the smokers of low-tar cigarettes than of high-tar cigarettes. Filter cigarettes may also be associated with lower lung cancer mortality. Although it is suggested that medium-nicotine, low-tar cigarettes may be the least unhealthy cigarettes, it is not yet known to what extent providing smokers with low-tar cigarettes may be beneficial.

82-0383. Kaelin, M. A. **Environmental Cancer: A New Course Proposal.** *Health Education* 12(6): 46-48, November-December 1981.

The objective of a health education course on the relationship of environmental exposure (such as smoking) to cancer is to equip students with the information necessary for making preventive decisions. It is suggested that cancer incidence due to environmental exposures may be as high as 90 percent. The first of five goals is an understanding of the logic of cancer control through primary prevention. Prevention of possible cancers by avoiding or eliminating environmental exposures does not have to wait until the cause of cancer at the cellular level has been identified. The second goal is understanding the strengths and weaknesses of the different methods of identifying carcinogenic exposures. Epidemiological studies are limited in scope because humans have to be exposed to the suspected carcinogen before it can be identified. Although about 20 percent of those who smoke cigarettes develop lung cancer, 80 percent do not. Bioassays are limited by the question of which animal species to use as a comparison for humans and because of the need to use large doses of a suspected carcinogen to compensate for the limited numbers of animals included in the research design. Goal three, recognizing the value and limitations of estimating carcinogenic risk, requires knowledge of the strength or effectiveness of carcinogens. Goal four is to appreciate the subjective nature of judgments about the acceptability of carcinogenic risk. Goal five is the recognition of the citizen's responsibility for developing policies that eliminate or avoid unacceptable carcinogenic risks. The attainment of such goals may help the student to have the information necessary to make knowledgeable cancer prevention policy decisions in the future.

82-0384. Keane, W. M.; Atkins, J. P., Jr.; Wetmore, R.; Vidas, M. **Epidemiology of Head and Neck Cancer.** *Laryngoscope* 91(12): 2037-2045, December 1981.

Epidemiologic factors relating to carcinogenesis of the head and neck are broad and varied. Occupational and environmental exposures to carcinogens have been well identified. Tumors may develop as a result of medical therapies. Susceptibility on a genetic basis has been shown, and the role of viruses in carcinogenesis has been further documented. Nevertheless, cigarette smoking and other forms of tobacco usage continue to be the major cause of head and neck neoplasms. Cigar and pipe smokers have a greater risk of lip cancer, an equal risk for oral cavity cancer, and a lesser risk of lung and laryngeal cancer when compared to cigarette smokers. Tobacco related tumors are also associated with various cultural practices: (1) Bidi smoking in India and Southeast Asia has been attributed to cancer of the tonsil, esophagus, hypopharynx, and base of tongue; (2) in India, Venezuela, and Panama, reverse smoking (lighted end inside the mouth) is related to cancer of the palate; (3) tobacco, mixed with lime, and placed in the gingivobuccal fold is related to a high incidence of cancer in this area in India; (4) snuff, with high amounts of chromium and nickel, used by South African Bantu, have been linked to increased incidence of nasal cancer.

82-0385. Kornegay, H. R.; Kastenbaum, M. A.; Mantel, N.; Harris, J. E.; DuMouchel, W. H.; MacDonald, E. J.; Hirayama, T. **Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer. (Letters).** *British Medical Journal* 283(6296): 914-917, October 3, 1981.

Four letters criticizing Hirayama's claim that nonsmoking wives of smokers have a higher risk of lung cancer than nonsmoking wives of nonsmokers (*British Medical Journal* 282: 183-185, January 17, 1981), and a reply by Hirayama are presented. Comments include the following: occasional smokers and nonsmokers were combined in one group, and ex-smokers were included with a group of light smokers; occasional smokers were not defined, and no explanation is given for these groupings; measurements of the wife's exposure to husband's smoke, and the rationale behind age-occupation standardized annual mortality rates were not given; indications are that a square root was not taken in a statistical calculation, thus the significance value is incorrect; it is unclear whether persons at risk, or person-years at risk, were used to calculate statistical significance, or whether the ages of wives were standardized (such calculations would make the analysis more conclusive); selection of the study population has not been explained and may be biased toward the industrially exposed segment; there are a large proportion of "unknowns" listed for "occupation" in the women surveyed; and the study was based on the ages of the husbands, rather than the wives, which is not justified. Hirayama states that though the survey was not random, the representativeness was satisfactory, as confirmed after the study. Since husbands' ages were closely associated with wives' ages, the husbands' ages were used. The number of cigarettes passively smoked by wives of light smokers was an average of 6.7/day, 8.4/day where husbands were moderate smokers, and 13.5/day with heavy-smoking husbands. Tables by wives' ages, and for age-occupation-specific mortality accompany the letter. See also 81-1088.

82-0386. Lemen, R. A.; Dement, J. M.; Wagoner, J. K. **Epidemiology of Asbestos-Related Diseases.** *Environmental Health Perspectives* 34: 1-11, February 1980.

A review of over 100 epidemiological studies suggests that individuals exposed to asbestos appear to have increased risk of developing respiratory diseases such as asbestosis, lung cancer, and pleural and peritoneal mesotheliomas, with an enhancement of lung cancer risk for those who also smoke cigarettes. It should be noted, however, that historic documentation of cigarette consumption patterns is lacking in most retrospective cohort studies on asbestos workers. Also, there is usually a sizable proportion of smokers among the general population, the group usually used as a comparison in studies. Therefore, the demonstrated lung cancer risk for industrial groups exposed to asbestos is of such a magnitude as to preclude identifying an independent etiologic role for smoking. The epidemiological studies have not shown an increase of pleural or peritoneal mesothelioma among smokers as compared to nonsmokers.

82-0387. Lancet. **Relative or Attributable Risk?** *Lancet* 2(8257): 1211-1212, November 28, 1981.

The significance of smoking in the etiology of conditions such as lung cancer and reduced birth weight is discussed in terms of relative and attributable risks. Relative risk, which can be statistically calculated in case—control studies, does not account for the actual number of disease cases which might be related to a given risk factor. Attributable risk is the measure of the effect of a risk factor on the causation of disease in the whole population rather than in an individual. If a risk factor is identified with a high relative risk, much may be learned about the mechanism of disease. If the risk factor has a low attributable risk due to low prevalence in the population, it would be of less interest for those planning preventive measures. Smoking, for example, gives a moderately increased relative risk of lung cancer, but is very prevalent among the whole population and thus has a very high attributable risk, making it an important consideration in preventive strategies. Maternal smoking has been found to have a significant but relatively much smaller relative risk for infant growth retardation than factors such as a history of small babies and hypertension with proteinuria. If attributable risk is examined, maternal smoking gains in importance. It is suggested that eliminating maternal smoking could reduce the number of small-for-date infants. Attributable risk may be useful in examining the effects of modifying risk factor exposure, but the attributable risk should only be equated with the expected change in disease frequency if the factor is a genuine causal agent and is not merely associated statistically with the disease. Even if there is a causal link, it should be remembered that risk factors are seldom fully independent.

82-0388. Loranskiy, D. N.; Popova, Ye. B. **Nekotorye Aspekty Izucheniya Kombinirovannogo Deystviya Proizvodstvennoy Sredy i Kureniya. [Some Aspects of Research Into the Combined Effects of Industrial Environment and Smoking.]** *Gigiena i Sanitariia* 3: 61-64, 1981, Russian.

Worldwide research of the last 10 years is reviewed on the role of smoking in the incidence of occupational cancer and other diseases in industrial workers occupied with production of carcinogenic or certain noncarcinogenic substances. Polish and Soviet data are cited on potentiation of carcinogenic effect of tobacco smoke by carcinogenic or noncarcinogenic substances (e.g., silicon, asbestos dust) in the atmosphere of industrial plants. Numerous data indicate that the effects of asbestos and nicotine on human organisms are multiplied when both act simultaneously. Polish authors determined that (1) the incidence of respiratory diseases among the workers in wool and cotton products manufacture is higher in smokers, and (2) symptoms of lung diseases among workers of a cement plant and agricultural workers are 2.8 and 7.8 times more frequent in smokers than in nonsmokers. Soviet authors established that sulfur dioxide in tobacco smoke increases carcinogenic effect of benzo(*a*)pyrene and simultaneously sensitizes the mucous membrane of the lungs to the effects of carcinogenic agents.

82-0389. MacMahon, B.; Yen, S.; Trichopoulos, D. **Coffee Drinking and Cancer of the Pancreas. (Letter).** *British Medical Journal* 283(6302): 1335, November 14, 1981.

An editorial (*British Medical Journal* 283(6292): 628, September 5, 1981) criticizing a study which examined the relationship between coffee drinking and cancer of the pancreas (*New England Journal of Medicine* 304(11): 630-632, March 12, 1981) is refuted. Since coffee drinking is associated with cigarette smoking, the critics felt that exclusion of patients with smoking-related diseases from the study may have removed heavy coffee drinkers from the control group. In response, it is noted that as cancer of the pancreas is a smoking-related disease, and coffee drinking is associated with smoking, then in excluding smoking-related cases, such as lung cancer patients, only inappropriate control subjects were left out. See also 82-0371 and 81-0775.

82-0390. Marshall, M. V.; McLemore, T. L.; Busbee, D. L.; Martin, R. R.; Griffin, A. C. **Effects of Selenium and Sulfur on Metabolism of Benzo(*a*)pyrene by Human Pulmonary Alveolar Macrophages.** In: Cooke, M.; Dennis, A. J. (Editors). *Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate.* Columbus, Ohio, Battelle Press, 1981, pp. 221-230.

Selenium levels in cigarette smoke may be a contributing factor to lung cancer incidence. Pulmonary alveolar macrophages (PAMs) obtained from cigarette smokers are less sensitive to trace metal toxicity as demonstrated with oxides of selenium and sulfur. Fly ash samples obtained from a coal-fired power plant are less toxic than fly ash from shale oil. The major benzo(*a*)pyrene (BaP) conjugates produced by human PAMs are sulfate and glutathione conjugates. Both sulfur and selenium enhance conjugation of BaP metabolites, which may be important in the prevention of human cancer. (Auth. Abs. Mod.)

82-0391. McCann, J. **Some Lung Cancer Called Reversible If Smoking Stops.** *Medical Tribune* 21(29): 1, September 17, 1980.

Results based on studies of lung patients suggested that early malignant lung changes may revert to normal if the patients quit smoking. Individuals who show noninvasive lung lesions have been able to reverse the pathology by giving up smoking, although further study is needed to learn if the same result might occur for microinvasive cancers. Dyskaryotic changes have also been found to subside in individuals who stopped smoking. Among a series of surgically treated patients, followed for 5 to 10 years, there was no recurrence of occult carcinomas since the patients gave up smoking.

82–0392. Nou, E.; Hillerdal, O. **Total Tobacco Consumption in an Unselected Bronchial Carcinoma Population.** *European Journal of Respiratory Diseases* 62: 152-159, June 3, 1981.

The smoking habits of an epidemiologically unselected group of patients with bronchial carcinoma in the Swedish county of Uppsala were compared with a control population to determine differences in the smoking habits between male and female patients, and in the various histological groups, and for possible differences between the histological groups within the same sex. Out of 273 bronchial carcinoma patients in the period 1971-1976, 82 percent were smokers (96 percent of the males and 34 percent of the females). In a control group 10-15 years earlier, there were 67 percent male and 26 percent female smokers. The difference in males is statistically significant, but not in females. However, 71 percent of younger female patients (30-64 years) were smokers and this difference from the controls is statistically significant. Ninety-eight percent of the males with epidermoid carcinoma and small cell carcinoma were smokers. Ninety percent of the 40 males with adenocarcinoma were smokers, in contrast to 24 percent of the 38 females. The smoking habits in females with adenocarcinoma were the same as in the female population of the county 15 years earlier. The mean age at the start of smoking was 19 years. The mean total tobacco consumption was 207 kg, 215 kg for males and 130 kg for females, and the mean average consumption per year was 4.6 kg. Fifty-four percent of the males smoked both cigarettes and pipe tobacco. The results verify the relation between smoking and bronchial carcinoma in men and also in younger women. (Auth. Abs. Mod.)

82–0393. Palisano, P. **The War on Cancer: No Quick Victory in Sight.** *Occupational Hazards* pp. 87-91, April 1980.

The greatest single action possible to reduce cancer incidence is to stop smoking. New lung cancer cases have doubled, and survival rates are low. More women are smoking, and smoking more; and over the next 20 to 30 years, lung cancer incidence in women may equal that in men. Cases are also increasing in leukemia, colon, pancreas, prostate, and bladder cancers. In an interview with Dr. Robert W. Kellermeyer, director of University Hospitals Cancer Center of Case Western Reserve University, the causes and cures of cancer are discussed. It is concluded that risk factors vary with each type of cancer; lung cancer had the highest estimated number of new cases for any cancer in 1980, and the greatest single risk factor for lung cancer is smoking.

82–0394. Pottern, L. M.; Morris, L. E.; Blot, W. J.; Ziegler, R. G.; Fraumeni, J. F., Jr. **Esophageal Cancer Among Black Men in Washington, D.C. I. Alcohol, Tobacco, and Other Risk Factors.** *Journal of the National Cancer Institute* 67(4): 777-783, October 1981.

The overall data from a case-control study of esophageal cancer among black men in Washington, D.C., revealed no significant risk associated with cigarette smoking and no consistent enhancement of risk following exposure to both alcoholic beverages and cigarette smoking. The next of kin or close friends of 120 black males who died of esophageal cancer and 250 similarly aged black males who died of other cancers were interviewed to identify the risk factors that may be responsible for the exceptionally high mortality from this cancer in Washington, D.C. The major risk factor was found to be alcoholic beverage consumption, which was estimated to be causally associated with about 80 percent of the neoplasms among the esophageal cancer subjects. Although no significant association was found between cigarette smoking and esophageal cancer, the discrepancy in part relates to different comparison groups. Controls in this study included persons who died from lung cancer and heart disease, conditions known to be induced by cigarette smoking, whereas other studies excluded as controls persons who died from smoking-related conditions. When comparisons were restricted by using only those controls who died of illnesses not known to be linked to smoking, a 90 percent increased risk of esophageal cancer and a dose-responsive relationship were observed for smokers. However, this smoking-associated risk was considerably reduced when controlled for ethanol intake. Possibly, the consumption of such large amounts of alcoholic beverages by the study population overwhelmed the risk that would have been due to cigarette smoking in a population consuming lesser amounts.

82–0395. Reddy, M. S.; Naik, S. R.; Bagga, O. P.; Chuttani, H. K. **Salivary Secretions in Oral Cancer Patients With Chronic Tobacco-Betel-Lime "Quid" Chewing.** *Journal of Oral Medicine* 36(1): 7-10, January-March 1981.

Flow rates of whole saliva obtained by (1) mechanical stimulation with force spitting and (2) 10 percent citric acid stimulation method, as well as salivary pH, amylase, sodium, and potassium were determined in two groups of habitual chewers of tobacco-betel-lime "quid." The first group consisted of 10 patients with oropharyngeal cancer, and the second of 25 otherwise healthy individuals. The two groups were well matched with respect to age, body weight, and sex, although oral cancer patients were somewhat older than healthy chewers. The salivary variables studied were no different in the two groups. The development of cancerous lesions does not appear to influence the salivary flow rates in quid chewers. (Auth. Abs. Mod.)

82–0396. Roe, F.J.C. **Avoidable Cancer Risks With Special Reference to Occupational Factors.** *British Medical Journal* 283(6304): 1421-1422, November 28, 1981.

Highlights of a study (*Journal of the National Cancer Institute* 66(6): 1191-1308, June 1981) which estimated current U.S. cancer mortality for 12 different factors are presented. Tobacco, the single most important identifiable factor, had an estimated contribution of 30 percent, compared to occupational factors, 4 percent; pollution, 2 percent; medicines and medical procedures, 1 percent; industrial products and food additives, each less than 1 percent; ionizing radiation and exposure to ultraviolet light, 3 percent; sexual and reproductive behavior, 7 percent; infections, 10 percent; and alcohol, 3 percent. Dietary factors contributed 35 percent to overall cancer mortality, with alcohol suggested as an epigenetic factor that can facilitate contact between tobacco smoke carcinogens and stem cells in the lining of the upper digestive tract. Lung cancer accounted for 11,000 of the 17,000 annual cancer deaths attributed to occupation, with smoking and specific industrial factors appearing to act synergistically in some cases. The study recommended a nationally representative case-control study of lung cancer in relation to smoking and occupation. Downward trends in U.S. mortality from cancers of most sites in men and women aged under 65 were reported, which could not be explained by improved treatment. There are downward trends in Britain for lung cancer mortality in men up to age 70 and women up to age 50, compared with downward trends in the U.S. for men up to 50 and a scarcely noticeable trend in women. The downward trends in both countries may be explained by decreasing tar yields of cigarettes, while the differences may be due to different past patterns of cigarette consumption. See also 81-1414.

82–0397. Sabadie, N.; Richter-Reichhelm, H. B.; Saracci, R.; Mohr, U.; Bartsch, H. **Inter-Individual Differences in Oxidative Benzo(*a*)pyrene Metabolism by Normal and Tumorous Surgical Lung Specimens From 105 Lung Cancer Patients.** *International Journal of Cancer* 27(4): 417-425, April 1981.

Oxidative benzo(*a*)pyrene (BaP) metabolism was studied in lung cancer patients undergoing surgical resection. Aryl hydrocarbon (BaP) hydroxylase (AHH) activity was determined in both normal and tumorous lung tissue specimens from the same patient. A more than 20-fold inter-individual variation in AHH activity was found in both the normal and the tumorous lung tissue samples investigated. The apparent K_M of the pulmonary enzyme ($1\text{-}2.5 \times 10^{-4}$M) was identical in tumorous and normal tissue. A positive correlation was found between the AHH activity in normal and tumorous tissue from all cancer patients ($r=0.24$; $p<0.05$). The ratio (R) of AHH activity in tumorous tissue to that in normal tissue from the same lung cancer patient was <1 in 73 patients, 1 in 2 patients, and >1 in 11. In 7 of 10 subjects who underwent surgical resection for suspected lung tumors, but in whom histological analysis revealed no malignant tissue, R was >1 for the AHH activity in the inflammatory tissue to that in normal tissue. In a subset of 43 patients with squamous-cell carcinoma, the same distribution was seen; in those patients, AHH activity in tumorous tissue correlated negatively ($r=-0.18$; $p>0.10$) with the number of cigarettes smoked per day prior to surgery. No differences were observed between the mean values for AHH activity in cases of squamous-cell carcinoma and those of adenocarcinoma. Whether differences in pulmonary AHH activity represent a host risk factor in persons who smoke remains to be investigated. (Auth. Abs. Mod.)

82–0398. Schraufnagel, D.; Peloquin, A.; Pare, J.A.P.; Wang, N.-S. **Differentiating Bronchioloalveolar Carcinoma From Adenocarcinoma.** *American Review of Respiratory Disease* 125(1): 74-79, January 1982.

The recognition of bronchioloalveolar carcinoma (BAC) as distinct from adenocarcinoma of the lung, is controversial. Using strict pathologic criteria, 43 consecutive patients with BAC were matched by year of diagnosis and compared with a similar number of patients with adenocarcinoma, and for contrast, with those with squamous and oat cell carcinoma of the lung. It was demonstrated that BAC is not sex-related, and is not as smoking-related as the other neoplasms. As a whole the BAC group had fewer smokers and they reported less lifelong cigarette consumption than those in any other lung cancer group. Unlike epidermoid carcinoma, BAC does not show a predilection for those occupations requiring manual labor. Also, BAC is frequently distinguishable radiologically from the other three by being smaller and peripheral. A pleural tag and an air bronchogram in a mass are rather specific, and BAC is less likely to have large airway involvement and adenopathy. The percentage of patients who were free of tumor after 2 years was greater in the BAC group than in the others, but the overall survival rate between the BAC group and the adenocarcinoma group was not. Based on inter-observer variability, there is some overlap pathologically between these two groups. However, when the overlap between the adenocarcinoma and the BAC groups is compared with that between the adenocarcinoma and the squamous cell carcinoma groups, the difference is not significant. It is concluded that BAC should be considered a distinct clinical entity. (Auth. Abs. Mod.)

82–0399. Shapiro, L. **Warning: Chewing Tobacco and Snuff May Be Dangerous To Your Health.** *Coal Age* 86(12): 74-79, December 1981.

Miners and others who chew tobacco or dip snuff are at greater risk of acquiring oral cancer or other diseases of the mouth, which indicates smokeless tobacco consumption should be considered a health hazard. Of the 18,400 males who are expected to develop oral cancer in the United States in 1981, 90 percent will have used a form of tobacco that is allowed to accumulate in the oral cavity: cigars, pipes, or smokeless tobacco. The incidence of oral cancer is high in the South, a region where snuff-dipping is a common custom among white women. Chewing and smoking have been found to act synergistically in increasing oral cancer risk. Among the general population, chewing tobacco and snuff consumption rose from 95 million lbs in 1970 to 124 million lbs in 1980, an increase possibly due to the smokeless tobacco offering a less expensive source of nicotine, as well as to advertising. Nitrosonornicotine is believed to be a carcinogen in smokeless tobacco. It may take decades for a chewer or dipper to acquire oral cancer. For miners, chewing tobacco and dipping snuff are a way of consuming tobacco without the fire hazards of smoking. In the 6 out of 10 underground miners and roughly 3 out of 10 surface miners who use chewing tobacco or snuff, the risk of oral cancer may be further compounded by exposure to coal dust. In addition to oral cancer, the use of smokeless tobacco may also cause gums to recede so that teeth become more vulnerable, drift from position, or even fall out.

82-0400. Shekelle, R. B.; Liu, S.; Raynor, W. J., Jr.; Lepper, M.; Maliza, C.; Rossof, A. H.; Paul, O.; Shryock, A. M.; Stamler, J. **Dietary Vitamin A and Risk of Cancer in the Western Electric Study.** *Lancet* 2(8257): 1185-1190, November 28, 1981.

Intake of dietary provitamin A (carotene) was inversely related to the 19-year incidence of lung cancer in a prospective epidemiological study of 1,954 middle-aged men. Intake of preformed vitamin A (retinol) and intake of other nutrients were not significantly related to the risk of lung cancer. Neither carotene nor retinol intake was significantly related to the risk of other carcinomas grouped together, although for men in whom epidermoid carcinomas of the head and neck subsequently developed, carotene intake tended to be below average. The consistency of the epidemiological evidence from diverse populations, the graded nature and temporal sequence of the association, its independence from cigarette smoking, and its coherence with the evidence from animals, all suggest that a diet relatively high in beta-carotene may reduce risk of lung cancer even among persons who have smoked cigarettes for many years. It is emphasized, however, that cigarette smoking increases the risk of other serious diseases, and there is no evidence that dietary carotene affects these other risks in any way. (Auth. Abs. Mod.)

82-0401. Tsokos, C. P.; MacDonald, E. J.; Lee, P. N.; Hirayama, T. **Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer. (Letters).** *British Medical Journal* 283(6304): 1464-1466, November 28, 1981.

Three letters criticize Hirayama's original article on lung cancer among nonsmoking wives of smokers (*British Medical Journal* 282: 183-185, January 17, 1981) and subsequent defense (*British Medical Journal* 283(6296): 914-917, October 3, 1981) on a number of points. The critical χ value of 3.29 reported by Hirayama is still not explained and is unreproducible from his published data. The age stratification is too coarse, and use of husbands' rather than wives' ages compounds the problem. The number of person-years at risk should have been used as the exposure variable, rather than the number of subjects. Inflated estimates of significance were reported because several causes of death were analyzed, but only lung cancer statistics were reported. The areas of Japan selected for study were in every instance related to industrial concentrations, not by random, resulting in influences from asbestos exposure. No discussion was presented on the quantity or toxicity of sidestream smoke inhaled by the wives. Some significant differences claimed in tables II and III of the October 3 letter by Hirayama are due to a statistical error. Hirayama responds to the latter two statements, pointing out that though more research is needed, a study by White and Froeb (*New England Journal of Medicine* 302(12): 720-723, March 27, 1980) suggests a considerable effect on the airways from passive smoke toxicity. Correct values are provided for the tables in error, though the errors do not influence the substance of the previous letter. See also 81-1088, 82-0385, and 80-1190.

82-0402. Weinberg, G. B.; Kuller, L. H.; Redmond, C. K. **The Relationship Between the Geographic Distribution of Lung Cancer Incidence and Cigarette Smoking in Allegheny County, Pennsylvania.** *American Journal of Epidemiology* 115(1): 40-58, January 1982.

The results of the Third National Cancer Survey showed substantial variations in lung cancer incidence rates for white males within Allegheny County, Pennsylvania. To explain these differences, two areas showing over a twofold difference in the 1970 age-adjusted incidence rates (127.7 compared with 59.0 per 100,000) were studied in greater detail. Estimates of smoking experience by age were determined in the two areas by survey sampling methods of white males 35 years of age and older. The results showed the high risk area had a significantly greater proportion of male current smokers and ex-smokers compared to the low risk area. Males in the high risk area also began smoking over 4 years earlier, and proportionally fewer men smoked filter cigarettes. Calculations of lung cancer risk based on these results and a model of risk from the prospective and retrospective studies showed that almost all of the observed difference in risk between areas was attributable to cigarettes. (Auth. Abs.)

82-0403. Wynder, E. L.; Stellman, S. D. **Environmental Factors in the Causation of Bladder Cancer.** In: Connolly, J. G. (Editor). *Carcinoma of the Bladder.* Progress in Cancer Research and Therapy, Volume 18, New York, Raven Press, 1981, pp. 25-36.

Environmental factors important in bladder carcinogenesis, including cigarette smoking, diet, artificial sweetener use, and occupational exposure are examined. It is estimated that, without cigarette smoking, bladder cancer would be reduced by about 40 percent in males and 10 percent in females. Although cigarette smoking is associated with bladder cancer, cigar and pipe smoking are not, suggesting that inhalation plays an important role. There may be a dose-response relationship, the bladder cancer risk increasing as the daily cigarette consumption rises. Those who smoke more heavily, however, may experience a decreased relative risk of bladder cancer, possibly due to less inhalation or to competing risks from other illnesses such as cardiovascular diseases and lung cancer. Smokers of either filtered or unfiltered cigarettes have about the same bladder cancer risk. The nature of cigarette smoke's bladder carcinogens or cocarcinogens remains uncertain. Information on the smoking habits of subjects should be obtained when evaluating the risk associated with occupational exposure, since smoking habits can vary among different occupational groups. It is recommended that further studies be made of the relationship between cigarette smoking and bladder cancer with particular attention to interactions with other environmental exposures.

See also, **82–0312, 82–0406, 82–0409, 82–0528**

NON—NEOPLASTIC RESPIRATORY DISEASES

82–0404. Ames, R. G.; Attfield, M. D.; Hankinson, J. L.; Hearl, F. J.; Reger, R. B. **Acute Respiratory Effects of Exposure to Diesel Emissions in Coal Miners.** *American Review of Respiratory Disease* 125(1): 39-42, January 1982.

No significant differences were found in ventilatory function changes between coal miners exposed to diesel emissions and those not exposed, although changes did occur in miners in both groups who smoked. A study was conducted to determine if acute respiratory effects, measured in terms of changes in forced vital capacity, forced expiratory volume in 1 second, and maximal expiratory flow rate at 50 percent of forced vital capacity, were related to exposure to diesel emissions. Sixty coal miners exposed to diesel emissions and 90 miners not exposed were tested before and after a work shift for ventilatory function changes. Significant work shift decrements in ventilatory function did occur in miners in both groups who smoked cigarettes. Decrements in ventilatory function during the work shift were concentrated in the current smokers, with all three measures showing significant decreases in smokers over nonsmokers and ex-smokers. Smoker-nonsmoker contrasts equaled or exceeded workshift—coal mine dust effect changes for both the exposed and the nonexposed miners. (Auth. Abs. Mod.)

82–0405. Bertram, J. F.; Rogers, A. W. **Recovery of Bronchial Epithelium on Stopping Smoking.** *British Medical Journal* 283(6306): 1567-1569, December 12, 1981.

Bronchial biopsy specimens were studied from 19 patients (five nonsmokers, eight current smokers, and six people who had given up smoking between 2 and 16 years previously). By using a recently developed morphometric technique, the structural state of the bronchial epithelium was assessed and described by a score, which specifies the extent to which the epithelial histology departs from the normal, through the changes of mucous-cell hyperplasia and squamous-cell metaplasia. The epithelium was nearer normal in former smokers than current smokers in each of six pairs matched for smoking history. The numerical scale permitted statistical analysis with a precision that is not possible with data based on subjective diagnosis alone: the results of comparing histology with pack-years of cigarette consumption were consistent with the hypothesis that structural recovery occurs in bronchial epithelium in people who stop smoking for over 2 years. (Auth. Abs.)

82–0406. Betts, T. E.; O'Sullivan, J. P.; Elson, L. A. **Comparative Lung Pathology of Rats After Exposure to Cigarette and Cigar Smoke.** *British Journal of Experimental Pathology* 62(4): 429-435, August 1981.

Additional evidence that cigar smokers are at lower risk for lung cancer is provided by this investigation of the nature of cigar tobacco which showed that exposed rats suffered less health damage from the smoke of air-cured and fermented cigar smoke than from the smoke of flue-cured cigarette smoke. The rapid heat drying during the curing of flue-cured Virginia tobacco (which inactivates the hydrolytic enzymes, producing a high leaf-sugar content) results in acidic cigarette smoke. However, the air-curing of cigar tobacco is a gradual process, resulting in lower sugar content. Sugar content is further reduced when cigar tobacco is fermented. Following exposure to the three types of smoke, rats were measured for lung damage, weight gain, and acute-phase protein increase. Lung consolidation and congestion, measured by increased lung weight, showed that rats exposed to air-cured, fermented cigar smoke had less damage than those exposed to either flue-cured cigarette or air-cured only cigar smoke. Rats exposed to the fermented cigar smoke were essentially the same in appearance as the control rats. Rats exposed to cigar smoke also showed a greater weight gain and a lower acute-phase protein increase than did their Virginia tobacco counterparts. These differences were compounded by the results obtained from measuring changes in the epithelium of the intrapulmonary airways. It is suggested that the different responses to the various types of smoke may be related to the chemistry of the smoke, and that the proportions of irritants, such as acrolein, aldehydes, and phenols, which are present in mainstream smoke, may be involved.

82–0407. Bolduc, P.; Jones, R.; Reid, L. **Mitotic Activity of Airway Epithelium After Short Exposure to Tobacco Smoke and the Effect of the Anti-Inflammatory Agent Phenylmethyloxadiazole.** *British Journal of Experimental Pathology* 62(5): 461-468, October 1981.

Exposure to tobacco smoke increased the mitotic activity in the airway epithelium in male Sprague-Dawley rats, the effect being modulated by the anti-inflammatory agent phenylmethyloxadiazole (PMO). Mitotic activity of extra- and intrapulmonary airway epithelium was studied in male rats exposed to tobacco smoke for 1, 2, 3, 7, or 14 days, with and without addition of PMO to the tobacco. In both control and exposed animals the mitotic index was greater in extrapulmonary regions than intrapulmonary ones. A single exposure to tobacco smoke significantly increased mitotic activity in both airway regions. This initial level of mitotic response was not maintained but was rapidly restored by 1 day free from tobacco exposure: the second peak was as high as the first. Exposure to tobacco plus PMO modified timing and amplitude of the mitotic response. The effect of PMO was somewhat paradoxical since the first peak occurred later, after 2 days of exposure, but the increase was almost twice that seen after tobacco alone. The timing of the second peak was the same as after tobacco alone, but its amplitude was only half. In each experimental group the mitogenic effect was exerted on an intact epithelium. In animals exposed to tobacco alone, or tobacco plus PMO, in extrapulmonary airways mitoses were located mainly in the basal region of the epithelium, whereas in intrapulmonary airways they were located mainly at the mid or superficial level. (Auth. Abs. Mod.)

82–0408. Calverley, P.M.A.; Leggett, R.J.E.; Flenley, D. C. **Carbon Monoxide and Exercise Tolerance in Chronic Bronchitis and Emphysema.** *British Medical Journal* 283(6296): 878-880, October 3, 1981.

Results of a study indicate that concentrations of carboxyhemoglobin frequently found in bronchitic patients who smoke may reduce their tolerance of everyday exercise, possibly by interfering with the transport of oxygen to exercising muscles. The effects of carbon monoxide on exercise tolerance as assessed by the distance walked in 12 minutes were studied in 15 patients with severe chronic bronchitis and emphysema. Each subject walked and would breathe either air or oxygen before and after exposure to sufficient carbon monoxide to raise their venous carboxyhemoglobin concentration by 9 percent. There was a significant reduction in the walking distance when the patients breathed air after exposure to carbon monoxide, and the significant increase in walking distance seen after exercise when breathing oxygen was abolished if carbon monoxide had previously been administered. It was concluded that continued smoking may play a direct part in the reduction of exercise tolerance seen in patients with severe chronic bronchitis and emphysema. Furthermore, the resultant raised carboxyhemoglobin concentration would negate any benefit of exercise from treatment with portable oxygen in such patients. Thus even patients with advanced hypoxic chronic bronchitis and emphysema may derive benefit from giving up smoking with a reasonable expectation of an increased exercise tolerance. (Auth. Abs. Mod.)

82–0409. Chretien, J. **La Fumee de Tabac. Principaux Mecanismes de Toxicite. [Principal Mechanisms of Tobacco Smoke Toxic Effects.]** *Clinical Respiratory Physiology* 17(2): 135-144, 1981, French.

Scientific literature, mostly English language, on the title subject is reviewed to explain why tobacco aerosol is a particularly toxic air pollutant that affects all human defense mechanisms at all levels. The importance of this toxicomania for the generation of obstructive respiratory insufficiency and lung cancer is explained by the combined effects of its highly toxic components and specific inhalation conditions on mechanical purification structures, immune defenses, and enzymatic systems.

82–0410. Chretien, J.; Huchon, G. **Maladies Respiratoires Professionnelles. Mecanismes Elementaires. [Occupational Respiratory Diseases. The Underlying Mechanisms.]** *European Journal of Respiratory Diseases* 62(Supplement 113): 9-23, 1981, French.

The role of particulate air pollutants and their physical characteristics in the development of occupational respiratory diseases is emphasized in this discussion of the underlying mechanisms of these diseases. Among the particulate pollutants noted are antigenic substances, cytotoxic, and carcinogenic compounds. The respiratory risk profile of an individual encompasses genetic risk, history of respiratory illnesses, exposure to other pollutants in addition to the occupational and specific exposure conditions. Examples are cited of potentiation of the bronchial cancer risk in a subject exposed to asbestos and simultaneous inhalation of tobacco smoke, and the manifestation of respiratory intolerance in smokers working in the plastics industry. It is concluded that smoking may potentiate the antigenic effect and cytotoxicity of certain pollutants.

82–0411. Colley, J.R.T.; Brasser, L. J. **Chronic Respiratory Diseases in Children in Relation to Air Pollution.** World Health Organization, Regional Office for Europe, Copenhagen, Euro Reports and Studies 28, 1980, 89 pp.

A close association between air pollution and various respiratory indices in children was found in a study restricted to children to remove or minimize the influence of other factors, such as occupational exposure and cigarette smoking. The study, initiated by the World Health Organization (WHO) Regional Office for Europe, utilized questionnaires and clinical examinations to gather information on the respiratory status of the children. Sulfur dioxide and standard smoke levels were measured in urban areas with varying air pollution levels in Czechoslovakia, Denmark, Greece, the Netherlands, Poland, Romania, Spain, and Yugoslavia. Mathematical analysis of the air pollution and respiratory indices show a strong association between air pollution and various respiratory indices in children, with smoke having a greater effect than sulfur dioxide. Caution is recommended before drawing definite conclusions, however, due to a relatively small number of observations, an uneven spread of air pollution values, and uncertainties about the relationship between prevalence levels found in different countries. A threshold for exposure below which no effects on the respiratory tract occur in children could not be determined, although no apparent effects on the prevalence of respiratory indices were found in some areas with low air pollution; however, it was suggested that initial effects might be found in areas with an annual mean concentration of sulfur dioxide and smoke of 50-200μg/m³. Protocol and procedural study information, dummy tables for pooled analyses, and a list of professional participants are included.

82-0412. Detels, R.; Sayre, J. W.; Coulson, A. H.; Rokaw, S. N.; Massey, F. J., Jr.; Tashkin, D. P.; Wu, M.-M. **The UCLA Population Studies of Chronic Obstructive Respiratory Disease.** *American Review of Respiratory Disease* 124(6): 673-680, December 1981.

Chronic obstructive respiratory disease symptoms and impaired lung function were more prevalent among smokers and nonsmokers in an area of high air pollution than in a less polluted area, with smokers tending to show worse results than nonsmokers in each area. The participants were 3,192 subjects, 25 to 39 years of age, in Lancaster, California, an area with low concentrations of most air pollutants; and 2,369 similar subjects in Glendora, California, an area exposed to high levels of photochemical oxidants, nitrogen dioxide, and sulfates. Respiratory data were gathered by questionnaires, spirometry, and the single-breath nitrogen test. The tests chiefly associated with small airways, as measured by the maximal expiratory flow rates at 50 percent and 75 percent of forced vital capacity below total lung capacity, and by change in the nitrogen concentration between 750 ml and 1,250 ml of expired air, found few or no differences between areas. There was a greater difference between areas than between smoking status in the prevalence of subjects with poor forced expiratory volume in 1 second and/or poor forced vital capacity, and in the mean peak maximal expiratory flow rate and closing volume. Overall, the prevalence of symptoms and the closing volume fraction, as well as most spirometric test results, were worse among the Glendora participants for both smokers and nonsmokers. It was suggested that there was an additive, not synergistic, association between smoking and exposure to the higher levels of air pollutants.

82-0413. Drath, D. B.; Shorey, J. M.; Huber, G. L. **Functional and Metabolic Properties of Alveolar Macrophages in Response to the Gas Phase of Tobacco Smoke.** *Infection and Immunity* 34(1): 11-15, October 1981.

Because it is believed that cigarette smoking is associated with an increased susceptibility to bacterial infection, it is thought that cigarette smoke may inhibit the antibacterial mechanisms of the respiratory system. The effect of whole tobacco smoke and the gas phase of tobacco smoke on the metabolism and phagocytic ability of alveolar macrophages was monitored over a 30-day exposure period. It was demonstrated that both the gas phase and whole tobacco smoke induced a weight loss in exposed rats. Alveolar macrophage oxygen consumption was markedly increased by both exposure regimens. Superoxide generation was not affected by whole tobacco smoke exposure but was increased in response to the filtered gas phase. Hexose monophosphate shunt activity was not altered by either treatment. When metabolic alterations were seen in response to the separate exposures, they were seen only after a phagocytic challenge to the macrophage and not when the cell was unchallenged. Neither whole tobacco smoke nor the gas phase had any significant effect on the ability of alveolar macrophages to phagocytize a viable challenge of *Staphylococcus aureus*. Results suggest that many of the metabolic and functional effects of tobacco smoke on alveolar macrophages can be attributed to the gas-phase component of whole tobacco smoke. (Auth. Abs. Mod.)

82-0414. Ejiofor, A.; Hutchison, D.C.S.; Baum, H. **Inhibition of Human Leucocyte Elastase Activity by Cigarette Smoke.** *Bioscience Reports* 1(9): 715-720, September 1981.

An aqueous extract of cigarette smoke can, in vitro, suppress the activity of leukocyte lysosomal elastase, an effect which may be an important variable for the pathogenesis of emphysema in smokers. The in vitro study indicates that inhibition is not due to pH change and it is nonspecific. The smoke solution used was a strong progressive inhibitor of activity in both purified crystalline elastase and in enzyme newly extracted from white blood cells, though the crude enzyme apparently showed a lower sensitivity compared to the crystalline preparation. Another lysosomal enzyme, acid phosphatase, is also inhibited by the cigarette-smoke solution. Acid glycosidases, however, are not affected. It is not certain which component of cigarette smoke was responsible for inhibition of elastase activity. (Auth. Abs. Mod.)

82-0415. Faden, A.; Mendoza, E.; Flynn, F. **Subclinical Neuropathy Associated With Chronic Obstructive Pulmonary Disease.** *Archives of Neurology* 38(10): 639-642, October 1981.

Cigarettes were implicated as potential neurotoxins in the pathogenesis of chronic obstructive pulmonary disease (COPD) in an examination of the cause of this condition. Twenty of 23 patients with COPD showed electrophysiologic evidence of peripheral nerve dysfunction. Abnormalities of sensory nerve conduction were most common, affecting the sural nerve (20 subjects), ulnar nerve (11), radial nerve (8), and median nerve (7). Six subjects had impairment of both sensory and motor nerve function, with the common peroneal being the most frequently affected motor nerve. Clinical signs of neuropathy were found in four patients. Cigarette smoking, expressed as pack-years, was correlated significantly with the electrophysiologic abnormalities. These findings indicate that subclinical polyneuropathy commonly occurs in association with COPD and that this COPD-related neuropathy is correlated with cigarette consumption. It is suggested that a substance or substances in cigarette smoke, such as nicotine, taken on a long-term basis, may be toxic to peripheral nerves. (Auth. Abs. Mod.)

82-0416. Fox, B.; Bull, T. B.; Makey, A. R.; Rawbone, R. **The Significance of Ultrastructural Abnormalities of Human Cilia.** *Chest* 80(6): 796-799, December 1981.

There were no significant differences found in the number of abnormalities of compound cilia or of their microtubular structures in the respiratory tracts in a group of smokers and nonsmokers. Cilia electron microscopic structures were studied from the inferior turbinate of the nose in 22 adults, and in 84 biopsies of the trachea and bronchi from 40 adults. Statistical analysis compared abnormalities from different sites in smokers and nonsmokers, patients with and without chronic pulmonary infections, and those with and without carcinoma of the lung. No significant differences in the number of examined abnormalities were found for any of the three conditions, including smoking status.

82-0417. Friedman, P. J.; Liebow, A. A.; Sokoloff, J. **Eosinophilic Granuloma of Lung. Clinical Aspects of Primary Pulmonary Histiocytosis in the Adult.** *Medicine* 60(6): 385-396, November 1981.

A high incidence of smoking was found among a study group of patients (n = 100) with eosinophilic granuloma of the lung (EG). Smoking histories, available for 69 patients, showed 2 nonsmokers, 12 ex-smokers, and the remaining 55 (80 percent) current smokers, compared to approximately 35 percent smokers in the general population. Clinical history, pathology, and radiology data were gathered, and statistical correlations made. Those with known smoking histories were divided into nonsmokers, and smokers of less than 10 pack-years, 11-25 pack-years, or more than 25 pack-years. When sex was controlled for age, no correlation was found with smoking history. The probability of having presented with any respiratory symptoms correlated weakly but negatively with the pack-years smoked. Almost all asymptomatic EG patients were heavy smokers. The results could be interpreted as showing that the chronic symptoms of heavy smoking submerged the EG symptoms. Smoking history did not appear to be related to radiographic evidence of increased lung volume, nor with the pulmonary function tests conducted except for abnormal gas exchange, which seemed to vary inversely with the amount smoked. No relation was found between smoking and the histologic extent of lesions, although lesion healing was associated with a longer smoking history. When the lesion activity was controlled for age, no significant relationship with smoking was apparent. The higher incidence of smoking among the patients than among the general population remained an unexplained finding.

82-0418. Hankins, D.; Drage, C.; Zamel, N.; Kronenberg, R. **Pulmonary Function in Identical Twins Raised Apart.** *American Review of Respiratory Disease* 125(1): 119-121, January 1982.

Genetic factors were found to be important in determining susceptibility to airway obstruction from cigarette smoke in this Minnesota Study of Twins Raised Apart. Pulmonary function was measured in 15 pairs of monozygotic twins and one set of monozygotic triplets who were separated at an average age of 3.8 months. Six pairs of twins were concordant for nonsmoking, three pairs and the triplets were concordant for cigarette smoking, and six pairs were discordant for respiratory problems (five pairs for smoking, one pair for asthma). Thirteen pulmonary function tests were done on each twin; however, only two tests of pulmonary function, the $FEF_{25\text{-}75\ percent}$ and V_{75}, could separate smokers from nonsmokers to a statistically significant degree. One additional test, the FEV_1, was the most sensitive way to separate smoking from nonsmoking twins when only paired data from twin pairs discordant for smoking were used. Intratwin differences between twins of a pair for these three pulmonary function tests were small and virtually identical for both concordant groups, whereas the intratwin differences in the discordant group were large and distinctly different from the concordant groups. (Auth. Abs. Mod.)

82-0419. Hedenstierna, G.; Alexandersson, R.; Kolmodin-Hedman, B.; Szamosi, A.; Tollqvist, J. **Pleural Plaques and Lung Function in Construction Workers Exposed to Asbestos.** *European Journal of Respiratory Diseases* 62(2): 111-122, April 1981.

In Sweden, an evaluation of respiratory symptoms and pulmonary function among 423 construction workers exposed to asbestos suggested that smoking may aggravate the effects of asbestos exposure. The workers were divided into three groups. Group I had been exposed to asbestos and displayed pleural plaques. Group II had been exposed to asbestos but displayed no pleural changes. Group III had not been exposed to asbestos and served as controls. The overall effects of asbestos exposure were examined for respiratory symptoms and analyzed by spirometry, airway closure, transfer factor of the lung, and static pressure-volume curve of the lung. The effect of smoking on the response of the lung to asbestos exposure was analyzed by studying smokers and nonsmokers in Group I separately, together with their matched controls. Smokers comprised ongoing smokers and those who had quit smoking during the last year. No significant differences were found in the spirometric variables between exposed and nonexposed smokers. Airway closure, calculated in percent of the expired vital capacity (CV percent), was higher for those who were asbestos exposed ($p = 0.03$). Several spirometric variables did, however, differ significantly between exposed and nonexposed nonsmokers. Also, the CV percent was much higher in the exposed subjects ($p = 0.001$), and the difference was larger than between exposed and nonexposed smokers. The investigation also found that the combined frequency of smokers and ex-smokers in Group I was 78 percent, compared with an expected frequency of about 55 percent in an average Swedish population.

82-0420. Kauffmann, F.; Perdrizet, S. **Effect of Passive Smoking on Respiratory Function.** *European Journal of Respiratory Disease* 62(Supplement 113): 109-110, 1981.

A study conducted on 6,000 men and 7,400 women in France found that 15 years of passive smoking can have a harmful effect on pulmonary function. The subjects were aged 25 to 59 years. Nonsmokers comprised 25 percent of the men and 70 percent of the women. Among these, 18 percent of the men and 54 percent of the women shared the household with a smoker or ex-smoker. Those nonsmokers not sharing a household with a smoker or ex-smoker were termed "true nonsmokers" or NSm. Those who lived with a current smoker at the time of the survey were termed PSm. No significant difference was found between NSm and PSm for either men or women for the age-adjusted forced expiratory volume in the first second (FEV_1) or the age-adjusted flow between 25 percent and 75 percent of expired forced vital capacity ($FEF_{25-75\ percent}$). Spirographic measurements and passive smoking were both related to socioeconomic class and the level of education. As the educational level of the man of the house increased, the percentage of PSm among the women showed a steady decrease. The percentage of PSm among the men showed an initial increase followed by a decrease. The FEV_1 and $FEF_{25-75\ percent}$ according to passive smoking, were adjusted for educational level and compared between NSm and PSm for men and women. There were significant differences for $FEF_{25-75\ percent}$ for both sexes, with larger measurements among the NSm than the PSm. For the FEV_1 readings, the measurements of NSm in each sex were also larger than those of PSm, though the difference was significant only for women. The number of men, however, was small.

82-0421. Leitch, A. G.; Lumb, E. M.; Kay, A. B. **Mediators of Hypersensitivity in the Sputum of Young, Symptomatic Cigarette Smokers.** *Clinical Allergy* 11(3): 257-262, 1981.

An investigation which measured the concentrations of mediators of hypersensitivity among smokers and nonsmokers found a significant increase in sputum histamine and a higher sputum/serum ratio of immunoglobulin E (IgE) in smokers, suggesting that mediators are involved in the bronchial inflammatory response of smokers, as in chronic bronchitis. The study was conducted on 25 young symptomatic smokers and 23 matched nonsmokers with an upper respiratory tract infection. The subjects were measured for sputum and blood eosinophils, IgE, immunoglobulin G (IgG), immunoglobulin A, immunoglobulin M, and sputum histamine. Although sputum histamine and the sputum/serum ratio of IgE were significantly raised in smokers, the nonsmokers had higher serum IgG concentrations. The IgG levels in both smokers and nonsmokers were within the generally accepted normal range, however. The differences were considered to be due to an infectious process. The histamine and IgE data suggest that mediators of hypersensitivity may help determine the subsequent progression of some smokers in developing chronic bronchitis.

82-0422. Lewis, D. J. **Factors Affecting the Distribution of Tobacco Smoke-Induced Lesions in the Rodent Larynx.** *Toxicology Letters* 9(2): 189-194, October 1981.

Tobacco smoke exposure induced laryngeal lesions in rats and hamsters. The rats were exposed for 6 or 12 weeks, at a high dose level of 4 x 10 minutes/day and a low dose level of 2 x 10 minutes/day. The hamsters were exposed for 10 weeks at a high dose level of 2 x 30 minutes/day or a low dose level of 1 x 30 minutes/day. Exposure was for 7 days per week. In both species, there was a nonuniform distribution of epithelial hyperplasia and squamous metaplasia. The changes were less severe for each species in the low-dose groups. The distribution of tobacco smoke-induced lesions is probably influenced by at least four interrelated factors: anatomy, air flow, histology, and the characteristics of the smoke. It is possible that pathological changes could occur in regions which project into, or cause a narrowing of the airway, since these regions might be exposed to a larger concentration of inhaled toxic constituents. The larynx is the first major constriction of the respiratory tract. Lesion distribution can be affected by histological features of the larynx such as epithelial type. The three major epithelial types in the larynx are respiratory, cuboidal, and squamous. Electrostatic charges on smoke particles may influence particle deposition in the larynx.

82-0423. Lippmann, M.; Leikauf, G.; Spektor, D.; Schlesinger, R. B.; Albert, R. E. **The Effects of Irritant Aerosols on Mucus Clearance From Large and Small Conductive Airways.** *Chest* 80(6): 873-877, December 1981.

One-hour inhalation exposures of submicrometer H_2SO_4 droplets by nonsmoking human volunteers indicated that submicrometer H_2SO_4 deposited primarily in the smaller airways slowed mucociliary clearance, while in the larger airways the deposition was smaller and mucus transport increased. The H_2SO_4 exposure was considered to have effects similar to those of air pollution, and of cigarette smoke to a certain extent. The subjects were exposed to concentrations of 0 (distilled water control), 100 $\mu g/m^3$, 300 $\mu g/m^3$, or 1,000 $\mu g/m^3$. The H_2SO_4 doses were administered using two different sized monodisperse γ-tagged Fe_2O_3 test aerosols. The larger sized Fe_2O_3 aerosol, 7.5 μm activity median aerodynamic diameter (AMAD), was deposited primarily in the larger bronchial airways, while the smaller 4 μm AMAD aerosol had a much greater fraction deposited in the smaller and more distal conductive airways. Thoracic retention of the Fe_2O_3 aerosols as a function of time after a brief inhalation was measured with external collimated radiation detectors. At the highest H_2SO_4 exposure, 1,000 $\mu g/m^3$, there was a pronounced transient slowing of bronchial mucociliary clearance of both the 7.5 and 4 μm Fe_2O_3. On the other hand, at the lowest H_2SO_4 concentration, 100 $\mu g/m^3$, there was a marked acceleration of the clearance of the 7.5 μm Fe_2O_3, but a slowing of the clearance of the 4 μm Fe_2O_3. Thus, submicrometer H_2SO_4, which deposits primarily in the distal airways, can slow mucociliary clearance in those airways. In the larger airways, where its deposition is minimal, the H_2SO_4 can, at the same time, accelerate mucus transport. (Auth. Abs. Mod.)

82-0424. Nemery, B.; Moavero, N. E.; Brasseur, L.; Stanescu, D. C. **Changes in Lung Function After Smoking Cessation: An Assessment From a Cross-Sectional Survey.** *American Review of Respiratory Disease* 125(1): 122-124, January 1982.

The evolution and degree of reversibility of various pulmonary function indexes several years after smoking cessation were studied by combining cross-sectional data from ex-smokers with longitudinal information on their smoking history. Cross-sectional lung function data of 51 ex-smokers (mean, 8 ± 7 years since cessation) were compared with those of 54 nonsmokers and 105 current smokers. All subjects were steel workers 45 to 55 years of age. To assess whether lung function indexes improve after smoking cessation, "expected" values were calculated for the ex-smokers, taking into account both duration of smoking and time since cessation. Observed values of forced expiratory volume in 1 second, vital capacity, residual volume, peak expiratory flow rate, slope of the N_2 single-breath washout, and bolus closing volume were significantly different from "expected" ones. This suggested that smoking cessation not only stopped the smoking-induced fast decline in lung function, but even led to some reversal toward nonsmoking values. This was already apparent in 22 subjects who had quit smoking less than 5 years previously. (Auth. Abs. Mod.)

82-0425. Orlandelli, E.; Boncajoni, M.; Fischetto, G.; Milani, L.; Squitieri, C.; Pizzone, G. **The Effect of Cigarette Smoke on Healthy and Asthmatic Subjects Airways.** *Acta Medica Romana* 18(1): 32-39, 1980.

Investigations conducted on healthy and asthmatic individuals found that smoking often reduced various indicators of respiratory function, although such changes were not always statistically significant. A total of 21 healthy subjects, including smokers and nonsmokers, were measured for airway resistance (Raw), specific airway conductance (SGaw), partial expiratory flow volume or PEFV (for 19 subjects), maximum expiratory flow volume (MEFV), and functional residual capacity (FRC), before and after smoking one cigarette. The FRC measurements showed no statistically significant changes after smoking, but all other measurements were significantly worse. The same measurements plus vital capacity (VC) were made for five smokers who had smoked 10 cigarettes in 5 hours. The smoking led to a reduction in SGaw, PEFV, and MEFV, although SGaw showed the only statistically significant change. The FRC, Raw, SGaw, PEFV, and MEFV, before and after smoking one cigarette, were also measured for five asthmatic subjects who had not had therapy for at least 24 hours. The variations, found to be similar to those of healthy subjects, were not statistically significant. Since it was not possible to draw positive conclusions from the data on the asthmatics, further studies on a larger number of cases are recommended.

82-0426. Pearle, J. L. **Exercise Performance and Functional Impairment in Asbestos-Exposed Workers.** *Chest* 80(6): 701-705, December 1981.

A respiratory study of 88 shipyard workers with extensive asbestos exposure found that pulmonary function abnormalities and roentgenographic pleuropulmonary disease were more common among the workers who smoked, although there appeared to be no correlation between smoking history and significant dyspnea. Workers were evaluated for dyspnea, smoking history, X-ray film abnormalities, static pulmonary function, and exercise performance. Smoking history was divided into three groups: those who had never smoked, those who had less than 10 pack-years of smoking (light smokers), and those who had 10 or more pack-years of smoking. Smoking habit characteristics were measured for forced expiratory volume (FEV_1), forced vital capacity (FVC), airways obstruction defined by an FEV_1/FVC ratio, diffusing capacity (DL_{CO}), dyspnea, rales, clubbing, pleural disease, and parenchymal disease. Dyspnea was graded from 0 (no dyspnea on exertion) to 3 (dyspnea at rest or on minimal activity). Pleural and parenchymal diseases were graded from 0 (absent) to 3 (severe). The FVC and FEV_1 were reduced more often in smokers. The FEV_1 difference between nonsmokers and heavy smokers was statistically significant; only one nonsmoker and no light smokers had reduced FEV_1/FVC, while 30 percent of the heavy smokers had airway obstruction. Twenty percent of the heavy smokers had reduced DL_{CO}, compared to one light smoker and no nonsmokers, a significant difference. Rales, pleural disease, and parenchymal disease were more common in smokers. The only worker with clubbing was a heavy smoker. The frequency of grades 2 and 3 dyspnea was roughly similar for all three groups. Large airways obstruction in asbestos workers is thus apparently caused by smoking, although smoking and asbestos exposure may have a synergistic effect. The sensation of dyspnea, however, may depend more on central factors than on functional abnormalities.

82-0427. Pearle, J. L. **Smoking and Duration of Asbestos Exposure in the Production of Functional and Roentgenographic Abnormalities in Shipyard Workers.** *Journal of Occupational Medicine* 24(1): 37-40, January 1982.

Smoking was found to contribute to the functional and roentgenographic abnormalities in 131 asbestos-exposed shipyard workers, in a comparison of duration of asbestos exposure and smoking history. Both exposure and smoking contributed to the frequency of abnormalities in forced expiratory volume (FEV_1) and forced vital capacity (FVC). In contrast, airways obstruction ($FEV_1/FVC < 70$ percent) was unrelated to exposure but correlated closely with smoking. Abnormalities in diffusion capacity were minimally associated with asbestos exposure, but were strongly related to smoking history, suggesting that diffusion impairment in these workers is more likely to be related to smoking and emphysema than to interstitial disease. Significant roentgenographic pleural abnormalities were associated with both duration of exposure and smoking. Interstitial disease did not correlate with exposure and was only mildly associated with smoking. (Auth. Abs.)

82-0428. Pham, Q. T.; Mur, J. M.; Cavelier, C.; Meyer-Bisch, C.; Chau, N.; Huong, N. D. **Indices Fonctionnels Respiratoires: Variabilite, Valeurs de Reference et Pouvoir Discriminant. [Pulmonary Function Indices: Variability, Predicted Values and Discriminating Power.]** *Clinical Respiratory Physiology* 17(6): 949-961, 1981, French.

Flow/volume curves, single-breath N_2 washout curves, single-breath, and steady-state carbon monoxide diffusion tests were performed on 229 firemen (aged 20 to 55 years) and repeated 1 week later on 183 of them. According to a questionnaire, 58 were asymptomatic nonsmokers, 130 were asymptomatic smokers, and 34 were smokers with symptoms of cough with or without sputum. Intra-individual variability of indices determined from tests performed at a week interval is not always aleatory, and it is different in the three groups. It is the same for inter-individual variability which is lowest in asymptomatic smokers and highest in symptomatic smokers. Age and height were the most important factors in inter-individual variability for spirometric indices but do not affect the variability of the slope of N_2 curve (ΔN_2) or fractional uptakes of CO. The ratio of residual variance (adjusted for age and morphometric parameters) on intra-individual variance indicated the choice of "sensitive" functional indices. This choice may be reached in a different way by comparison of means of groups distinguished according to symptoms or smoking habits. Reference equations for indices derived from each test are proposed. The problem of the choice of "sensitive" indices and the problem of reference groups and reference values are discussed. (Auth. Abs.)

82-0429. Rogers, W. R.; McCullough, B.; Caton, J. E. **Cigarette Smoking by Baboons: In Vivo Assessment of Particulate Inhalation Using Bronchoalveolar Lavage to Recover (^{14}C)Dotriacontane.** *Toxicology* 20(4): 309-321, 1981.

An investigation quantitatively demonstrated that cigarette-smoking baboons inhaled particulate matter into the lungs, retaining an average of 9 percent. A bronchoalveolar lavage method for recovery of (^{14}C)dotriacontane was developed. First, nine baboons were exposed to a known dose of (^{14}C)dotriacontane-labeled particulate matter delivered in a manner providing extensive deposition of particulates in the lung. The lungs of these passively exposed animals then were lavaged so that the efficiency of recovery of the standardized lavage procedure could be determined. Second, nine baboons actively smoked labeled cigarettes, and the lungs of these animals were lavaged to recover labeled (^{14}C)dotriacontane. The total amount of particulate matter present in the lungs was estimated using the efficiency factor previously determined. Although average retention was 9 percent, differences among animals were considerable, with inter-animal variability related to differences in number, volume, duration, and pressure of puffs. The retention of particulate matter by baboons is similar to particulate retention by other animal smoke inhalation models. (Auth. Abs. Mod.)

82-0430. Rutishauser, M. **Lungenveraenderungen bei Jugendlichen Rauchern. [Pulmonary Disorders in Juvenile Smokers.]** *Schweizerische Rundschau fuer Medizin (PRAXIS)* 70(38): 1636-1637, September 15, 1981, German.

The maximum expiratory flow vs. volume curves were recorded in 94 smokers, aged 14 to 18 years, and 99 nonsmokers of the same age bracket during forced expiration of the total lung capacity up to residual volume. The teenagers were considered as smokers if they had smoked 10 or more cigarettes/day for a minimum half-year and had inhaled smoke. The curves were recorded while the subjects were breathing air or an 80 percent helium-oxygen mixture. Comparison of the curves obtained with air and helium shows an evident difference between smokers and nonsmokers with respect to the iso-flow point, i.e., the expiratory volume expressed in vital capacity percent at which flow rates of both gases are equal. The average iso-flow point for the smokers, boys and girls, was at 24 percent vs. 5 percent for nonsmokers. Only 12 nonsmokers had an iso-flow point at over 14 percent, probably due to air pollution or a previous virus-caused respiratory disease. Thus, a high percentage of the symptoms-free smokers exhibited functional disorders in small airways, which can be considered as an early indicator of obstructive bronchitis. One year later an increase in iso-flow with increased cigarette smoking was determined in 26 of the smokers. Iso-flow decreased in three smokers after they stopped smoking. It is concluded that the iso-flow method appears to be suitable for detecting disorders and their reversibility in small airways of the smokers.

82-0431. Wanner, A. **Alteration of Tracheal Mucociliary Transport in Airway Disease. Effect of Pharmacologic Agents.** *Chest* 80(6): 867-870, December 1981.

Smokers usually had a lower tracheal mucus transport rate than nonsmokers in an investigation of cigarette smoking and chronic bronchitis. Studies were also made of tracheal mucus transport in cystic fibrosis and bronchial asthma, although the effects of smoking were not mentioned. The relationships among smoking history, respiratory function, and tracheal mucus transport rates were examined in nonsmokers, young smokers, and patients with chronic bronchitis. Although 5 of 13 (38 percent) of young smokers showed rates within the range of age-matched nonsmokers, the rest showed greatly decreased values. Neither smoking history nor pulmonary function distinguished smokers with decreased rates from smokers with normal rates. Among young ex-smokers, five out of nine (56 percent) had rates within the range of age-matched nonsmokers, while the rest showed markedly decreased rates. Neither smoking history nor pulmonary function distinguished ex-smokers with decreased rates from ex-smokers with normal rates. Reduced tracheal mucus transport rate and minimal airway dysfunction appeared to be the earliest indicators of impaired function in cigarette smokers, whether presented together or separately. Chronic bronchitis has been associated with a decreased rate of tracheal mucus transport, although the decrease may not be related to the direct depressant effects of cigarette smoke.

82-0432. Wehner, A. P. Effects of Inhaled Asbestos, Asbestos Plus Cigarette Smoke, Asbestos-Cement and Talc Baby Powder in Hamsters. In: Wagner, J. C.; Davis, W. (Editors). *Biological Effects of Mineral Fibres.* Volume 1. Proceedings of a Symposium held at the International Agency for Research on Cancer, Lyon, France, September 25-27, 1979, IARC Scientific Publications No. 30, INSERM Symposia Series Volume 92, 1980, pp. 373-376.

Chronic exposure of hamsters to chrysotile asbestos resulted in severe asbestosis in all animals and in increased mortality; concomitant exposure to cigarette smoke (three times a day, 5 days per week) decreased body weight but did not affect type or severity of asbestotic lesions. Chronic exposure to asbestos-cement dust increased the number of macrophages and ferruginous bodies. Exposure to talc baby powder caused no significant changes. Deposition of talc particles in the lungs was demonstrated by X-ray fluorescence and X-ray diffraction and by a study with neutron-activated talc. No malignant tumors were observed in any of these studies. (Auth. Abs. Mod.)

82-0433. Wicht, C. L.; Kotze, T.J. van W. Die Invloed van die Rookgewoonte op die Simptome van die Diffuse Obstruktiewe Pulmonale Sindroom. [The Influence of Smoking on the Symptoms of the Diffuse Obstructive Pulmonary Syndrome.] *South African Medical Journal* 60(1): 26-30, July 4, 1981, Afrikaans.

In a randomized sample of the Bellvillé population, a high rate for smoking was found in men (71 percent) and women (38 percent). Of the smokers, 73 percent of men and 42 percent of women started smoking before the age of 21; men were heavier smokers than women. An inverse relationship between social class and smoking was partially shown. A distinct relationship between smoking in men and symptoms of cough, production of sputum, and grade 2 dyspnea was also shown. In women, there was a relationship to the symptoms of morning cough and sputum production but, in contrast to men, not to the symptoms of chronic cough and sputum production relating to chronic bronchitis. It appears that men are more susceptible to chronic bronchitis than women. A total of 32 percent men and 30 percent women had stopped smoking. Various reasons were given for discontinuing or decreasing smoking, a personal decision being of more importance than a doctor's recommendation. (Auth. Abs.)

82-0434. Wilde, W. Mucociliary Clearance of the Slug Mucosa Exposed to Dust and Cigarette Smoke. (Letter). *Medical Journal of Australia* 1(9): 494-495, May 2, 1981.

The adverse effects of cigarette smoke on slug behavior and mucus composition may be useful in assessing effects on the respiratory mucosa in humans, which show similarities in function. Physiological changes in the slug (and snail) are simple and rapid, as in exposure to house dust, which elicits contraction, and a resting period, followed by increased activity. The slug produces a tenacious mucus to embed the dust, and then sheds it like a snake. Microscopic study showed increased ciliary beating and an increase in fibers of acid glycoconjugates in the mucus. A slug exposed to cigarette smoke showed similar changes in behavior and mucus composition: it contracted, rested for a few seconds, and then showed increased activity. Mucus stains again indicated increased amounts of fibers of acid glycoconjugates, which are also seen in the sputum of smokers. In response to an irritant, changes in the mucus of both slugs and humans include changes in fine structure with a marked decrease in the mesh width seen in electron microscopical studies of the basic mucus lattice work.

82-0435. Wu, M.-C. Study on Maximal Expiratory Flow and Volume in Chinese. II. Normal Smoking Adults. *Journal of the Formosan Medical Association* 80(2): 174-182, February 1981.

Maximal expiratory flow (MF) and volume parameters derived from simultaneous maximal expiratory flow-volume and volume-time curves obtained by the forced vital capacity (FVC) maneuver were studied in 88 asymptomatic normal smoking adults, consisting of 75 men and 13 women, 20 to 77 years of age. Ten maximal expiratory parameters—FVC, forced expiratory volume in the first second (FEV_1), FEV_1/FVC, peak expiratory flow rate (PEFR), MF75, MF50, MF25, MF10, MF75/MF50, and MF50/MF25—adjusted for sex, age, and body height, were compared with those obtained in normal nonsmoking adults for detecting the early abnormal ventilatory function. In the all-smoking group, there were 36 cases (41 percent of 88 smokers) with abnormal FEV_1/FVC, MF75/MF50, and/or MF50/MF25. In heavy smokers, 19 cases (51 percent of 37 heavy smokers) had abnormal FEV_1/FVC, MF75/MF50, and/or MF50/MF25. In all smokers, a half of the cases with abnormally low FEV_1/FVC were attributable predominantly to decreased FEV_1 and the other half to increased FVC, while most of the cases with abnormally high MF75/MF50 and MF50/MF25 were predominantly due to decreased MF50 and MF25 respectively. MF75/MF50 and MF50/MF25 were more effective in discriminating between the normal smokers and nonsmokers. The high MF50/MF25 value with decreased MF25 was speculated as the earliest discriminant index in normal asymptomatic smokers. High incidences of abnormally high MF75/MF50 and MF50/MF25 with decrease of MF50 and MF25 reflecting increased time constant in late expiration indicate that there might be a significant portion of asymptomatic smokers having evidence suggestive of small airway abnormality. (Auth. Abs.)

82-0436. Zwilling, B. S. Emphysema, Smoking, and the Alveolar Macrophage. *Immunology Today* 2(8): 155-156, August 1981.

Alveolar macrophages and leucocytes appear to be largely responsible for the generation of emphysema with cigarette smoke exposure having a profound effect on the hemostatic mechanisms of the lung. A current view is that the destruction of lung alveoli is mediated by elastolytic enzymes. A sequence of events underlying the formation of these emphysematous lesions is proposed. The role of cigarette smoke appears critical to the initiation of this sequence by which the macrophage activity is increased with the production of a neutrophil chemotactic factor, the major source of these enzymes. The process is amplified by the production of elastin fragments, through digestion of elastin, which is apparently chemotactic for macrophages. Failure of the antiproteases to control the elastolytic process has been attributed to the action of cigarette smoke which leads to the inactivation of antiprotease activity in lung lavage fluid. Transient imbalance thus created between lung proteases and antiprotease could be sufficient to account for injury to alveolar walls with repeated cycles causing slow destruction of the alveolar walls of chronic smokers.

See also, **82–0374**

CARDIOVASCULAR DISEASES

82–0437. American Heart Association. **Secondary Prevention in Myocardial Infarction Survivors. Joint Recommendations by the ISFC Scientific Councils on Arteriosclerosis, Epidemiology and Prevention, and Rehabilitation.** *Circulation* 65(1): 216A-219A, January 1982.

Recommendations for physicians on the secondary prevention of coronary heart disease, particularly among survivors of myocardial infarction, are provided according to individual risk factors and drugs. Seven published studies which report favorable effects from cessation of smoking after myocardial infarction are referenced. Results vary in detail, but the studies support these conclusions: (1) the risk of fatal reinfarction or sudden death is reduced by 20 to 50 percent; (2) nonfatal reinfarction may be reduced; and (3) the benefit is apparent in the first 5 years after myocardial infarction, and the adverse effect of continued smoking may still be dose-related. It is mandatory that physicians should vigorously advise all their patients with coronary heart disease to discontinue all forms of smoking. Success depends on the adoption by all members of the medical team of a committed and informed approach. Health personnel should not smoke. All health-care personnel should be involved in advice about smoking. The potential adverse effects of giving up smoking (such as an increase in weight, depression, or irritability) can usually be prevented by careful counseling; few patients require specialist attention from a physician, dietitian, or psychologist. Other risk factors and drugs discussed are physical activity, hypertension, diet and lipids, diabetes mellitus, beta-blockers, platelet-active drugs, and anticoagulants.

82–0438. Auerbach, O.; Garfinkel, L. **Myocardial Mural Arterial Fibrosis and Cigarette Smoking: A Comparative Study 1955-1960 Versus 1970-1977.** *Bulletin of the New York Academy of Medicine* 57(9): 759-775, November 1981.

Cigarette smokers who died between 1955 and 1960 showed far more advanced fibrous thickening of the blood vessels in the heart wall than did those who died between 1970 and 1977. Differences between the two increased with the number of cigarettes smoked during life. Nonsmokers had the fewest advanced changes, and about the same percentages were found in both the earlier and the later groups. Results suggest that the reduction of advanced fibrous thickening in smokers who died in the later period may be due, at least in part, to the reduced tar and nicotine content of the cigarettes they smoked for the last 5 to 10 years of their lives. No American cigarette mass produced during this period had as high a tar/nicotine content as the brand with the lowest tar/nicotine during the earlier period. The data are consistent with the epidemiologic findings which have shown a 14 percent decrease in coronary heart disease in low tar/nicotine cigarette smokers when compared to high tar/nicotine smokers. (Auth. Abs. Mod.)

82–0439. Beaglehole, R.; Trost, D. C.; Tamir, I.; Kwiterovich, P.; Glueck, C. J.; Insull, W.; Christensen, B. **Plasma High-Density Lipoprotein Cholesterol in Children and Young Adults. The Lipid Research Clinics Program Prevalence Study.** *Circulation* 62(4): 83-92, November 1980.

The decline in high-density lipoprotein (HDL) cholesterol in adolescent males and the postadolescent sex differences in HDL cholesterol levels were investigated. Prevalence data are presented for 1,639 white males and females aged 6 to 25 from six populations of the Lipid Research Clinics Program. The association of HDL cholesterol with age, smoking habits, and anthropometric, clinical chemistry, and nutritional variables was examined by correlation and regression analyses. Although mean HDL cholesterol levels in children aged 6 to 10 were lower in females than in males, HDL cholesterol levels gradually increased with age in females, while in males the mean HDL cholesterol levels declined from 55.9 mg/dl at ages 6 to 10 to 45.5 mg/dl at ages 18 to 25. In young adults, HDL cholesterol levels were an average of 10.5 mg/dl higher in females than in males. In the 6- to 10-year-old age group, none of the study variables was significantly associated with HDL cholesterol. In the 11- to 17-year-old subjects, significant associations were noted with several variables, especially in the males, such as changes in gonadal hormone levels showing the most important influence on HDL cholesterol levels. The strongest association was an inverse association with weight in males. In the young adults aged 18 to 25 years, significant inverse associations were found with weight and alkaline phosphatase in both sexes, and with sucrose and smoking in females. The consistent inverse associations between smoking, sucrose intake, and HDL cholesterol indicate that the well-documented effects of these variables on HDL cholesterol levels in older adults are already apparent in adolescents and young adults. It is therefore recommended that further studies be made of the effects of smoking cessation and limitation of sucrose on HDL cholesterol in young adults. (Auth. Abs. Mod.)

82–0440. Bing, R. J.; Sarma, J.S.M.; Weishaar, R.; Rackl, A.; Pawlik, G. **Biochemical and Histological Effects of Intermittent Carbon Monoxide Exposure in Cynomolgus Monkeys (*Macaca fascicularis*) in Relation to Atherosclerosis.** *Journal of Clinical Pharmacology* 20(8/9): 487-499, August-September 1980.

Indications are that there is no association between periodic carbon monoxide exposure and the development of atherosclerosis in cynomolgus monkeys. Animals were exposed to 200 to 400 ppm carbon monoxide (CO) in the inspired air for 10 alternate half hours daily for approximately 12 months. These conclusions are reached from both histological and biochemical studies (aortic cholesterol content, arterial cholesterol influx, aortic oxygen consumption, as well as plasma triglyceride concentrations and high-density lipoprotein cholesterol to total cholesterol ratios). A rise in plasma and tissue free fatty acid (FFA) was observed in the experimental group exposed to carbon monoxide. However, the increase in FFA tissue levels was not believed to be due to any inhibitory effect of CO on the electron transfer system since mitochondrial respiratory function was not depressed. (Auth. Abs.)

82–0441. Christlieb, A. R.; Warram, J. H.; Krolewski, A. S.; Busick, E. J.; Ganda, O. P.; Asmal, A. C.; Soeldner, J. S.; Bradley, R. F. **Hypertension: The Major Risk Factor in Juvenile-Onset Insulin-Dependent Diabetics.** *Diabetes* 30(Supplement 2): 90-96, 1981.

Smoking was evaluated for its role in hypertension- and coronary-related deaths in diabetics, and was not found to influence mortality significantly. In a study of the relation of deaths of diabetic patients due to renal disease or coronary disease, and its relation to hypertension, smoking factors were considered. Hypertension is more prevalent among diabetics over age 24 than in the general population and was the major risk factor in death from renal disease. In insulin-dependent diabetics with juvenile onset, hypertension appeared between 20 to 30 years of age; and only one patient with hypertension was among survivors after 40 years of diabetes. Smoking in the group of 40-year survivors is similar to that in the general population and does not appear to be a significant risk factor in diabetic patients dying of coronary heart disease without hypertension.

82–0442. Ernst, E. **Weniger Postoperative Tiefe Venenthrombosen bei Rauchern: Protektiver Effekt des Zigarettenrauchens? [Lower Incidence of Postoperative Deep Vein Thrombosis in Smokers: Protective Effect of Cigarette Smoking?]** *Munchener Medizinische Wochenschrift* 123(35): 1279, August 28, 1981, German.

Several hypothetical explanations are offered for the statistically significant findings of alleged reduction in the risk of postoperative deep vein thrombosis (DVT) in cigarette smokers compared with nonsmoking patients. The up-to-date unexplained findings by different authors were obtained from patients suffering from a variety of conditions. Pipe or cigar smoking did not have the same effect as cigarette smoking. Explanation of the controversial findings could contribute to an understanding of DVT etiology or could indicate the method of DVT prevention.

82–0443. Foster, J. R. **Arrhythmogenic Effects of Carbon Monoxide in Experimental Acute Myocardial Ischemia: Lack of Slowed Conduction and Ventricular Tachycardia.** *American Heart Journal* 102(5): 876-882, November 1981.

Carbon monoxide (CO) may not be arrhythmogenic during early minutes of acute myocardial ischemia. To study the pathophysiologic effects of cigarette smoking and CO on the ischemic heart, 14 dogs underwent thoracotomy and recording of bipolar epicardial electrograms during 6-minute left anterior descending (LAD) coronary artery occlusions. The epigrams showed no change in degree of ischemic myocardial conduction slowing after CO, and no significant difference between CO and non-CO LAD occlusions in incidence of spontaneous ventricular tachycardia. Thus CO, at clinically encountered carboxyhemoglobin levels, may not have an arrhythmogenic effect of epidemiological importance in relation to arrhythmias or sudden death during initial minutes of myocardial ischemia.

82–0444. Gilbert, H. S. **Diagnosis and Management of Polycythemias.** *Comprehensive Therapy* 7(5): 15-26, May 1981.

Smoking appears to adversely affect the condition of polycythemia, defined as an increased concentration of erythrocytes in the circulating blood. Polycythemia may be indicated by elevated hemoglobin content, red blood cell count, and hematocrit. If there is a borderline hematocrit elevation to levels below 55 percent, the patient's history should be checked for smoking, among other factors. Smoking can have a diuretic effect, a stimulus reversible within 72 hours. Those who show a hematocrit of 55 percent or more should be handled at once. Smoking and other contributory factors should be corrected or removed. Relative polycythemia patients have a normal red cell mass in the presence of a polycythemic hematocrit level. The majority of such patients tend to be smokers. Smoking can reduce plasma volume and may cause erythrocytosis. Erythrocytosis or absolute polycythemia is diagnosed when the observed red cell mass is 25 percent greater than predicted. Isolated erythrocytosis can occur as compensation for generalized tissue hypoxia. The tissue hypoxia can be contributed to by carboxyhemoglobin, the levels of which can be greater in smokers. An initial evaluation of inappropriate erythrocytosis can involve a careful search for causes of localized, renal tissue hypoxia, in view of the kidney's role as the tissue oxygen sensor of the body. There also can occur erythrocytosis with stigmata of myeloproliferative disease. Polycythemia should receive careful evaluation and appropriate management.

82-0445. Gillespie, M. N.; Diamond, L. **Spasmogenic Activity of Tobacco Smoke Components on Coronary Arteries: A Possible Link Between Tobacco Smoking and Ischemic Heart Disease.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco Health Research Institute, University of Kentucky, Lexington, November 1-2, 1979. 1980, pp. 212-232.

It is proposed that the association between tobacco smoking and ischemic heart disease may involve the ability of nicotine or other tobacco smoke components to influence those normal homeostatic mechanisms which are responsible for regulating the coronary circulation and, in doing so, initiate coronary artery spasm. Various mechanisms thought to underlie the development of coronary vasospasm are discussed in terms of possible interactions with tobacco smoke components. Evidence is presented demonstrating that in isolated canine coronary arteries, nicotine lacks the ability to induce spasm directly, to influence coronary adrenergic nerve terminals, or to modify coronary spasm initiated by adrenergic agents. These results do not preclude the possibilities that nicotine or other tobacco smoke components may influence coronary prostaglandin biosynthesis to initiate spasm. Furthermore, the possibility remains that tobacco smoke components such as cadmium or carbon monoxide may interfere with coronary smooth muscle cell function by altering the cellular mechanisms of calcium regulation which sets the stage for the development of coronary artery space. (Auth. Abs.)

82-0446. Glueck, C. J.; Taylor, H. L.; Jacobs, D.; Morrison, J. A.; Beaglehole, R.; Williams, O. D. **Plasma High-Density Lipoprotein Cholesterol: Association With Measurements of Body Mass. The Lipid Research Clinics Program Prevalence Study.** *Circulation* 62(4): 62-69, November 1980.

Relationships between weight, height, and ponderal indexes and plasma high-density lipoprotein (HDL) cholesterol and triglyceride were assessed by the Lipid Research Clinics Prevalence Study for 6,865 white children and adults (3,517 males and 3,348 females). Overall, weight, weight/height, weight/height^2, and weight/height^3 were significantly and inversely associated with plasma HDL cholesterol levels and positively associated with plasma triglyceride levels. When the relationships of Quetelet index (weight/height^2) to HDL cholesterol were assessed by multiple regression analysis, including the explanatory variables of cigarette smoking, alcohol intake, and exogenous estrogen hormone use, the Quetelet index was significantly and inversely associated with HDL cholesterol levels in children (by age 12-16 years) and adults of both sexes. After covariance adjustment for smoking, alcohol intake, hormone use, and age, mean HDL cholesterol levels for individuals at the Quetelet tenth percentile were 3 mg/dl higher than for those at the fiftieth percentile, and these in turn were 3-4 mg/dl higher than for those at the ninetieth percentile. These differences in HDL cholesterol between the low, intermediate, and high percentiles of Quetelet index corresponded to the range of HDL cholesterol differences reported to be related to coronary heart disease mortality. Improved approaches to the primary prevention of atherosclerosis may be realized by a better understanding of the inverse relationship of body mass to HDL cholesterol levels. (Auth. Abs.)

82-0447. Goldbourt, U.; Keys, A. **Diet and All-Causes Death Rate. (Letters).** *Lancet* 2(8254): 1047-1048, November 7, 1981.

The conclusion that the amount of saturated fats in the diet is a major factor in predicting coronary heart disease (CHD) mortality (*Lancet* 2(8237): 58-61, July 11, 1981) is criticized. It is argued that the findings of correlations between lipids and CHD deaths are very small compared to the contributions of serum cholesterol, blood pressure, and cigarettes. It is not possible to conclude that a raised high density lipoprotein (HDL) cholesterol level increases the risk of early myocardial infarction, and that cigarette smoking is associated with a decreased risk. In response, Keys states that the purpose of the paper was not to predict the fate of an individual from his eating habits, but rather to show that consideration of average diets of the cohorts accounts for a substantial part of the variance in death rates, and to demonstrate that a low coronary death rate is not necessarily offset by high mortality from other causes. No suggestion that HDL cholesterol and smoking are dominating risk factors without regard to each other or to other variables occurred. See also 82-0458.

82-0448. Greenhalgh, R. M. **Mechanical and Biochemical Factors Influencing Arterial Reconstruction.** *Annals of the Royal College of Surgeons of England* 63(6): 399-404, November 1981.

There are many factors which contribute to the outcome of arterial reconstruction—the site and extent of disease and its rate of progression as well as mechanical and biochemical factors. Mechanical influences are described in 10 patients in whom dilatation of their Dacron graft occurred between 1.4 and 4.5 years after reconstructive surgery. The outcome of arterial reconstruction has also been examined in relation to a number of biochemical risk factors. Only carboxyhemoglobin levels (associated with cigarette smoking) were higher among those patients whose reconstructions failed during a 5-year followup compared with those whose reconstructions remained patent. There is little evidence that dietary advice is merited, but all patients should stop smoking cigarettes before elective arterial surgery. (Auth. Abs.)

82–0449. Haines, C. M.; Ward, G. W. **Recent Trends in Public Knowledge, Attitudes, and Reported Behavior With Respect to High Blood Pressure.** *Public Health Reports* 96(6): 514-522, November-December 1981.

Growing public awareness of the consequences of high blood pressure and the role of factors such as smoking has been shown through surveys conducted during the 1970s. The results may help evaluate the effectiveness of previous health education efforts and identify future needs. Information from hypertensives and from the general population reveals how they feel about their health; their sources of health information; how many believe high blood pressure to be a very serious disease; personal definitions of hypertension and high blood pressure; perceived causes of high blood pressure; and how many believe high blood pressure is a likely cause of heart trouble, stroke, or kidney trouble. Reported prevalence of high blood pressure by demographic characteristics has also been surveyed. Smoking was one of the growing publicly perceived causes of high blood pressure: of volunteered responses, 4 percent identified smoking as a cause in 1973; 10 percent in 1979. When selecting causes from a list, the percentages who chose smoking were much higher. Among hypertensives, those whose reportedly prescribed treatment included the reduction or cessation of smoking rose from 3 percent in 1973 to 9 percent in 1979. Hypertensives may be encouraged to quit smoking, since cigarette smoking is a major co-risk factor in cardiovascular disease. The percentage of hypertensives who continued to smoke actually changed little, remaining at about 29 percent; however, a larger proportion of hypertensives quit smoking than did the general population (47 percent and 35 percent, respectively, in 1979). Such surveys provide useful information and also offer further challenges in high blood pressure control.

82–0450. Harris, J. E. **Cigarette Smoking and the Decline in Coronary Heart Disease Mortality.** In: Havlik, R. J.; Feinleib, M. (Editors). *Proceedings of the Conference on the Decline of Coronary Heart Disease Mortality.* National Heart, Lung and Blood Institute, National Institutes of Health, October 24-25, 1978, NIH Publication No. 79-1610, 1979, pp. 212-214.

The decline in recent years in the prevalence of smoking and coronary heart disease (CHD) mortality among older males is reported. However, although cigarette smoking prevalence has declined, average cigarette intake among current smokers has increased. Data collected from 1964 to 1975 on cigarette consumption among male and female former smokers (grouped as less than 5 cigarettes/day, 5-14, 15-24, 25-34, 35-44, and 45 or over) suggested that the fraction of former heavy smokers increased over time. The increase in cigarette intake by current smokers may therefore indicate changes in the habits of the remaining smokers or the introduction of new smokers. It is suggested that CHD mortality rates for former smokers may take up to 5 to 10 years to decline to the rates of nonsmokers. A large majority of male former smokers (aged 35 to 54 in 1966) stopped smoking between 1966 and 1970. After 1970, CHD mortality in this age—sex group declined, although the mortality decline may be coincidental.

82–0451. Haskell, W. L.; Taylor, H. L.; Wood, P. D.; Schrott, H.; Heiss, G. **Strenuous Physical Activity, Treadmill Exercise Test Performance and Plasma High-Density Lipoprotein Cholesterol. The Lipid Research Clinics Program Prevalence Study.** *Circulation* 62(4): 53-61, November 1980.

During the Lipid Research Clinics North American Prevalence Study, plasma lipoprotein determinations and treadmill exercise testing were performed on 2,319 white men and 2,067 white women ages 20 years or older randomly selected from population surveys by nine clinics in the United States and Canada. Before exercise testing, participants were asked if they performed any strenuous physical activity on a regular basis. Data were analyzed to determine the relationship of plasma high-density lipoprotein (HDL) cholesterol to treadmill exercise test performance and a self-report of strenuous activity. Neither treadmill exercise test duration nor heart rate response to submaximal exercise was significantly related to HDL cholesterol levels for either men or women. However, participants who reported some strenuous physical activity generally had higher HDL cholesterol levels than those who reported none, and the more active men ages 30-49 years and active women ages 20-39 had significantly higher values ($p<0.05$). It was noted that very active men and women smoked cigarettes less often than less active subjects at ages 40-49 years (men, 26 percent vs. 34 percent; women, 21 percent vs. 32 percent), as did very active men at ages 50-59 years (26 percent vs. 34 percent). When HDL cholesterol was adjusted for age, body mass index, alcohol use, cigarette smoking, and interclinic population variation, more active men (41.7 vs. 45.2 mg/dl; $p=0.0001$) and more active women (59.6 vs. 57.7 mg/dl; $p=0.02$) had higher HDL cholesterol levels than their sedentary counterparts. Thus, the association between HDL cholesterol and reported physical activity was, at least in part, independent of other factors that influence HDL cholesterol concentration, but was not associated with exercise tolerance as determined by treadmill exercise testing. (Auth. Abs. Mod.)

82–0452. Hickey, N.; Mulcahy, R.; Daly, L.; Bourke, G.; Graham, I. **Trends in Cigarette Smoking Among Coronary Patients Between 1961 and 1975.** *Cardiology* 68(3): 180-187, 1981.

In Ireland between 1961 and 1975 a study of 663 male patients with acute coronary heart disease found a decline in the proportion of cigarette smokers and an increase in the proportion of ex-smokers and nonsmokers. The proportion of current smokers decreased from 87.1 percent in the 1961-1966 period to 66.1 percent in the 1972-1975 period. The ex-smokers increased from 9.1 percent to 22.2 percent, and the nonsmokers from 3.8 percent to 11.7 percent. Changing diagnosis, altered prevalence of other risk factors, and social class changes in patient referral would not appear to have accounted for the observed smoking trends. It is suggested that the observed trends in cigarette-smoking habits may be real and that they may represent a change in the epidemiological pattern of coronary heart disease in Ireland. (Auth. Abs. Mod.)

82-0453. Hirsh, P. D.; Campbell, W. B.; Willerson, J. T.; Hillis, L. D. **Prostaglandins and Ischemic Heart Disease.** *American Journal of Medicine* 71: 1009-1026, December 1981.

Smoking and other risk factors may promote cardiovascular diseases such as atherosclerosis at least partly through alterations in the production and actions of prostaglandins. Other risk factors include hyperlipidemia, diabetes mellitus, hypertension, sex hormones, age, heredity, emotional stress, and diet. Their effects may be partly due to an imbalance between thromboxane A_2 (TxA_2) and prostaglandin I_2 (PGI_2). There is evidence to suggest such an imbalance is caused by smoking. First, smoking increases platelet activity as well as pro-aggregatory prostaglandin production. Second, smoking potentiates the effects of hyperlipidemia on platelet aggregability. Third, nicotine inhibits the ability of the coronary arteries to synthesize PGI_2-like substances in vitro, an effect that is more pronounced in vascular tissue from smoking patients than from nonsmoking controls. Smoking can alter prostaglandin production and platelet function in potentially harmful ways, possibly promoting cardiovascular disease.

82-0454. Hjermann, I.; Holme, I.; Velve Byre, K.; Leren, P. **Effect of Diet and Smoking Intervention on the Incidence of Coronary Heart Disease.** *Lancet* 2(8259): 1303-1310, December 12, 1981.

Cessation of smoking and the lowering of blood lipids by a change in diet appeared to reduce significantly the incidence of coronary heart disease (CHD) in a study group of healthy but high-risk middle-aged men. A total of 16,202 men, aged 40-49 years, were screened for coronary risk factors. Of these, 1,232 healthy, normotensive men at high risk of CHD were selected for a 5-year randomized trial to show whether lowering of serum lipids and cessation of smoking could reduce the incidence of CHD. Men were admitted to the trial if they had serum cholesterol levels of 7.5-9.8 mmol/l (290-380 mg/dl), coronary risk scores (based on cholesterol levels, smoking habits, and blood pressure) in the upper quartile of the distribution, and systolic blood pressures below 150 mm Hg (mean of two measurements). The men in the intervention group were recommended to lower their blood lipids by change of diet and to stop smoking. Mean serum cholesterol concentrations were approximately 13 percent lower in the intervention group than in the control group during the trial. Mean fasting serum triglycerides fell by 20 percent in the intervention group compared with controls. Eighty percent of the men in both groups smoked tobacco daily at the start of the study. The mean tobacco consumption per man decreased by 45 percent more in the intervention group than in the control group. However, only 25 percent of the smokers in the intervention group completely stopped smoking compared with 17 percent in the control group. Diagnosis of events of cardiovascular disease during the study was made blindly according to predefined criteria by two cardiologists not involved in the study. At the end of the observation period the incidence of myocardial infarction (fatal and nonfatal) and sudden death was 47 percent lower in the intervention group than in the controls ($p=0.028$, two-tailed log rank test). When the incidence of strokes was added, the difference between the groups was still significant. It is concluded that in healthy middle-aged men at high risk of CHD advice to change eating habits and to stop smoking significantly reduced the incidence of the first event of myocardial infarction and sudden death. Statistical analysis, by Cox's proportional hazards model, shows that the reduction in incidence in the intervention group is correlated with the reduction in total cholesterol and to a lesser extent with smoking reduction. (Auth. Abs. Mod.)

82-0455. Holme, I.; Enger, S. C.; Helgeland, A.; Hjermann, I.; Leren, P.; Lund-Larsen, P. G.; Solberg, L. A.; Strong, J. P. **Risk Factors and Raised Atherosclerotic Lesions in Coronary and Cerebral Arteries. Statistical Analysis From the Oslo Study.** *Arteriosclerosis* 1(4): 250-256, July-August 1981.

No significant relationship between cigarette smoking and raised lesions in coronary and cerebral arteries was found during a study of possible atherosclerosis risk factors in Norwegian men aged 40 to 49 years. In addition to the number of cigarettes smoked daily, other risk factors measured included serum cholesterol, serum triglycerides, systolic blood pressure, and diastolic blood pressure. Subjects were also questioned about previous cardiovascular disease, diabetes mellitus, and physical activity at work and at leisure. Suggested reasons for the lack of association between cigarette smoking and the extent of atherosclerosis which was considered somewhat surprising, included (1) a weak relationship between cigarette smoking and coronary atherosclerosis which the study did not detect; (2) smoking habits might be different in Norway than in other parts of the world; or (3) smoking might interact with coronary raised lesions caused by factors other than cigarette smoking. If cigarette smoking does not affect atherosclerotic lesions, the strong clinical association between cigarette smoking and coronary heart disease might be due to the effect of smoking on the myocardium and its conduction system rather than on atherosclerotic lesions.

82-0456. Holme, I.; Helgeland, A.; Hjermann, I.; Leren, P.; Lund-Larsen, P. G. **Physical Activity at Work and at Leisure in Relation to Coronary Risk Factors and Social Class. A 4-Year Mortality Follow-Up. The Oslo Study.** *Acta Medica Scandinavica* 209(4): 277-283, 1981.

In a Norwegian study of the association between physical activity, social class, and coronary risk factors, coronary heart disease (CHD) mortality showed a decrease with increased leisure activity and an increase with increased work activity. The 15,000 subjects screened were apparently healthy men, 40 to 49 years of age, without known cardiovascular disease or diabetes. Serum cholesterol, systolic blood pressure (SBP), and number of cigarettes smoked were associated negatively with physical leisure activity but positively with physical activity at work. Men in lower social classes were less active at leisure but more active at work than men in the higher classes. In a multivariate analysis of variance with coronary risk score (based on SBP, serum total cholesterol, and number of cigarettes), social class, and physical activity, the predictive power of physical leisure activity for future death was almost as good as the coronary risk score. Physical activity at work, on the other hand, was not an independent risk factor either for total or for CHD mortality. (Auth. Abs. Mod.)

82-0457. Hugod, C. **Myocardial Morphology in Rabbits Exposed to Various Gas-Phase Constituents of Tobacco Smoke.** *Atherosclerosis* 40(2): 181-190, October 1981.

An investigation of several gas-phase constituents of tobacco smoke found that carbon monoxide (CO), hydrogen cyanide (HCN), nitric oxide (NO), and carbonyl sulphide (COS) at the levels tested had no significant effect on rabbit myocardial morphology. The study was designed to determine if the gases examined might be atherogenic, using a blind morphological evaluation technique to avoid being misled by preparation or other artifacts. An equal number of test and control rabbits were used, the test animals exposed in air-tight chambers. The test exposures were: 200 ppm CO for 2 and 6 weeks; 0.5 ppm HCN for 4 weeks; 0.5 ppm HCN and 200 ppm CO for 1 week; 0.5 ppm HCN plus 200 ppm CO plus 5 ppm NO for 2 weeks; and 50 ppm COS for 7 weeks. No significant changes in myocardial morphology from exposure to the gases could be demonstrated. It is still not known which constituent or constituents of tobacco smoke are responsible for the increased risk of atherosclerotic cardiovascular disease associated with smoking. It is suggested that future research establish a set of morphological criteria for myocardial damage.

82-0458. Keys, A.; Aravanis, C.; Van Buchen, F.S.P.; Blackburn, H.; Buzina, R.; Djordjevic, B. S.; Dontas, A. S.; Fidanza, F.; Karvonen, M. J.; Kimura, N.; Menotti, A.; Nedeljkovic, S.; Puddu, V.; Punsar, S.; Taylor, H. L. **The Diet and All-Causes Death Rate in the Seven Countries Study.** *Lancet* 2(8237): 58-61, July 11, 1981.

Among 12,763 men aged 0-59, 1,512 died in 10 years, 413 from coronary heart disease. The 16 cohorts differed in all-causes as well as in coronary death rate. Those differences were not related to cohort differences in age, relative weight, activity, smoking habits, or percentage calories from total proteins or fats in the diet but were related to differences in blood pressure, serum cholesterol, and percentage calories from saturated fatty acids. The correlation with saturates was r=0.47 for all causes, r=0.84 for coronary death rate. The all-causes death rate was correlated with saturates even when other dietary variables were controlled in multiple regression. The findings do not prove that saturates in the diet cause increased mortality but are consistent with the hypothesis that risk of early death is increased by diet saturates in populations in which coronary disease is a major death cause. There is no support for the suggestion that the advantage for coronary disease of a diet restricted in saturated fats may be offset by increased noncoronary mortality. (Auth. Abs. Mod.)

82-0459. Landgraf, H.; Ehrly, A. M.; Saeger-Lorenz, K.; Fischer, G. **Erythrocyte Filtration and Smoking.** *Scandinavian Journal of Clinical and Laboratory Investigation* 41(Supplement 156): 199-201, 1981.

Erythrocyte filterability may be influenced by "acute" smoking with subsequent changes of red blood cell (RBC) filterability and by chronic inhalation of cigarette smoke which may lead to chronic alterations. To evaluate chronic alterations of RBC deformability, the erythrocyte filtration rates of 60 young, healthy chronic smokers (30 female, 30 male) were compared with those of 60 young, healthy nonsmokers (30 female, 30 male). The comparison showed a higher filtration rate in the male nonsmokers group than in the group of the male smokers; however, the difference was not statistically significant. Female smokers and nonsmokers did not show any noticeable difference in their RBC filtration rates. (Auth. Abs.)

82-0460. Levy, R. I. **Declining Mortality in Coronary Heart Disease.** *Arteriosclerosis* 1(5): 312-325, September-October 1981.

Although cardiovascular disease became the leading cause of death in the United States by 1930, there has been a decrease in the age-adjusted death rates over the last 30 years. Since 1950, cardiovascular disease death rates have dropped by more than 35 percent; over two-thirds of this decline occurred in the last 10 years. Possible factors that may account for the decline include the development of coronary care units, improved emergency medical services, cardiopulmonary resuscitation, advances in surgical and medical tratment, Medicare, Medicaid, or changes in lifestyle such as less smoking. There is a dose—response relationship between the number of cigarettes smoked by an individual and the risk of cardiovascular disease. Studies indicate that more than 90 percent of the increased risk disappears after 1 year of smoking cessation, and can be almost completely erased after 10 years. Also, since 1963, the per capita tobacco consumption has declined by more than 29 percent. Other risk factors discussed relative to the decline in cardiovascular disease mortality are hypertension, and cholesterol and diet.

82-0461. Mulligan, J. J.; Hojnacki, J. L.; Cluette, J. E.; Kew, R. R.; Stack, D. J.; Nicolosi, R. J.; Flanagan, S. **Cigarette**

Smoke-Induced Depression in LCAT Activity. *Experimental and Molecular Pathology* 35(1): 25-35, August 1981.

The effect of acute inhalation of cigarette smoke on plasma cholesterol esterification by lecithin-cholesterol acyltransferase (LCAT) in atherosclerosis-susceptible White Carneau pigeons was examined. Pigeons were assigned to four treatment groups: (1) Shelf Control fed a chow diet and not exposed to smoke products; (2) Sham pigeons fed a cholesterol-saturated fat diet and exposed to fresh air by the Lorillard smoking machine; (3) low nicotine-low carbon monoxide (LoLo) animals also fed the cholesterol diet and exposed to low concentrations of these cigarette smoke products; and (4) high nicotine-high carbon monoxide (HiHi) birds fed the cholesterol diet and subjected to high concentrations of these inhalants. Both Control and Sham birds had significantly higher LCAT activity (percentage esterification per minute) than HiHi pigeons. Experiments designed to determine whether altered enzyme and/or substrate were responsible for depressed activity revealed no smoke-related modification in substrate efficiency. In addition, Sham and HiHi pigeons had similar concentrations of plasma-free cholesterol, high density lipoprotein (HDL) cholesterol, cholesteryl ester and phospholipid, and similar HDL phospholipid and cholesteryl ester fatty acid profiles. Results demonstrate that acute inhalation of tobacco smoke containing high levels of nicotine and carbon monoxide reduces LCAT activity by two mechanisms: (1) impairment of enzyme efficiency as estimated by in vitro analysis; and (2) in vivo reduction in levels of LCAT cofactor, HDL apoprotein A-I. (Auth. Abs. Mod.)

82–0462. Nissinen, A.; Tuomilehto, J.; Elo, J.; Salonen, J. T.; Puska, P. **Implementation of a Hypertension Control Program in the County of North Karelia, Finland.** *Public Health Reports* 96(6): 503-513, November-December 1981.

A hypertension control project conducted in North Karelia County, Finland, during 1972-1977 consisted of training health personnel, educating the people about hypertension and risk factors such as smoking, and organizing the detection, treatment, and followup of hypertensives. The training given to local health care personnel for participation in the hypertension project was designed to standardize the diagnostic and treatment procedures of physicians, to unify the techniques of clinic nurses, and to promote cooperation among health care workers and program staff. The health education of the public was designed to make people aware of hypertension as a risk factor in cardiovascular diseases, to emphasize the importance of treating high blood pressure, and to publicize the services for screening, treatment, and followup of hypertension. Information was given through newspapers and radio. Health education materials such as leaflets offered information about various topics, including the cessation of smoking, and were distributed chiefly during peoples' visits to health centers for blood pressure measurements. Hypertensives were listed on a central hypertension register. Most of the hypertensives were treated by the staff of the local health centers. Among the duties of the specially trained public health nurses was to use health education to motivate patients to follow treatment and attend followups, to stop smoking, to make dietary changes, and to increase physical exercise. A survey in North Karelia and a reference area found that care of hypertensives was more systematic in North Karelia and that its health care personnel were more satisfied with the cardiovascular disease care given. In North Karelia, 45 percent of the physicians and 34 percent of the public health nurses considered the antismoking effort of their local health centers to be sufficient, compared with reference area figures of 30 percent and 19 percent, respectively.

82–0463. Nitter-Hauge, S.; Erikssen, J.; Thaulow, E.; Vatne, K. **Angiographic and Risk Factor Characteristics of Subjects With Early Onset Ischaemic Heart Disease.** *British Heart Journal* 46(3): 325-330, September 1981.

Sixty-six consecutive patients under 40 years of age with angiographically documented coronary artery disease and coronary heart disease took part in a study aimed at (1) identifying the presence of factors which might explain the premature onset of ischemic heart disease, and (2) assessing the distribution and severity of the coronary artery lesions. For comparison, a study of risk factors in 1,832 men defined as "normals," according to a recent comprehensive examination, were used. Coronary angiography showed a preponderance of one vessel disease. In particular, left anterior descending artery lesions were common, which might have contributed to the early manifestation of disease. The findings show that the typical early onset coronary heart disease case is an overweight, heavily smoking male "blue collar" worker, with high serum levels, a marginally raised blood pressure, and a high prevalence of coronary heart disease among first degree relatives. (Auth. Abs.)

82–0464. Petkyavichyus, R. V.; Grinkevichene, O. K.; Boyarskas, Yu. L.; Shachkute, A. A.; Zaborskis, A. A. **Epidemiologicheskie Issledovaniya Nekotorykh Predvestnikov Ateroskleroza Sredi Shkol'nikov 10—15 Let. [Epidemiologic Study of Atherosclerosis Precursors in 10-15-Year-Old Schoolchildren.]** *Kardiologiya* 20(12): 63-68, 1980, Russian.

Results of the first stage of a study, initiated in 1977, of 2,296 boys and girls selected at random among the schoolchildren in Kaunas, Lithuanian SSR, are presented. Children from two city districts with atherosclerosis precursors were screened, based on the results of medical examinations and a questionnaire survey of the children and their parents. Five precursors including smoking were considered as criteria for screening. The children who had smoked at least once in the preceding 3-month period (7.8 percent of the sample, mostly boys) were considered as smokers for the purpose of screening. Among the screened children, 36.7 percent had one precursor, 14.0 percent—two precursors, and 4.5 percent—three precursors. Precursors distribution of the boys was not significantly different from that of the girls. The children with precursors are scheduled for a more detailed examination and intensive prophylaxis, e.g., group lectures for the smokers.

82-0465. Rawson, G. **Results and Cautionary Implications of a Randomized Controlled Trial of Screening for Risk Indices and Intervention.** *Public Health* 95(5): 276-283, 1981.

A randomized controlled trial, conducted to investigate the benefit of personal health education versus general practitioner referral following screening for selected risk factors (mainly cardiovascular) including smoking, found no apparent benefits on a range of physiological and biochemical variables at 6-month rescreening. Significant decreases in female smoking and alcohol consumption and male obesity were noted as a result of health education. Subjects were divided into three treatment groups: (1) no referral or intervention provided; (2) referral to a health education unit, advice by a doctor, and educational materials with followup visits and counseling; and (3) referral to a general practitioner of the subject's choice, who was provided with copies of the screening results. Smokers in the three groups were asked to keep a log chart, and were advised and encouraged in keeping with the group treatment. However, it is noted that decreases in cigarette smoking may take more than 6 months to become evident through spirograms. There was some evidence of untoward side effects of such screening: (1) more new abnormalities were generated over the 6-month period in the treatment than control groups, (2) the treatment of false positives may not only be wasteful but injurious, and (3) examples of apparent "replacement" of one risk factor by another subsequent to treatment were apparent. It was concluded that individual identification and treatment of risk factors must be approached with extreme caution.

82-0466. Reese, M. E. **Effects of Tobacco Smoke Exposure on the Coagulation System.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco Health Research Institute, University of Kentucky, Lexington, November 1-2, 1979. 1980, pp. 233-258.

Data from rodent, primate, and human experimental studies after exposure to mainstream and sidestream smoke and nicotine injection are used to determine if such exposure can induce a hypercoagulable or thrombophilic response in vivo. Blood samples used were from male Sprague Dawley rats, stumptail monkeys, and human patients who were coal miners with varying degrees of respiratory dysfunction. Effects of mainstream smoke exposure on rats include a loss of body weight, which may be due to the stress of being handled and the restraint during exposure, and a potential hypercoagulable condition. Nicotine administered subcutaneously to rats resulted in a hypercoagulable response. Data from studies of human plasma from both smokers and nonsmokers indicate that smokers in the high-dose group (>45 pack years) exhibit a hypercoagulable response as compared to ex-smokers and nonsmokers. Smoking had no effect on the coagulation system of the stumptail monkeys. In contrast to mainstream exposures, studies show that exposure of rats to high doses of sidestream or passive smoke had no apparent effect on blood clotting parameters.

82-0467. Rhomberg, H. P. **Koronare Risikofaktoren in einer Landlichen Bevolkerung und deren Beeinflussbarkeit innerhalb von 3 Jahren. [Coronary Risk Factors in a Rural Population and the Potential of Risk Reduction After Three Years of Intervention.]** *Wiener Klinische Wochenschrift* 93(16): 3-30, September 4, 1981, German.

A field survey of coronary risk factors, including smoking, was carried out in 1977 among the population of a Tyrolean village, Oberperfuss, near Innsbruck, Austria. Participation of the rural population in the baseline survey was 72.5 percent of the total population aged 20 to 64 years. The primary purpose of the survey was to determine the incidence of the three major coronary risk factors (smoking, hypercholesterinemia, and hypertension); and subsequently to lower the incidence of those factors in the surveyed population with the help of a health education program and local practitioners. Questionnaire responses indicated that in 1977, 43.5 percent of 308 men and 19.6 percent of 326 women had smoked at least one cigarette, cigar, or pipe per day. Two-thirds of the smokers had consumed 16 to 20 cigarettes per day (men), or 11 to 15 cigarettes per day (women); in addition, the men had started smoking earlier in life. The 1980 followup survey of people with risk factors indicated that 9.3 and 9.6 percent of the original male and female smokers became nonsmokers; and the other smokers had reduced their actual nicotine intake, although not significantly.

82-0468. Roskamm, H. **Herzinfarkt im Jugendlichen Alter (unter 40 Jahren): Risikofaktoren, Koronarmorphologie, Prognose und Progression der Koronargefaesssklerose. [Myocardial Infarction in Young Age Groups (Below 40): Risk Factors, Coronary Morphology, Prognosis and Evolution of Coronary Vessels' Sclerosis.]** *Cardiology* 68(Supplement 1): 23-30, 1981, German.

A coronary vessel disease was detected by coronary angiography in 649 male and 30 female patients, all below the age of 40, with diagnosed transmural myocardial infarction. Significantly, 93 percent of the male (and 67 percent of the female patients) had smoked over 10 cigarettes per day compared to only 63 percent of the normal male population. It is concluded that cigarette smoking must be considered as the most frequent and probably the most important risk factor of myocardial infarction at a young age. It follows that cigarette smoking acts by a trigger mechanism, possibly by increasing thrombus formation even in patients with a weak coronary sclerosis. In contrast to other risk factors, smoking is estimated to be a higher risk factor in younger than in older patients.

82-0469. Salonen, J. T.; Puska, P.; Kottke, T. E.; Heinonen, O. P. **Coronary Risk Factor Clustering Patterns in Eastern**

Finland. *International Journal of Epidemiology* 10(3): 203-210, September 1981.

Differences in coronary risk factor clustering between men and women, aged 25 to 59, in a random population sample (n=12,000) from eastern Finland are described. Subjects completed a questionnaire, were interviewed, and physical measurements recorded. Smoking, high saturated fat diet, increased serum cholesterol, and elevated blood pressure showed clustering among men but not among women. Smoking tended to cluster in men with high saturated fat intake and hypercholesterolemia. Increased serum cholesterol clustered with high blood pressure among both men and women. This risk factor clustering may contribute to the exceptionally high coronary heart disease incidence among men in the area. The reverse was true for women: smoking was less prevalent among women with elevated blood pressure or high saturated fat intake and did not differ by serum cholesterol. (Auth. Abs. Mod.)

82-0470. Saloojee, Y.; Cole, P. V.; Clyne, C.; Arch, P. J.; Jarvis, M. J.; Russell, M.A.H. **Nicotine, Carbon Monoxide, and Heart Disease. (Letters).** *Lancet* 2(8254): 1044-1045, November 7, 1981.

The findings of a study by Dr. Wald (*Lancet* 2(8250): 775-777, October 10, 1981) absolving nicotine as a cause of coronary heart disease (CHD) is disputed in three separate replies. The first states that the patterns of exposure to nicotine in inhaling and noninhaling smokers are very different, and nicotine delivery to the heart or other organs cannot easily be predicted. The study also failed to consider the possibility of a synergistic relationship between nicotine and carbon monoxide. The higher mortality from CHD in cigarette smokers may thus be related to an increased myocardial oxygen demand caused by nicotine and a decreased myocardial oxygen supply produced by carboxyhemoglobin (COHb). It is concluded that cigarette smokers are subjected to dual stresses while primary pipe or cigar smokers, who have low COHb concentrations, are not. The second letter supports the contention of Dr. Wald that COHb levels in the blood of smokers may correlate well with the risk of acquiring vascular disease. Data from a study by Clyne and Arch, along with the findings of the study under discussion, suggest that COHb, or a substance as yet unmeasured but with similar blood levels, does provide a sensitive index for the risk of developing vascular disease. The final letter discusses the use of cotinine in nicotine studies and critically examines the data on blood COHb as reported, concluding that the original study does not advance the understanding of whether nicotine, carbon monoxide, or some other constituent of cigarette smoke is responsible for the excess risk of CHD in smokers. See also 82-0477.

82-0471. Schwalb, H.; Eckmann, F.; Bruninghaus, H. **Psychopharmaka und Kardiale Risikofaktoren. [Psychotropic Drugs and Cardiac Risk Factors.]** *Der Nervenarzt* 52(9): 549-553, September 1981, German.

The incidence of the most important cardiac risk factors was studied in 983 long-term hospitalized mental patients treated with psychotropic drugs and compared with 743 similar patients, who had not been treated with psychotropic drugs. The statistically processed data were corrected to exclude the possible effect of age and sex variables. The incidence of risk factors, including smoking of more than five cigarettes/day, was found to be higher in the patients treated with tricyclic neuroleptics exclusively or simultaneously with nontricyclic drugs (mostly butyrophenon). In contrast, the incidence of risk factors in the patients treated exclusively with nontricyclic drugs was lower than in the controls. The higher incidence of cigarette smoking in the patients treated with tricyclic neuroleptics was statistically significant in men only. Obviously, the type of mental disease is also responsible for the higher incidence of a risk factor in the patients treated with tricyclic drugs.

82-0472. Slone, D.; Shapiro, S.; Kaufman, D. W.; Rosenberg, L.; Miettinen, O. S.; Stolley, P. D. **Risk of Myocardial Infarction in Relation to Current and Discontinued Use of Oral Contraceptives.** *New England Journal of Medicine* 305(8): 420-424, August 20, 1981.

A study of myocardial infarction rates among women who were former or are current oral contraceptive users found that rate-ratio estimates increased with duration of former use, suggesting an increased rate of infarction, while rate-ratio estimates declined with increased smoking among former oral contraceptive users. A total of 556 women with infarction, 25 to 49 years old, were compared with 2,036 age-matched controls. Among current users, the overall rate-ratio estimate was 3.5, with no statistically significant trend for duration of current use. For past use, in those who were at least 40 years of age, the rate-ratios increased with duration of use, from 1.0 for past use of less than 5 years, to 1.6 for 5 to 9 years, and 2.5 for 10 or more years, a trend that was considered statistically significant. Among past users (5 or more years) aged 40 to 49, the rate-ratio estimates declined with increased smoking, from 2.5 and 2.9 among nonsmokers and ex-smokers respectively, to 1.5 for those who smoked 1 to 24 cigarettes a day, and 1.4 for those who smoked at least 25 cigarettes a day, a trend considered statistically significant. It was suggested that the study data should be interpreted with caution, until replicated by other studies.

82-0473. Sugrue, D. D.; Thompson, G. R.; Oakley, C. M.; Trayner, I. M.; Steiner, R. E. **Contrasting Patterns of Coronary Atherosclerosis in Normocholesterolaemic Smokers and Patients With Familial Hypercholesterolaemia.** *British Medical Journal* 283(6303): 1358-1360, November 21, 1981.

Cigarette smoking and familial hypercholesterolemia appeared to promote differing patterns of coronary atheroma in a study group of patients. An angiographic comparison was made of the extent and severity of coronary artery disease in 25 patients with heterozygous familial hypercholesterolemia and

25 normocholesterolemic patients with coronary artery disease in whom heavy cigarette consumption was the chief risk factor. The patients with familial hypercholesterolemia were younger and included a much higher proportion of women than the smokers. Significantly more patients with familial hypercholesterolemia had disease of the main stem of the left coronary artery (8 vs. none, $p<0.05$) and triple-vessel disease (18 vs. 4, $p<0.05$). Disease affecting only distal vessels occurred in five smokers, whereas all the patients with familial hypercholesterolemia showed a combination of proximal and distal lesions. These findings suggest that cigarette smoking and familial hypercholesterolemia predispose to different patterns of coronary atheroma. Early coronary angiography with a view to coronary artery bypass surgery seems desirable in symptomatic patients with familial hypercholesterolemia because of the common association of this disorder with life-threatening left main-stem disease. (Auth. Abs. Mod.)

82-0474. Sypczynski, A.; Sterkowicz, S. **Nawyk Palenia Tytoniu u Chorych z Przewlekla Choroba Niedokrwienna Serca. [Smoking Habit in Patients With Chronic Ischemic Heart Disease.]** *Wiadomosci Lekarskie* 33(20): 1611-1615, October 15, 1980, Polish.

The effect of smoking on the incidence of chronic ischemic heart disease was studied in 118 male and female inpatients who had a myocardial infarct (group 1) and 128 male and female inpatients with diagnosed ischemic heart disease but without a history of infarct (group 2). The study was carried out at the Provincial Integrad Hospital in Wloclawek, Poland, using the answers of the former patients to a questionnaire about their smoking habits. The percentage of smokers and ex-smokers among the group 1 patients was significantly higher than in group 2. Over 50 percent of the respondents in both groups knew that smoking was dangerous to their health even before they got sick and only a few percent learned about this health risk after their discharge from the hospital. The infarct incident motivated most of the respondents in group 1 to try stopping or reducing smoking; however, 26 percent continued smoking. Over 70 percent of respondents in both groups cited lack of willpower and force of addiction as the main obstacles to breaking the habit and acknowledged positive effects of smoking cessation on their health.

82-0475. Talbott, E.; Kuller, L. H.; Perper, J.; Murphy, P. A. **Sudden Unexpected Death in Women: Biological and Psychosocial Origins.** *American Journal of Epidemiology* 114(5): 671-682, November 1981.

Multiple regression analysis showed that cigarette smoking, psychiatric history, and death of a significant other were the most important factors contributing to differences between women who died suddenly from arteriosclerotic heart disease and a group of control women. Subjects were 80 women who died from arteriosclerotic heart disease from March 1977 to October 1978, and 80 age-matched controls, all from Allegheny County, Pennsylvania. Detailed examinations (post mortem) were conducted on 42 of the 80 sudden cardiac cases. Cases were more often heavy smokers and had fewer children than the control population. Fifteen of the 80 women who died suddenly of heart disease and three controls had a definite history of psychiatric disease. Cases also had more often experienced the death of a significant other within 6 months prior to their own death. Thirteen women died suddenly under the age of 45; all six deaths due to arteriosclerotic heart disease were of women who smoked.

82-0476. Tatami, R.; Mabuchi, H.; Ueda, K.; Ueda, R.; Haba, T.; Kametani, T.; Ito, S.; Koizumi, J.; Ohta, M.; Miyamoto, S.; Nakayama, A.; Kanaya, H.; Oiwake, H.; Genda, A.; Takeda, R. **Intermediate-Density Lipoprotein and Cholesterol-Rich Very Low Density Lipoprotein in Angiographically Determined Coronary Artery Disease.** *Circulation* 64(6): 1174-1184, December 1981.

A study of 182 patients found positive associations of coronary artery disease (CAD) with very low-density lipoprotein (VLDL) cholesterol, intermediate-density lipoprotein (IDL) cholesterol, and low-density lipoprotein (LDL) cholesterol, as well as with the nonlipid risk factor of smoking. The high-density lipoprotein (HDL) cholesterol showed a negative association. The other nonlipid risk factors measured were age, sex, obesity, glucose tolerance, mean systolic blood pressure, and mean diastolic blood pressure. Among nonlipid risk factors, the IDL cholesterol levels were significantly associated with smoking in men. It is suggested that cigarette smokers have a high incidence of CAD due to a high level of IDL as well as a low level of HDL.

82-0477. Wald, N. J.; Idle, M.; Boreham, J.; Bailey, A.; Van Vunakis, H. **Serum Cotinine Levels in Pipe Smokers: Evidence Against Nicotine as Cause of Coronary Heart Disease.** *Lancet* 2(8250): 775-777, October 10, 1981.

The serum levels of cotinine (a nicotine metabolite) were found to be significantly higher for a study group of male pipe smokers than in male cigarette smokers, cigar smokers, and nonsmokers, indicating that nicotine is probably not the major cause of excess risk of coronary heart disease mortality in cigarette smokers. Serum cotinine and carboxyhemoglobin (COHb) levels were measured in men aged 35-64 years, to learn if the nicotine or carbon monoxide (CO) in tobacco smoke might be the cause of the excess risk of coronary heart disease mortality associated with smoking. Cigarette smokers appear to have substantial excess risk, cigar smokers a smaller excess risk, and pipe smokers little, if any, extra risk. The mean COHb level for nonsmokers was only 0.9 percent, compared with 4.7 percent for cigarette smokers, and intermediate readings of 2.9 percent and 2.2 percent for cigar and pipe smokers, respectively. Although the COHb results were consistent with CO being responsible for excess risk in smokers, the study evidence in favor of such a hypothesis was not considered strong. Some other smoke constituent correlated with CO might be the principal etiological agent. The mean cotinine level was highest in pipe smokers at 389 ng/ml, compared with 306 ng/ml in cigarette smokers, 121 ng/ml in cigar smokers, and no cotinine in nonsmokers. The observation that pipe smokers have high cotinine levels, but little if any excess risk, suggests that nicotine is not the major cause of the excess risk found in cigarette smokers.

See also, **82-0377, 82-0553**

PREGNANCY AND INFANT HEALTH

82-0478. Chemistry and Industry. **Cadmium Toxicity and Smoking Linked.** *Chemistry and Industry* 19: 661, October 3, 1981.

Maternal smoking and cadmium toxicity may be linked in causing changes in the placental blood system. A study of subcutaneous injections of cadmium chloride given to rats during pregnancy found no difference in fetal weights between the exposed and unexposed groups, but the exposed population showed a significantly thicker feto-maternal blood barrier. There also appeared to be a decrease in the volume density of the maternal vascular system. The changes apparently corresponded to those observed in the placentae of smoking women. It was pointed out that the placentae of smoking mothers contain about twice as much cadmium as those of nonsmoking women, due to the cadmium in cigarette smoke. It is possible, however, that similar effects in pregnant women are due to the carbon monoxide present in cigarette smoke. Cadmium may not have a role in the development of prostrate cancer.

82-0479. Cushner, I. M. **Maternal Behavior and Perinatal Risks: Alcohol, Smoking, and Drugs.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 2. Palo Alto, California, Annual Reviews Inc., 1981, pp. 201-218.

Maternal consumption of tobacco, alcohol, and drugs can adversely affect perinatal health. Maternal smoking has been associated with a reduced rate of fetal growth. There also appears to be an increased risk of perinatal mortality for infants whose mothers smoke. Debate continues over whether the adverse influence of smoking is due to direct toxic effects or indirectly from undernutrition. It is suggested that women can be helped to achieve adequate nutrition and cessation of smoking. Smoking may also affect the health of the women themselves. The increase in the lung cancer mortality rate for women appears to be related to the increase in smoking among women. Reductions in tobacco, alcohol, and drug consumption among pregnant women can lead to health improvements for infants and women, as well as families. Attempted reductions should receive input from physicians, nurses, and pharmacists; biomedical, health services, social, and behavioral scientists; program administrators; health policy makers; and those who allocate and manage health care resources.

82-0480. Divers, W. A., Jr.; Wilkes, M. M.; Babaknia, A.; Yen, S.S.C. **Maternal Smoking and Elevation of Catecholamines and Metabolites in the Amniotic Fluid.** *American Journal of Obstetrics and Gynecology* 141(6): 625-628, November 15, 1981.

To assess the effect of maternal smoking on fetal adrenergic activity, simultaneous measurements in amniotic fluid of the parent catecholamines, dopamine (DA), norepinephrine (NE), and epinephrine (E), as well as the specific intraneuronal deaminated metabolites of DA, 3,4-dihydroxyphenylacetic acid (DOPAC), and of NE, 3,4-dihydroxyphenylglycol (DOPEG), were made by radioenzymatic assay. In the second trimester, a significant ($p<0.002$) and selective elevation of the mean DOPEG concentration was noted in the amniotic fluid of smokers ($n=8$) as compared to nonsmokers ($n=36$). In the third trimester, significant elevations were found in the mean amniotic fluid concentration of E ($p<0.0002$) and NE ($p<0.0005$), as well as DOPEG ($p<0.0002$), of smokers ($n=12$) when compared to nonsmokers ($n=12$). There were no significant differences in amniotic fluid concentrations of DA and its deaminated metabolite DOPAC. Since compartmentalization of catecholamines exists between the maternal and fetal circulations, the elevated levels of NE, E, and DOPEG in the amniotic fluid of smokers suggests fetal adrenergic activation as a result of fetal hypoxia and/or by a direct effect of nicotine on the fetal adrenergic system. (Auth. Abs.)

82-0481. Hammer, R. E.; Goldman, H.; Mitchell, J. A. **Effects of Nicotine on Uterine Blood Flow and Intrauterine Oxygen Tension in the Rat.** *Journal of Reproduction and Fertility* 63(1): 163-168, September 1981.

Cigarette smoking during pregnancy has been shown to increase the incidence of abortion, developmental anomalies, and small-for-date infants, possibly by reducing the availability of oxygen within the human uterus, and influencing the conceptus even before it implants. A study was undertaken to determine whether the nicotine-induced reduction in uterine blood flow concomitantly decreases the concentration of oxygen within the uterine lumen. Subcutaneous injection of nicotine (0.5 or 5 mg/kg body weight) resulted in a marked and prolonged reduction in uterine blood flow and intrauterine oxygen tension in pseudopregnant rats. By 10 minutes after nicotine administration (5 mg/kg) uterine perfusion was reduced by 40 percent, remained suppressed for 90 minutes, and returned to the pretreatment level by 120 minutes. Rats receiving the 0.5 mg nicotine/kg also showed a marked reduction in uterine blood flow, although the response was slower in onset and longer in duration. Nicotine (5 mg/kg) also resulted in a sustained decrease in intrauterine oxygen tension from a control value of 48.9 ± 3.6 to 22.2 ± 2.6 mmHg at 45-60 minutes and 21.7 ± 1.5 mmHg at 60-90 minutes. The frequency and amplitude of fluctuations in intrauterine oxygen tension were still reduced by 90 minutes after treatment. (Auth. Abs. Mod.)

82-0482. Hoshino, K.; Hayashi, Y.; Takehira, Y.; Kameyama, Y. **Influences of Genetic Factors on the Teratogenicity of Environmental Pollutants: Teratogenic Susceptibility to Benzo(*a*)pyrene and *Ah* Locus in Mice.** *Congenital Anomalies* 21(1): 97-103, 1981.

C57BL/6 female mice were mated with C57BL/6, DBA/2, or BDF, male mice (B-Groups), and DBA/2 female mice were mated with DBA/2, C57BL/6, or BDF, males (D-Groups). C57BL/6 mice are Ah responsive (aromatic hydrocarbon responsive) and DBA/2 mice are Ah nonresponsive. On day 8 of pregnancy, mice of each group were injected ip with benzo(*a*)pyrene (BaP) in corn oil at a dose of 150 or 300 mg/kg. Untreated and vehicle-treated pregnant mice served as controls. Pregnant mice were killed on day 18 and the fetuses were examined for mortality, growth, external, and skeletal abnormalities. Fetal mortality increased with the dose of BaP in all the experimental groups except the mating group of DBA/2 x DBA/2. The decrease of fetal body weight was dose dependent in all groups, and was more prominent in D-Groups than in B-Groups. The incidence of cervical ribs increased with the dose of BaP in D-Groups but not in B-Groups. These results suggest that the fetuses with Ah responsive genotype were more susceptible to BaP in terms of fetal mortality and the fetuses from the mothers with Ah nonresponsive genotype were more susceptible in terms of the decrease in fetal body weight and the increase in the incidence of cervical ribs. (Auth. Abs.)

82-0483. Keeler, R. F.; Shupe, J. L.; Crowe, M. W.; Olson, A.; Balls, L. D. ***Nicotiana glauca*-Induced Congenital Deformities in Calves: Clinical and Pathologic Aspects.** *American Journal of Veterinary Research* 42(7): 1231-1234, July 1981.

Seven calves born to seven cows fed *Nicotiana glauca* during portions of the first trimester of gestation were deformed at birth. Deformities increased in severity as the calves aged. At birth, calves typically had arthrogryposis of the forelimbs or curvature of the spine; severity varied among calves. In four calves necropsied at about 15 months of age, there was general malpositioning and misalignment of the distal ends of the radius and ulna and the proximal ends of the metacarpal bones. Carpal joints were severely affected, fetlock joints were moderately affected, and pastern joints were slightly affected, with lateral rotation of forelimbs common; severity varied among calves. In one of the four calves, there was moderate torticollis and scoliosis resulting from wedging of some of the cervical and thoracic vertebrae and abnormal cranial curvature of the left thoracic ribs. Histologic changes were not noticed in muscle, brain, spinal cord, or endocrine organs of the four calves. (Auth. Abs.)

82-0484. Kuzma, J. W.; Kissinger, D. G. **Patterns of Alcohol and Cigarette Use in Pregnancy.** *Neurobehavioral Toxicology and Teratology* 3(2): 211-221, Summer 1981.

Maternal smoking and drinking have been implicated as factors in the adverse outcome of pregnancies. This California study of over 12,000 women demonstrates that a large proportion of them smoke or drink during pregnancy. Drinking habits appear to be associated with age, ethnicity, marital status, income, education, church attendance, and prenatal care, but not with gestational age, breast-feeding, or self-reported health. Smoking shows associations with income and education. Drinking and smoking are closely interrelated, showing strikingly parallel associations with such factors as use of caffeine or illicit drugs. Subgroups of pregnant women who exhibited differing drinking habits were found not comparable on certain other characteristics which potentially influence the health of neonates. The importance is reemphasized of utilizing large populations, adjusting for confounding variables, and paying particular attention to smoking habits. (Auth. Abs.)

82-0485. Lehtovirta, P.; Forss, M. **The Acute Effect of Smoking on Uteroplacental Blood Flow in Normotensive and Hypertensive Pregnancy.** *International Journal of Gynaecology and Obstetrics* 18(3): 208-211, 1980.

The acute effect of smoking on uteroplacental blood flow was studied by the ^{133}Xe method in 12 normotensive and 11 hypertensive pregnant women at term. A standard cigarette caused an acute decrease in intervillous blood flow in the normotensives but normalized within 15 minutes. In the other group, smoking produced a progressive increase in this flow, whose basal presmoking value was lower in the hypertensive women. The myometrial blood flow was equal in both groups before smoking but significantly higher in the hypertensive group immediately afterwards. Smoking produced a constant increase in systolic, diastolic, and mean arterial blood pressure in the normotensive women. In the others, diastolic and mean arterial blood pressure increased only temporarily, while systolic blood pressure remained unchanged. Heart rate increased in both groups. Repeated decreases in the intervillous blood flow could explain growth retardation of the fetus and some other complications of pregnancy in normotensive smokers. It is assumed that postischemic reactive hyperemia in the uterine vessels caused the increased flow in hypertensive pregnant women after smoking but it is not recommended as beneficial because of the increased placental transfer of the harmful elements found in tobacco smoke. (Auth. Abs.)

82-0486. Linn, S.; Schoenbaum, S. C.; Monson, R. R.; Rosner, B.; Stubblefield, P. G.; Ryan, K. J. **No Association Between**

Coffee Consumption and Adverse Outcomes of Pregnancy. *New England Journal of Medicine* 306(3): 141-145, January 21, 1982.

Interview and medical-record data of 12,205 nondiabetic, nonasthmatic women were analyzed to evaluate the relation between coffee consumption and adverse outcomes of pregnancy. Low birth weight and short gestation occurred more often among offspring of women who drank four or more cups of coffee a day and more often among the offspring of smokers. The frequency of smoking was over three times greater among heavy coffee drinkers than among women who drank no coffee or tea. More of the heavy coffee drinkers had previously had one or more poor obstetric outcomes, such as a stillbirth or a spontaneous or induced abortion. Heavy coffee drinkers also weighed more. After controlling for smoking (3 or more cigarettes a day at the time of delivery), other habits, demographic characteristics, and medical history by standardization and logistic regression, no relation between low birth weight or short gestation and heavy coffee consumption was found. Furthermore, there was no excess of malformations among coffee drinkers. These negative results suggest that coffee consumption has a minimal effect, if any, on the outcome of pregnancy. However, the findings do add to the data indicating that smoking is associated with poorer pregnancy outcomes.

82-0487. Manchester, D. K. **Range of Environmentally Responsive Monooxygenase Activities in Human Placental Microsomes Determined by Direct Fluorescence Techniques.** *Biochemical Pharmacology* 30(7): 757-762, 1981.

A positive history of maternal cigarette smoking was associated with an increased likelihood of detection of benzo(*a*)pyrene (BaP) metabolism and a nearly fourfold increase in microsomal 7-ethoxyresorufin (ERR) *O*-deethylase activity in this study of human placental metabolism. Two direct fluorometric assays of in vitro xenobiotic oxidation were adapted for use with placental microsomes. A total of 103 placentas were studied. NADPH-dependent (micotinamide-adenine dinucleotide phosphate dehydrogenase-dependent) disappearance of BaP was found in the majority of placental preparations from pregnancies in which maternal smoking had occurred, but was observed only rarely in its absence. *O*-Deethylation of ERR was demonstrable in all cases. *O*-Deethylase activity correlated positively with BaP disappearance ($r=0.87 \pm 0.16$) when mothers smoked cigarettes and was significantly higher than activity in preparations from nonsmokers. A shift in apparent K_m toward ERR from 10^{-5}M to 10^{-7}M was observed as a consequence of maternal smoking. Addition of α-naphthoflavone markedly inhibited *O*-deethylation in induced placentas, whereas stimulation of in vitro metabolism occurred in noninduced placentas. These results establish operational definitions of induction in human placental monooxygenase systems and provide a basis for quantitative descriptions of fetal environmental responses. (Auth. Abs. Mod.)

82-0488. Manchester, D. K.; Jacoby, E. H. **Sensitivity of Human Placental Monooxygenase Activity to Maternal Smoking.** *Clinical Pharmacology and Therapeutics* 30(5): 687-692, November 1981.

Human placental monooxygenase activity is sensitive to both active and passive cigarette smoke exposure. The in vitro oxidation of 7-ethoxyresorufin was studied in placental microsomes from 47 full-term pregnancies. Placental activities were compared in four groups formed on the basis of maternal smoking history as follows: nonsmokers ($n=12$), passive smokers ($n=16$), 1 to 20 cigarettes/day ($n=13$), and 20+ cigarettes/day ($n=5$). Passive smoking was defined as living with someone who smoked cigarettes. Umbilical cord plasma thiocyanate concentrations were measured to confirm histories and were higher when mothers smoked (80±10 µM, 1 to 20 cigarettes/day; 116±8 µM, 20+ cigarettes/day) than when mothers did not smoke (34±3 µM, nonsmokers; 35±3 µM, passive smokers). 7-Ethoxyresorufin *O*-deethylase activity was increased 10- to 30-fold in placentas from smokers. Activity toward 7-ethoxyresorufin in placentas from smokers was markedly inhibited in vitro by addition of 7,8-benzoflavone. Among nonsmokers, percent inhibition of *O*-deethylase activity by 7,8-benzoflavone was greater ($p<0.05$) in placentas from women passively exposed to cigarette smoke. (Auth. Abs. Mod.)

82-0489. New Scientist. **Children Seem to Grow Out of Fetal Smoking.** *New Scientist* 91(1264): 285, July 30, 1981.

Although maternal smoking apparently reduces infant weight, results of this study showed that the offspring catch up in mental and physical development by the age of 10 with children whose mothers did not smoke. Interviews were conducted with 233 mothers (including 100 who had smoked during pregnancy) of 10-year-old children. Babies of mothers who smoked averaged about 200 g lighter than those of nonsmokers. The children were then compared in four categories: physical growth and weight, intellectual functioning and academic achievement, personality, and popularity and social functioning. Results indicated that the 10-year-old children of mothers who had smoked during pregnancy differed little from the others. It is pointed out, however, that the study surveyed only surviving children, and did not cover the possibility that smoking may increase the incidence of miscarriages or make very young babies more subject to illness. Smoking during pregnancy is still not to be encouraged.

82-0490. Phillips, T. C., III. **Effects of Maternal Smoking on the Fetus.** *Journal of the Medical Association of Georgia* 70(11): 787-790, November 1981.

Maternal smoking can give the mother poorer protection against infection, poorer ventilation, decreased respiratory enzyme function, and poorer circulation, which may help compromise the health of the fetus. Maternal smoking may increase perinatal mortality, and cause a reduction in birth weight and height. The incidence of delivery complications can increase. An increased incidence of minimal brain dysfunction has been found in the children of mothers who smoked during pregnancy. A fetus' lungs can also be affected. Elevated levels of carboxyhemoglobin can hurt maternal oxygen transport capacity. Maternal smoking can also lead to increased congenital heart defects and vascular damage before birth. Children of smokers more often smoke, too, and at an earlier age, than children of nonsmokers. Knowledge about the effects of maternal smoking appears to be much less among women than among the scientific community. It is suggested that physicians should inform pregnant patients of the risks of smoking.

82–0491. Vir, S. C.; Love, A.H.G.; Thompson, W. **Zinc Concentration in Hair and Serum of Pregnant Women in Belfast.** *American Journal of Clinical Nutrition* 34(12): 2800-2807, December 1981.

Incidence of congenital malformations of the central nervous system is one of the highest in Belfast and a possible association of this with zinc status has been postulated. Serum and hair levels of zinc were estimated in 28 nonpregnant women and 60 pregnant women at 20 to 22 weeks of gestation (2nd trimester), 36 to 37 weeks of gestation (3rd trimester), and 3 days postpartum. The effect of parity, history of oral contraceptive usage, smoking habit, and alcohol consumption on zinc status were investigated. The mean serum zinc concentration was significantly lower in the pregnant subjects compared to the nonpregnant and showed a continuous significant decline with increasing duration of pregnancy. Mean hair concentration in the various stages of study of pregnant women and nonpregnant women were comparable. However, a progressive decrease in hair zinc concentration was noted with advancing pregnancy; the decline between 2nd and 3rd trimester was statistically significant. Mean serum zinc concentration in umbilical cord blood was approximately twice that in maternal blood. Smoking had no effect on zinc concentration, while the effect of alcohol and parity was inconsistent. Oral contraceptive usage before conception appeared to lower hair zinc concentration and increase serum zinc concentration during pregnancy. Two subjects had spontaneous abortion and these had serum zinc in the lower range and hair zinc values in the higher range. The remaining 58 subjects gave birth to normal neonates weighing 2,500 to 4,500 g. The study revealed that zinc nutrition of the majority of the expectant mothers was adequate for normal growth and development of fetuses. The possible cause and implication of observed changes in zinc metabolism are discussed. (Auth. Abs. Mod.)

See also, 82–0308, 82–0387, 82–0537

OTHER DISEASES AND CONDITIONS

82–0492. Bashir, K. R.; Raman, S.; Knott, V. J.; Bulmer, D. R.; Hurtig, J. B. **Influence of Alcohol and Tobacco Use on Sodium Thiopental Requirements in General Anesthesia.** *Canadian Family Physician* 27: 229-235, February 1981.

Hospital charts of 700 patients who had undergone upper gastrointestinal surgery were reviewed to examine the relationship between alcohol abuse and dose of intravenous sodium thiopental (Pentothal) required to induce general anesthesia. Patients who required a high sodium thiopental dose (greater than 6.08 mg/kg) exhibited a higher incidence of alcoholism, heavy drinking, and heavy smoking, compared to patients who required low sodium thiopental dose (greater than 3.42 mg/kg and less than 4.75 mg/kg). Alcoholics and heavy drinkers, the majority of whom were heavy smokers, required a greater mean sodium thiopental dose than heavy smokers who were neither alcoholics nor heavy drinkers. Possible future research into the apparent cross tolerance between alcohol and sodium thiopental is discussed, particularly the possibility that the family physician may be able to use sodium thiopental dose requirement as a marker for early detection and diagnosis of alcoholism. It is suggested that it would be useful to include a drinking and smoking history on anesthesia records with estimates of respective consumption. (Auth. Abs. Mod.)

82–0493. Bazhanov, N. O.; Salyayev, V. N. **Vliyaniye Kholinomimetikov na Skorost' Razvitiya Gipotermii. [Effect of Cholinomimetics on the Rate of Hypothermia Development.]** *Farmakologiya i Toksikologiya* 44(1): 40-43, 1981, Russian.

Development rate of cholinergic hypothermia at 33, 26, and 19°C rectal temperatures was determined in 120 male rats cooled in water at 7°C after intraperitoneal injection of various cholinomimetics, including nicotine at the dose of 4 mg/kg. The underlying hypothesis was that artificial hypothermia affects the reaction of cholinergic receptors to cholinomimetics. In fact, at the stages of mild (at 33°C) and moderate (26°C) hypothermia, nicotine decreases the rate of hypothermia development by 13.5 and 18.3 percent, respectively, compared with the controls. These statistically significant decreases were more important than those induced by the other cholinomimetics. At the stage of a deep hypothermia (at 19°C), nicotine was without effect on the rate of development. It is concluded that nicotine stimulates both central and peripheral nicotine-cholinergic receptors. Calculations show that nicotine stimulated central N-cholinergic receptors alone are responsible for 8.1, 18.3, and 18.9 percent decreases in the rates of mild, moderate, and deep hypothermia, respectively. Thus, a generally positive reaction of organism to stimulation of cholinoreactive system is evidenced by a decrease in hypothermia development rate, mainly due to stimulation of central cholinergic receptors.

82–0494. Becker, C. G.; Van Hamont, N.; Wagner, M. **Tobacco, Cocoa, Coffee, and Ragweed: Cross-Reacting Allergens That Activate Factor-XII-Dependent Pathways.** *Blood* 58(5): 861-867, November 1981.

Antigens isolated from cocoa powder, ground coffee, and ragweed pollen were found to be immunologically cross-reactive with a glycoprotein antigen from cured tobacco leaves and from cigarette smoke condensate; they contained similar polyphenol haptens; and were capable of activating factor XII-dependent pathways in normal plasma. The antigens were compared by hemagglutination inhibition assay, passive cutaneous anaphylaxis technique, electrophoresis, and isoelectric focusing. The cross-reactivity of the tobacco, cocoa, coffee, and ragweed antigens was due in part to the presence of similar polyphenol haptens resembling quercetin or rutin. The factor XII-dependent pathways involved coagulation, fibrinolysis, and kinin generation in normal human plasma. Since other commonly ingested or inhaled substances show cross-reactivity with tobacco and can activate factor XII-dependent pathways, it is possible that they too are involved in the pathogenesis of cardiovascular disease. Cigarette smoking may be a more prominent cardiovascular risk factor because of the relatively easy absorption of even fairly large molecules after inhalation.

82–0495. Bergstrom, J. **Short-Term Investigation on the Influence of Cigarette Smoking Upon Plaque Accumulation.** *Scandinavian Journal of Dental Research* 89(3): 235-238, June 1981.

Cigarette smoking was found to have an antibacterial potency to prevent or restrict plaque formation, in this study of the influence of cigarette smoking on plaque accumulation in young adult males (24-37 years of age) who endeavored to stop smoking. Plaque accumulation was evaluated photogrammetrically during a 5-day smoking period and, after an interval of at least 30 days, during a nonsmoking period of the same length. It was found that the amount of plaque was comparatively larger and plaque accumulation rate consequently increased in smokers throughout the smoking period. The differences were small, however, and not statistically significant. The result substantiates the view that cigarette smoking has no antibacterial potency so as to reduce plaque formation on tooth surfaces. (Auth. Abs. Mod.)

82–0496. Biswas, N. M.; Patra, P. B. **Role of Testosterone Propionate on Spermatogenesis Following Chronic Nicotine Administration in Immature Rats.** *Indian Journal of Experimental Biology* 19: 604-606, July 1981.

Immature rats exposed to nicotine showed decreased body weight, reduced testicular and secondary sex organ weight, and arrested spermatogenesis; but testosterone treatment appeared to be useful therapy. Wistar strain rats were divided into four groups. The first group consisted of saline-treated controls. The second group was given nicotine for 32 days. The third group was given nicotine for 32 days with subsequent testosterone treatment on days 26 to 32. The fourth group comprised nicotine-treated animals who received testosterone at a higher dose on days 26 to 32. Testosterone therapy at high and low doses prevented testicular degeneration, increasing the testicular and secondary sex organ weight. Body weight also significantly increased after testosterone injections. It was suggested that nicotine may induce testicular degeneration by inhibiting pituitary gonadotropin release.

82–0497. Clarke, N. G.; Shephard, B. C.; Hirsch, R. S. **The Effects of Intra-Arterial Epinephrine and Nicotine on Gingival Circulation.** *Oral Surgery, Oral Medicine, Oral Pathology* 52(6): 577-582, December 1981.

Epinephrine and nicotine were found to influence gingival blood flow in rabbits, supporting the hypothesis that acute necrotizing ulcerative gingivitis (ANUG) could be the result of ischemia promoted by predisposing factors such as smoking. Vascular stasis, stress, and smoking were said to combine and induce severe vascular constriction at the end arterial locations of the gingivae, resulting in ischemic necrosis. Clinical doses of epinephrine and nicotine were infused intra-arterially and the gingival circulation was monitored by the heat-diffusion principle. It was found that the chemicals caused a severe reduction in flow rates in spite of greatly increased pressure within the system. The finding supported the hypothesis that ischemia could play an important role in the initiation of ANUG. (Auth. Abs. Mod.)

82–0498. Dai, W. S.; Kuller, L. H.; LaPorte, R. E.; Gutai, J. P.; Falvo-Gerard, L.; Caggiula, A. **The Epidemiology of Plasma Testosterone Levels in Middle-Aged Men.** *American Journal of Epidemiology* 114(6): 804-816, December 1981.

A Multiple Risk Factor Intervention Trial of 243 men, that was investigating the effects of smoking and other influences on plasma testosterone level, found that smoking showed an indirect positive association. The subjects were all considered to be free of heart disease and were between ages 35 and 37 at entry to the trial. Several diseases such as coronary heart disease and cancer have been associated with sex hormones, making the study of the epidemiology of the hormones important. Having shown that a single morning blood draw was sufficient for characterization of plasma testerone levels, a specimen was taken from each subject. The variables examined included smoking, age, alcohol consumption, obesity, exercise, medication, and psychosocial factors. Free and total testosterone levels decreased with age and obesity. Plasma testosterone levels were similar among those taking and not taking medications. There was no significant relationship of testosterone level with history of alcohol intake, exercise, or with psychosocial factors such as type A behavior, speed impatience, job involvement, and hard driving, except for a borderline significant positive relationship between free testosterone and impatience. Smoking was positively correlated with testosterone level, but regression analysis showed this to be an indirect association, through a relationship with age. Smoking was negatively related to age and so appeared to be positively related to testosterone.

82-0499. Ducot, B.; Mayaux, M.-J.; Spira, A. **Testicular Varicoceles and Tobacco Consumption.** *Fertility and Sterility* 36(5): 686-687, November 1981.

No significant difference related to cigarette smoking was found among subjects with and without testicular varicocele, in a population of infertile and fertile men. In a study of 576 men consulting for infertility, the proportion of smokers among those with and without varicocele was 59 percent and 57 percent, a difference that was not significant. The distribution of daily cigarette consumption among the smokers was similar for those with and without varicocele. Smokers with varicocele smoked 16.9 ± 9.6 (mean ± standard deviation) cigarettes a day. Of this group, 76 percent inhaled. Smokers without varicocele smoked 17.4 ± 8.9 cigarettes a day, and 80 percent of them inhaled. The differences were not considered significant. A study of 168 prevasectomy, fertile men found that 35 percent of those with varicocele were smokers, compared to 43 percent of those without varicocele. (The difference was not significant.) The distributions of consumption (14.9 ± 11.0 vs. 15.1 ± 8.6) and of inhalation (89 percent vs. 71 percent) were similar. It is suggested that any suspected relationship between cigarette smoking and increased testicular varicocele incidence should be examined using larger samples before any interpretation is attempted.

82-0500. Koenig, C.; Classen, H. G. **Der Einfluss von Zigarettenrauchen und Kaltereizen auf die Hauttemperatur von Rauchern und Nichtrauchern. [Effects of Cigarette Smoking and Low-Temperature Stimulus on Skin Temperature of Smokers and Nonsmokers.]** *Die Medizinische Welt* 32(34): 42-44, August 21, 1981, German.

Skin temperature was measured on toe and finger tips and in the forehead under standardized conditions before and during smoking of one filter cigarette containing 0.8 mg of nicotine. A total of 39 volunteers of both sexes, including 18 smokers and 21 nonsmokers, participated in the experiment. The smokers abstained from smoking for at least 12 hours before the experiment. In an additional experiment, skin temperature was measured after the contralateral arm was immersed in water at 13°C for 2 minutes. The blood vessel reaction to this low-temperature stimulus was identical in both smokers and nonsmokers; thus, sensitivity of the vessel muscles to this stimulus was not measurably different in smokers and nonsmokers. In contrast, temperature of the finger and toe tips after smoking decreased significantly more in smokers than in nonsmokers and the minimum temperature was recorded later in the smokers. This result is explained by the difference in smoking habits, primarily smoke inhalation that clearly increases nicotine resorption through the mucous membrane.

82-0501. Lindemeyer, R. G.; Baum, R. H.; Hsu, S. C.; Going, R. E. **In Vitro Effect of Tobacco on the Growth of Oral Cariogenic Streptococci.** *Journal of the American Dental Association* 103(5): 719-722, November 1981.

Most pouch (loose-leaf) and plug (pressed) chewing tobacco with high sugar contents are able to support the growth of the oral cariogenic microorganisms *Streptococcus mutans* and *S. sanguis* in vitro. Snuff and unprocessed tobacco, although not able to stimulate growth of these organisms, do not inhibit growth. Using tobacco products which were representative of the popular brands of smokeless tobacco, a preliminary study employing the disk diffusion technique was conducted to provide a simple indication of growth stimulation or inhibition. Because results of the preliminary study suggested that growth was enhanced rather than inhibited, a turbidometro method was used to quantitate growth. Results of the turbidometric assay correlated well with the preliminary disk diffusion assays.

82-0502. Rona, R. J.; Florey, C. du V.; Clarke, G. C.; Chinn, S. **Parental Smoking at Home and Height of Children.** *British Medical Journal* 283(6303): 1363, November 21, 1981.

Family studies in England and Scotland appeared to show that passive smoking at home can adversely affect the growth of children. Information was collected on the height of children in 28 study areas as well as the number of people smoking at least five cigarettes a day at home and the child's respiratory symptoms. The children were divided into three groups, based on whether there were no smokers, one smoker, or two or more smokers at home. A strong inverse association was found between height and the number of smokers at home. Children with one smoker at home had an intermediate mean height compared with the other two groups. After adjustment for the child's birth weight to allow for maternal smoking, the trend was still significant in England but borderline in Scotland. When additional adjustments were made for the father's social class, the mother's and father's heights, and the number of siblings, the trend was still significant for the English sample but not for the Scottish one. A similar analysis by sex indicated a significant trend for boys in England after all other independent variables had been allowed for. The trend was also observed in girls in Scotland. The number of respiratory symptoms in the previous 12 months did not explain the association between height and smokers at home. Passive smoking at home appeared to affect the growth of children.

82-0503. Rycroft, R.J.G.; Smith, N. P.; Stok, E. T.; Middleton, K. **Investigation of Suspected Contact Sensitivity to Tobacco in Cigarette and Cigar Factory Employees.** *Contact Dermatitis* 7(1): 32-38, January 1981.

Erythema, urticaria, and mild eczema are not uncommon among cigarette and cigar factory workers. Two independent cases of suspected occupational allergic contact dermatitis from tobacco are reported to illustrate the difficulties inherent in the accurate diagnosis of this condition. In the first case a diagnosis of contact sensitization to tobacco was made whereas in the second it was rejected. The reason for the different conclusions in the two patients is explained in detail and an investigatory procedure is outlined whereby the comparatively rare case of genuine sensitivity to tobacco can be distinguished from other more common dermatoses. (Auth. Abs. Mod.)

82-0504. Sonnenberg, A.; Mueller-Lissner, S. A.; Vogel, E.; Schmid, P.; Gonvers, J. J.; Peter, P.; Strohmeyer, G.; Blum, A. L. **Predictors of Duodenal Ulcer Healing and Relapse.** *Gastroenterology* 81(6): 1061-1067, December 1981.

Smoking adversely affected the healing of duodenal ulcers in a population known to have a high healing incidence. Two double-blind studies were performed with 134 patients over 4 weeks and with 66 patients over 1 year. The short-term treatment involved treating with cimetidine at 1 g/day, pirenzepine at 75 mg/day, or placebo. Treatment during the long-term study involved either cimetidine at 400 mg at bedtime, pirenzepine at 30 mg at bedtime, or placebo. In the short-term study, abstinence from smoking was one of the factors shown to increase healing incidence. Other factors increasing healing incidence were female sex, moderate alcohol consumption, young age, and cimetidine treatment. In the long-term study, cimetidine prevented ulcer relapse. Smoking had no statistically significant effect on the outcome among the cimetidine and pirenzepine groups, but did favor duodenal ulcer relapse in the placebo group.

82-0505. Vaddadi, K. S. **Niacin Flushing and Schizophrenia.** *Medical Hypotheses* 7(5): 599-600, May 1981.

Nicotine may be antagonistic toward nicotinic acid (or niacin) in the peripheral vasculature. Niacin and nicotine are chemically similar. Nicotine can cause vasoconstriction when given intravenously in large doses, which may antagonize possible vasodilatation effects of niacin. Schizophrenics may smoke heavily and have high circulating blood nicotine levels, possibly blocking niacin receptors. The absence of niacin flushing in the majority of schizophrenics even after administering large doses of niacin may not be due to absent or reduced formation of prostaglandins, as some suggest. Instead, the lack of flushing may be due to niacin receptor blockage at the periphery by circulating nicotine, mutually antagonistic actions between nicotine and niacin, or an endogenous agent blocking the niacin/nicotine receptors. Normal smokers and normal nonsmokers, as well as schizophrenic nonsmokers and heavy smokers, should be studied for any differences in niacin-induced flushing.

82-0506. Zetterstrom, O.; Osterman, K.; Machado, L.; Johansson, S.G.O. **Another Smoking Hazard: Raised Serum IgE Concentration and Increased Risk of Occupational Allergy.** *British Medical Journal* 283(6301): 1215-1217, November 7, 1981.

Geometric mean immunoglobulin E (IgE) serum concentrations were found to be higher in smokers than in nonsmokers among a general population sample and among workers exposed to occupational allergens. One worker group was composed of pharmaceutical factory employees exposed to ispaghula powder (used in bulk laxatives), and the other consisted of workers at a coffee roastery who were tested with green coffee bean and castor bean extracts. Radioallergosorbent and skin-prick allergen tests were performed, and the evidence for sensitization against occupational allergens appeared to be more common in workers who smoked. Smoking workers showed a higher prevalence of apparent atopic symptoms such as sensitization and higher IgE levels than did nonsmoking workers. It was suggested that smoking's influence on IgE antibody production might be caused by damage to the airway mucosa and by affecting the handling of inhaled antigens, promoting a shift in antibody production from immunoglobulin G to IgE.

See also, **82-0441, 82-0521**

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

82-0507. Aaro, L. E.; Hauknes, A.; Berglund, E.-L. **Smoking Among Norwegian Schoolchildren 1975-1980. I. Extent of Smoking in the Age Group 12-15 Years, 1975.** *Scandinavian Journal of Psychology* 22(3): 161-169, 1981.

Changes in smoking patterns among Norwegian schoolchildren and recommendations for further antismoking efforts are discussed in this 1975 survey of 213,600 pupils from grades 6 to 9 (age 12-15). The headmaster of each of the 2,824 schools participating in the study filled in reports giving the local frequencies of daily smokers, occasional smokers, and nonsmokers for each grade and for boys and girls separately. The school reports also contained questions about demographic properties of the school. A sample of 6,968 questionnaires from the pupils was also submitted for statistical analysis. Compared with previous studies, the percentage of smokers among girls proved to have increased considerably. The tendency found in many western industrialized countries, that smoking is increasing among girls more than among boys, or that boys are reducing smoking to a greater extent than are girls, was confirmed also for Norway. In all 19 countries there was a higher percentage of daily smokers among girls than among boys. Higher percentages of occasional smokers or pupils who had tried smoking were found in small schools and scattered settlements. Norway may be divided into geographical areas according to the differences in the smoking habits of girls and boys. In the counties in the south and to the west, girls have not surpassed boys to the same extent as in the rest of the country. From previous political-cultural research it is known that the south and west are characterized by temperance and prohibition movements, higher frequencies of religious attendance, as well as a distinctiveness in voting behavior. (Auth. Abs. Mod.)

82-0508. Baettig, K. **Smoking and the Behavioral Effects of Nicotine.** *Trends in Pharmacological Sciences* 2(6): 145-147, June 1981.

Studies of animals and humans suggest that nicotine has a major role in the smoking habit. In the inhaling smoker, nicotine is absorbed chiefly by the pulmonary membranes and can show up in the blood plasma within seconds after inhalation. The nicotine absorbed can produce measurable pharmacological and psychopharmacological effects. In animal studies, the behavioral effects of nicotine can show a biphasic curve. Below a critical point, nicotine can have a predominantly stimulatory effect, and above it a predominantly depressant effect. Among humans, smokers may show a decrease of performance for smoking deprivation. Smoking gives relief from anxiety and stress among smokers. Psychotic and neurotic traits appear more often in smokers than nonsmokers. Smoking can increase the heart rate and lead to peripheral vasoconstriction. Such effects may be due at least partly to increased release of norepinephrine from endings of the peripheral sympathetic system and release of catecholamine from the adrenal medulla, accompanied by increased plasma levels of cortisone and growth hormone. The smoke exposure of a smoker seems due more to the style or pattern of smoking than to the yield of the cigarette.

82-0509. Benfari, R. C.; Eaker, E.; Stoll, J. G. **Behavioral Interventions and Compliance to Treatment Regimes.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 2. Palo Alto, California, Annual Reviews Inc., 1981, pp. 431-471.

The status of health behavioral interventions is reviewed, concentrating on adherence to prescribed treatment (secondary prevention) and alteration of risk factors or unhealthy behaviors such as smoking (primary prevention). Smoking behavior has been associated with heart diseases, malignant neoplasms, cerebrovascular diseases, influenza and pneumonia, arteriosclerosis, bronchitis, emphysema, and asthma. Numerous variables that can influence treatment compliance and risk factor change are delineated. A literature review is also presented; emphasis is on recent selected studies using behavior intervention in risk factor change and in prescribed treatment regimes. Behavior strategies for clinical application are listed and described, including reinforcement procedures, shaping techniques, systematic desensitization, relaxation techniques, hypnotherapy, behavior contracts, self-management, modeling, behavior rehearsal, cognitive restructuring, thought-stopping techniques, cognitive dissonance, creative problemsolving, decisionmaking, imagery training, and assertiveness training. The therapeutic relationship as well as the behavioral assessment and therapy process are discussed.

82-0510. Berggreen, S. **Parental Smoking at Home and Height of Children.** *British Medical Journal* 283(6306): 1612, December 12, 1981.

Other explanations are offered concerning the observation that the more people smoke at home the smaller the children (*British Medical Journal* 283(6303): 1363, November 21, 1981). Smoking is an expensive habit, and in a family with heavy tobacco consumption there is less money for other things beneficial to health. Because smoking reduces the appetite, meals prepared by the smoker may not be attractive or constitute a well-balanced diet for the children. Other psychological, social, and economic factors can affect the health of family members directly or indirectly. See also 82-0502.

82-0511. Bergler, R. **Advertising and Cigarette Smoking. A Psychological Study.** Bern, Switzerland, Hans Huber Publishers, 1981, 188 pp.

The present state of knowledge related to cigarette smoking and advertising is reviewed under three headings: (1) the motivation behind cigarette smoking, (2) the effects of mass communications, and (3) advertising and cigarette smoking. Relative to the motivation behind cigarette smoking, the influences of both demographic and psychological variables are discussed. Concepts and variables of the effectiveness of mass communication are investigated. Available literature on advertising and cigarette smoking is analyzed. It is concluded that future research into the origins and causes of smoking behavior will have to look at the problem much more from the point of view of social psychology and the psychology of personality, and that advertising can no longer be regarded as the major dependent variable governing this behavior.

82-0512. Bernard, P.-M.; Tremblay, J.; Rousseau, N.; Begin, C. **Prevalence de l'Usage de la Cigarette en Milieu Scolaire: Quebec, 1978. [Cigarette Smoking Prevalence Among School Children: Quebec, 1978.]** *L'Union Medicale du Canada* 109(12): 1724-1733, December 1980, French.

A questionnaire about smoking habits was administered to a sample of 6,211 French students (ages 7 to 12) in primary and secondary classes in metropolitan Quebec. Analysis of the

6,116 answers shows that about 32 percent of the boys and 24 percent of the girls had tried cigarette smoking before the age of 9, that is, in the primary classes. Girls start smoking 5 to 9 months later than boys, but by age 12 more girls than boys have tried cigarette smoking. The percentage of occasional and regular smokers was stable in the primary classes (7 to 9 years old), but it more than doubled for boys and nearly quintupled for girls during the transition from the primary to secondary classes. Thus, the last primary class before transition to the first secondary class is the target group for optimum preventive intervention with health education programs aimed at preventing smoking by schoolchildren.

82-0513. Biener, K. **Exraucherinnen. [Women Ex-Smokers.]** *Munchener Medizinische Wochenschrift* 123(25): 1035-1038, June 19, 1981, German.

Previous smoking habits, motivation for smoking cessation, smoking cessation methods, and factors contributing to and problems associated with smoking cessation were determined by interviewing 100 female ex-smokers and comparing them with 174 male ex-smokers. The interviewed women comprised barely 10 percent of 1,036 female employees randomly selected from 44 factories in German-speaking Switzerland. The earlier surveyed male ex-smokers were a 14 percent fraction of the similarly selected 1,260 male employees. Only the ex-smokers who had previously smoked more than nine cigarettes/day were included in the study. Fear of health damage or first symptoms of illness motivated 39 percent of the women to stop smoking; pregnancy was the cause of smoking cessation in 9 percent. A slow reduction of smoking was found to be the most successful tactic by 23 percent of the women compared to only 4 percent of the men. In their decision to stop smoking, women were much less influenced by a physician than men and were more often influenced by the spouse, other relatives, coworkers, or friends and also by the mass media. Sports had a direct effect on 21 percent of women vs. 28 percent of men ex-smokers. The men and women ex-smokers experienced equally the problem of compensation, while half the women and three-fifths of the men gained weight. Equal percentages of the women and men agreed that willpower was necessary to stop smoking.

82-0514. Bone, R. C.; Phillips, J. R.; Chowdhury, P. **The Smoking Habit: Physical Dependence on Nicotine.** *Journal of Respiratory Diseases* 2(5): 10-16, May 1981.

Cigarette smoking leads to a physical dependence on nicotine. As in other dependence disorders, cessation causes not only subjective symptoms but also objective physical withdrawal effects. Social factors important in acquiring the smoking habit have their greatest impact before age 20; stop-smoking efforts should focus on adolescents and children and should emphasize the dependence aspects. Questionnaire responses illustrate the complexity of the smoking habit; 50 percent of those who stopped smoking did so abruptly, while 50 percent reduced smoking gradually. The same technique does not work for everyone, and attempts to help patients stop smoking will be more successful with better understanding of the specific factors involved in acquisition, maintenance, and cessation of cigarette smoking. (Auth. Abs. Mod.)

82-0515. Chassin, L.; Presson, C. C.; Bensenberg, M.; Corty, E.; Olshavsky, R. W.; Sherman, S. J. **Predicting Adolescents' Intentions To Smoke Cigarettes.** *Journal of Health and Social Behavior* 22(4): 445-455, December 1981.

Adolescents' intentions to smoke cigarettes were predicted by Ajzen and Fishbein's (1970) model and by the distal variables derived from Jessor and Jessor's (1977) problem-behavior theory, as well as by several other variables relevant to cigarette smoking. The Ajzen and Fishbein model uses proximal variables. It suggests that adolescent intentions to smoke can be predicted from attitudes toward smoking and from adolescents' normative beliefs about whether significant people in their social world feel they should smoke. The distal predictor variables of personality and perceived environment were chosen from two of Jessor and Jessor's multivariate systems. The Ajzen and Fishbein model was found to have significant predictive ability for all smoking statuses in both middle-school and high-school subjects. The Jessor and Jessor distal variables also had significant predictive ability for all smoking statuses in the two groups, but accounted for less of the variance in behavioral intentions. Additionally, except for subjects who smoked, the distal variables were found to add only a small amount of explained variance in the behavioral intentions to the Ajzen and Fishbein model when the two sets of variables were combined in hierarchical regressions. Subjects' estimates of smoking prevalence, health beliefs, and number of friends who smoke were also found to bear some relation to smoking intentions. The study's results may be useful in helping identify those adolescents most at risk of beginning smoking, and in selecting target audiences for prevention programs. The prevention programs could perhaps be tailored to the characteristics of the adolescents at risk. (Auth. Abs. Mod.)

82-0516. Cummins, R. O.; Shaper, A. G.; Walker, M.; Wale, C. J. **Smoking and Drinking by Middle-Aged British Men: Effects of Social Class and Town of Residence.** *British Medical Journal* 283(6305): 1497-1502, December 5, 1981.

In 7,735 men (aged 40-59), selected at random from general practices in 24 towns throughout Britain, pronounced differences were noted in the prevalences of smoking and drinking between the social classes. Social class differences also existed for frequency and quantity of drinking, type of beverage, and several aspects of smoking behavior. Increasing amounts of smoking were associated with higher prevalences of moderate to heavy drinking, particularly in daily rather than weekend drinkers. Between drinking groups, however, the relation with smoking was more U-shaped, with light and heavy drinkers smoking more than moderate drinkers. The lowest rates of moderate to heavy smoking were observed in frequent light drinkers, particularly in the nonmanual workers. The proportion of moderate to heavy drinkers was no higher among ex-cigarette smokers than among current smokers. When the data were examined by town of residence, social class differences persisted. Controlling for social class still showed pronounced differences between towns in both smoking and drinking behavior. These data confirm that town of residence and social class have independent effects on smoking and drinking. The established regional and social class differences in cardiovascular disease may be due in part to the independent influences of town and social class on smoking and drinking behavior. (Auth. Abs.)

82–0517. Dawley, H. H., Jr.; Carrol, S. F.; Morrison, J. E. **The Discouragement of Smoking in a Hospital Setting: The Importance of Modeled Behavior.** *International Journal of the Addictions* 16(5): 905-910, 1981.

Results of a hospital study indicate that modeled smoking behavior among hospital employees tends to promote smoking behavior among patients and visitors. A high status model meant to resemble a medical doctor, and a "peer" model meant to resemble a patient, smoked in the presence of patients and visitors waiting in a no-smoking area. The results clearly showed an increased smoking rate among subjects when models smoked, with women slightly more influenced to smoke than men. No significant difference was found between models. The findings tend to support the importance of health personnel, particularly doctors, refraining from smoking in the presence of patients. (Auth. Abs. Mod.)

82–0518. De Haas, J. H.; De Haas-Posthuma, J. H. **Niet-rokers in Nederland. [Non-smokers in the Netherlands.]** *Hart Bulletin* 12: 51-55, April 1981, Dutch.

In a Dutch survey of the smoking habits of adults over 15 years of age, it was found that nonsmokers (never- and ex-smokers) now outnumber smokers. The development of the smoking pattern of this century has determined the present day differences in sex, age, and social class of never- and ex-smokers: 80 percent of elderly women compared to only 30 percent of elderly men; but 67 percent of young men who have never smoked. In men, the percentage of never-smokers drops with increasing age and with lower social class; in women, it drops only to age 35, and then it starts to rise. In men, the prevalence of ex-smokers rises with age. The lowest percentages of ex-smokers were found in men and women of the lower social classes. In the seventies the increase in the percentage of ex-smokers in both sexes was small, while the increase of nonsmokers in men can be attributed to boys who did not start the smoking habit. If the increase of nonsmokers continues, the prevalence of never- and ex-smokers will increase considerably, a change for the better for the prevention of coronary heart disease and other smoking-related diseases. It is concluded that antismoking campaigns should concentrate on teenagers and on men and women of the lower social classes. (Auth. Abs. Mod.)

82–0519. Dobbs, S. D.; Strickler, D. P.; Maxwell, W. A. **The Effects of Stress and Relaxation in the Presence of Stress on Urinary pH and Smoking Behaviors.** *Addictive Behaviors* 6(4): 345-353, 1981.

Smokers subjected to stress in a behavior study were found to smoke more than those in a more relaxed environment. Male heavy smokers (32) who had indicated a fear of public speaking were randomly assigned to one of three stress groups or a control group. The three stress groups were informed that the last phase of the experiment involved giving a speech to a group of graduate students. They then listened to either a stress enhancing tape, taped relaxation instructions, or a neutral tape. Stress instructions were omitted for the control subjects, and they listened to the neutral tape. Objective (skin conductance response) and subjective (state anxiety) measures of arousal indicated that the treatments had their intended effects. Following treatment, subjects were left alone for a 35-minute period during which their smoking behaviors were monitored. The relaxation group smoked significantly fewer centimeters of cigarettes ($p<0.01$) and at a significantly slower rate ($p<0.01$) than the subjects in the two other stress groups. The relaxation group also showed a lower puff rate and a less acidic urinary pH at posttreatment than did the two other stress groups. Increased smoking in the stress groups may have been to replenish the nicotine lost by the more rapid rate of urinary excretion, or the lowering of urinary pH levels, causing physiological cravings. Relaxation may help smokers cope with stress that would otherwise lead to an increase in smoking. (Auth. Abs. Mod.)

82–0520. Dull, R. T.; Williams, F. P., III. **Marihuana, Alcohol and Tobacco: Reassessment of a Presumed Relationship.** *Journal of Drug Education* 11(2): 129-139, 1981.

Current marihuana literature posits a strong relationship between alcohol, tobacco, and marihuana. The evidence for this relationship, however, is drawn largely from studies which have used youthful samples. Using a random survey of the general Texas population (age 17 and over), the present study found little relationship between the three substances. Multiple regression analysis supported this finding by indicating that less than 8 percent of explained variation in marihuana use was due to both alcohol and tobacco. In a further effort to explain this contradictory finding, an age variable was introduced. Results indicated that youthful samples tend to overestimate the relationships between the three substances and cannot be generalized to other populations. A likely explanation of association found in youthful samples would focus on simultaneous experimentation rather than causal interrelationships among the substances themselves. (Auth. Abs.)

82–0521. Dwyer, T.; Calvert, G. D.; Baghurst, K. I.; Leitch, D. R. **Diet, Other Lifestyle Factors and HDL Cholesterol in a Population of Australian Male Service Recruits.** *American Journal of Epidemiology* 114(5): 683-696, November 1981.

While major emphasis is on dietary factors, particularly fat intake, smoking is identified as one factor associated with plasma high density lipoprotein (HDL) cholesterol concentrations in this study of 530 Australian male service recruits. Tobacco consumption was measured according to cigarettes per day. Other factors examined included nutrient intake, percent energy intake, age, physical fitness, weight, height, and alcohol consumption. Tables listing associations between HDL cholesterol and the various study factors are included.

82–0522. Fagerstrom, K.-O.; Bates, S. **Compensation and Effective Smoking by Different Nicotine Dependent Smokers.** *Addictive Behaviors* 6(4): 331-336, 1981.

Smokers who are more highly dependent on nicotine appear to be more skillful at compensating for alterations in nicotine level than less dependent smokers. In this study of smoking behavior, a total of 14 smokers were measured for their dependence on nicotine by a Tolerance Questionnaire. Each individual smoked four cigarettes, of which two had high-nicotine levels (1.5 mg) and two had low-nicotine levels (0.5 mg). Smoking behavior was measured by a Smoking Score, a composite measure based on number of puffs, puff duration, and interpuff intervals. Data analysis indicated that the effect of the type of cigarette smoked was significant at the 0.1 percent level while the effect of nicotine dependence on smoking was significant at a 5 percent level. Low-nicotine cigarettes were smoked much more effectively than high-nicotine cigarettes. Subjects who were more dependent smoked more effectively than those who were less dependent, regardless of cigarette nicotine content, indicating that the nicotine dependence of the smokers influenced the way in which they smoked.

82–0523. Frank, R.; Lebowitz, M. D. **The Risk of Staying In.** *American Review of Respiratory Disease* 124(5): 521-522, November 1981.

The problem of indoor air pollution, which is greatly aggravated by smoking, will require better design, construction, and furnishing of homes, and commercial and public buildings, with greater public education and increased medical and technical information before it is solved. Toxic elements in tobacco smoke (carbon monoxide, nitrogen oxides, polycyclic organic matter, pharmacologically active agents, alpha-emitting radionuclides, formaldehyde) are a threat to nonsmokers (passive smoking), as well as smokers, although specific effects are still largely undetermined. Although one study (*American Review of Respiratory Disease* 124(2): 143-148, August 1981) found no conclusive evidence of respiratory effects from passive smoking, another (*Science* 208: 464-472, May 2, 1980) did show that the mass of particulates generated by smoking can overwhelm most residential air ventilation or exchange systems. A related problem arises with the growing need to conserve energy; well-insulated homes with reduced ventilation will have a reduced air exchange rate, raising the level of indoor air pollution. Further study of this problem and the specific effects of indoor air contaminants is recommended. See also 81-1809 and 80-1378.

82–0524. Gallup Report. **Gallup Smoking Audit: Percentage of Americans Who Smoke Lowest in 37 Years. Perceptions of Health Hazards May Explain Decline.** *Gallup Report* (Report No. 190): 2-17, July 1981.

The percentage of smokers in the United States today is the lowest recorded in 37 years of regular Gallup Poll audits: 35 percent reported smoking within the past week, compared with 43 percent in 1972. Since 1972, the decline in percentage of smokers has been most pronounced among younger adults (18 to 29 years old). Current survey highlights include: (1) the proportion of heavy smokers has declined over the last 4 years; (2) 66 percent say they would like to quit, the same proportion as recorded in the 1978 survey; (3) one-third of the 65 percent of American adults who do not presently smoke did so at one point in their lives; (4) 8 of 10 current smokers have tried to quit but only one-fourth managed to last 6 months or more without smoking; (5) smoking has shown a pronounced downward trend over the last decade among persons with a college background; (6) the decline has been somewhat greater among men than among women; and (7) in general, consumption levels among all smokers are down. It was also found that 22 percent of teenagers smoke, the same ratio found in surveys conducted in 1977 and 1979, with slightly more girls than boys smoking. In addition, of those surveyed, the proportion who believe smoking to be a cause of heart disease has increased from 60 percent in 1969 to 74 percent in 1981, while the number who perceive smoking as a cause of lung cancer has grown from 71 to 83 percent since 1971. There has also been an increase in the percentage who believe that smoking is a cause of birth defects, as well as in the proportion who say that smoking is a cause of throat cancer. A growing number also favor a harder line on smoking: 46 percent think Federal and State taxes on cigarettes should be increased, compared to 38 percent in 1977, while 43 percent think there should be a complete ban on cigarette advertising, compared with 36 percent in 1977; however, similar majorities in both surveys oppose a complete ban on the sale of cigarettes. According to the Toronto-based Gallup poll, 45 percent of Canadians are classified as smokers, 54 percent of Canadians think all advertising for cigarettes should be banned, and 59 percent favor separate areas in restaurants for those who smoke.

82–0525. Greden, J. F.; Procter, A.; Victor, B. **Caffeinism Associated With Greater Use of Other Psychotropic Agents.** *Comprehensive Psychiatry* 22(6): 565-571, November-December 1981.

A study of hospitalized patients appeared to indicate a significantly greater use of selected psychoactive drugs such as tobacco among high caffeine consumers, whether the individuals were psychiatric or nonpsychiatric patients. The study involved 81 psychiatry patients and 124 nonpsychiatry patients who were divided in three groups according to caffeine intake:

(1) low consumers (0-249 mg per day), (2) moderate consumers (250-749 mg per day), and (3) high consumers (750 mg or more per day). The psychiatric and nonpsychiatric patients did not differ significantly in their caffeine use. In the total study sample, 69 percent of high caffeine consumers smoked tobacco during monthly use, compared with 31 percent of low caffeine users. Cigarette use was larger in all caffeine-using subgroups among psychiatric patients when compared with nonpsychiatric patients. Smoking was reported among 82 percent of psychiatric patients who ingested more than 750 mg caffeine per day. Compared with nonpsychiatric patients, more high caffeine-consuming psychiatric patients reported occasional (defined as once a week or less) and regular (twice a week or more) use of cigarettes.

82–0526. Greenberg, B. S. **Smoking, Drugging and Drinking in Top Rated TV Series.** *Journal of Drug Education* 11(3): 227-234, 1981.

An analysis of the top 10 prime time fictional series from the 1979-80 television season indicates a near complete absence of smoking and illicit drug incidents. By contrast, the offering and consumption of alcoholic beverages is markedly larger than found in prior studies which did not separate out top-rated programs from lesser viewed shows. The incidence of alcohol consumption for the top 10 shows at the end of the season ranged from 3 to 16.5 incidents per program hour. A parallel analysis of the two top-rated soap operas for the season yielded a lower drinking rate and a similar absence of smoking and drug incidents. (Auth. Abs.)

82–0527. Gritz, E. R. **Problems Related to the Use of Tobacco by Women.** In: Kalant, O. J. (Editor). *Alcohol and Drug Problems in Women.* Research Advances in Alcohol and Drug Problems, Volume 5, New York, Plenum Press, 1980, pp. 487-543.

An analysis of tobacco use among women, with comparative reference to men, is provided, including discussions on anthropological and historical trends, epidemiological surveys, psychological and physiological factors, health risks, pregnancy, and smoking cessation. Historically, women in the United States began to take up cigarette smoking in significant numbers after 1930. Epidemiological data suggest that the smoking habits of men and women are becoming progressively similar with increased smoking in the female population resulting from earlier recruitment and heavier smoking practices. Speculations are that smoking serves as a pharmacological prompting mechanism to further mobilize stress reactions for women and that it serves different psychological purposes for men and for women, primarily in the area of negative-effect reduction. In addition to the risk of cancer, cardiovascular disease, chronic obstructive pulmonary disease, and peptic ulcer disease which have been linked to smoking, women must be concerned with the interactive risk between the use of oral contraceptives and smoking, the relationship between smoking and age of natural menopause, and the risks associated with pregnancy and smoking. Although it is suggested that men have greater success than women in giving up smoking, few studies report success rates by gender and such an assertion awaits further confirmation.

82–0528. Hakulinen, T.; Pukkala, E. **Future Incidence of Lung Cancer: Forecasts Based on Hypothetical Changes in the Smoking Habits of Males.** *International Journal of Epidemiology* 10(3): 233-240, September 1981.

Estimates of possible future lung cancer rates in Finland were found to be influenced by hypothetical changes in the proportion of smokers among the population. A simulation model was constructed on the basis of which effects of different changes in smoking habits on lung cancer incidence in the future could be quantified directly. A fictitious population, comparable to the entire male population of Finland with respect to smoking habits, was simulated by computer to experience selective general mortality and different lung cancer risks due to different smoking habits and changes in them. The distribution of smoking habits in the population was estimated on the basis of previous country-wide surveys and on published tobacco consumption statistics. The effects of different smoking habits on general mortality and lung cancer incidence were determined on the basis of previous studies. If the proportion of persons starting smoking is considered to be constant (30 percent of nonsmokers aged 10-14, 15 percent of those aged 15-19, and 5 percent of those aged 20-24 years) in each consecutive 5-year period after 1975, and the proportion of smokers stopping in each category of smokers is 20 percent in the same periods, the age-adjusted incidence rate in the year 2000 would be $46/10^5$ person-years. In 1975, the corresponding figure was $71/10^5$. If the proportion stopping is only 10 percent, the rate in 2000 would be $64/10^5$. If nobody stopped smoking, the figure would be $88/10^5$. Introduction of different alternatives for starting of smoking does not have a material effect on the forecasts up to the year 2000. After that year, however, different alternatives for the proportion of new smokers, the numbers of cigarettes smoked, and the age at starting have a considerable effect upon the forecasts. Based on the assumptions made, a postponement of 10 years in starting age has the same effect on lung cancer incidence as cutting the proportions of those starting by one half. Postponement of the starting age by 20 years would eliminate most of the lung cancer cases caused by smoking. To prevent a new rise in lung cancer incidence in the early 2000s in Finland, special efforts should be concentrated on persuading youths who started smoking in the 1970s to stop. (Auth. Abs. Mod.)

82–0529. Halama, P. **Ist der Soldat, der Gewohnheitsraucher ist, bei Vermindertem oder Fehlendem Nikotinkonsum in Stress-Situationen Psychisch voll Belastbar? [From a Psychiatric Point of View, Is a Soldier Who Is a Habitual Smoker Able To Serve in a Full Military Capacity During Stress Situations When He Smokes Less or Abstains?]** *Wehrmedizinische Monatsschrift* 24(11): 344-350, 1980, German.

Nicotine is a drug which is able to produce a physical and psychical dependence. During the phase of nicotine abstinence there are symptoms of deprivation such as disorders in somatic central nervous and psychic systems. Symptoms of psychical nicotine abstinence are, e.g., latent aggressivity, irritation, and disturbances in concentration. During the phase of nicotine abstinence the disposition in general is weak. From the military point of view the psychical exercise is endangered. (Auth. Abs.)

82-0530. Iverson, D. C. (Editor). **Promoting Health Through the Schools. A Challenge for the Eighties.** *Health Education Quarterly* 8(1): 1-117, Spring 1981.

As many as 40 percent of American children aged 11 to 14 have one or more of the risk factors for heart disease or cancer, including smoking. Promoting health means discouraging such detrimental behaviors as smoking. Cigarette use originates via peer pressure and social influences, and is pharmacologically reinforced into a stubborn habit. Both smoking and nonsmoking students are conformists, but belong to different reference groups. Smoking is more likely to be discouraged if such a habit is seen to be unsophisticated rather than a health risk. Classrooms must not be turned into mini-clinics for smoking cessation, alcohol abuse, etc., but should keep to the basic goal of nurturing growth, knowledge, and reason. Specific areas covered in this collection of conference reports include: (1) overview of the health problems of children and youth; (2) factors which influence child and adolescent health behavior; (3) the organization, implementation, and evaluation of school-based health promotion programs; (4) possible funding support for school health programs; and (5) a blueprint for school programs for the 1980s.

82-0531. Johnston, L. D.; Bachman, J. G.; O'Malley, P. M. **Highlights From Student Drug Use in America 1975—1980.** U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHHS No. (ADM) 81-1066, 1980, 119 pp.

Reported prevalence and trends in drug use among high school seniors show 71 percent had tried cigarettes, 31 percent had smoked in the past month, and 14 percent smoked a pack per day or more. Daily cigarette use was reported more often than any other drug, with slightly more females (14.7 percent) smoking than males (13.5 percent). The largest difference was related to college plans (8 percent college-bound vs. 21 percent noncollege-bound). No differences relating to urbanicity were found, but regional differences were apparent; the Northeast (17 percent) was highest, followed by the North Central and South, with the West being lowest (8 percent). Cigarette use showed a slight downward trend from 1979 (-4.0 percent), with use among males dropping more than females. Sixteen percent of the seniors reported initial use prior to 10th grade; 10 percent began smoking in grades 10-12. More students (64 percent) perceived smoking as harmful to health than in previous years, which correlates to downward trend in usage. The majority perceived their friends as disapproving of the smoking habit, a large upward trend, while 71 percent themselves disapproved of smoking, and 43 percent believe smoking should be prohibited by law. Smoking showed a negative relationship with grades, and a positive relationship with truancy and hours worked at a part-time job. Both cigarette and alcohol use predicted the use of marihuana and other drugs. Other categories of drug use reported are marihuana (including hashish), inhalants, hallucinogens, cocaine, heroin, other opiates, stimulants, sedatives, and tranquilizers.

82-0532. Kaprio, J.; Koskenvuo, M.; Sarna, S. **Cigarette Smoking, Use of Alcohol, and Leisure-Time Physical Activity Among Same-Sexed Adult Male Twins.** In: *Twin Research 3: Epidemiological and Clinical Studies.* New York, Alan R. Liss, Inc., 1981, pp. 37-46.

Studies conducted in Finland on cigarette smoking, use of alcohol, and physical activity among same-sexed adult male twins appeared to indicate that genetic factors play a relatively small role in the level of such habits or variables. Questionnaires were used to gather information about the variables from 1,537 monozygotic (MZ) and 3,507 dizygotic (DZ) male pairs aged 18 and over. Factor analyses were carried out on the variable groups to yield a condensed score for each variable group. There was a steady increase in the smoking factor with age until 50-54 years, after which a slight decrease occurred. A high correlation was found between the cigarette smoking and alcohol factors, and a small negative correlation for the physical activity and cigarette smoking factors. All three traits presented significant heritable factors in univariate analysis, although it was important to determine if this was due mostly to intracorrelations between traits and the high heritability of only some components or due to independent heritable factors in all three traits. In cluster analysis, both MZ and DZ twin pair members were in the same cluster more often than expected, but the overall MZ-DZ difference was relatively small. This indicated that genetic influence on the levels of the variables was probably relatively small. It appears probable that some of the increased disease concordance observed in twin pairs is due to an increased similarity in risk-factor behaviors. Familial aggregation of disease may also be partly due to familial aggregation of smoking, drinking, and physical inactivity.

82-0533. Kozlowski, L. T.; Director, J.; Harford, M. A. **Tobacco Dependence, Restraint and Time to the First Cigarette of the Day.** *Addictive Behaviors* 6(4): 307-312, 1981.

Early smokers (those who start smoking within 30 minutes of waking) were found to be more nicotine dependent and less successful in attempts to quit than late smokers (those who start smoking more than 2 hours after waking) in this three-part study of tobacco dependence. In the first part of the study, a questionnaire (180 questions) was administered on daily lifestyle (drinking, sleeping, eating, daily activities, other drugs used) to college undergraduates (54 females, 39 males) who smoked, with a set of questions on smoking habits and perceived reasons for such behavior. Late smokers gave as their main reasons for not smoking in the morning: (1) it made them feel dirty, smelly, or cigarettes didn't taste good (72 percent); and (2) it made them feel sick, dizzy, or experience physical discomfort (55 percent). Only one subject gave "restraint" as a reason for late smoking. The second part, a study of urinary pH, was unable to prove the hypothesis that those who were more sensitive to smoke had more alkaline urines (less excreted unmetabolized nicotine). The third part, of 50 adults (20 males, 36 females) in a 4-week smoking cessation program, found that those still abstinent 6 months later had waited longer to have their first daily cigarette than those who had resumed smoking. It was concluded that the timing of the first cigarette of the day may prove to be a useful indicator of the extent of a smoker's involvement with tobacco.

82-0534. Kumar, R.; Lader, M. **Nicotine and Smoking.** In: Essmac, W. B.; Valzelli, L. (Editors). *Current Developments in Psychopharmacology.* Volume 6. Spectrum Publications, 1981, pp. 127-164.

The pharmacology of nicotine and its effects in the central nervous system is reviewed. Both human and animal studies are considered, in relation to determining whether people smoke for the different pharmacological effects of tobacco, or whether smoking habits are governed by behavioral factors. The pharmacology of nicotine in the central nervous system, animal behavior related to these effects, the role of nicotine in smoking behavior in man, and the reinforcing properties of nicotine and smoking are discussed. Animal studies examined include: (1) tests of spontaneous motor activity, learning, and performance in rodents; (2) nicotine and brain mechanisms of reward; (3) studies of tolerance; (4) nicotine abstinence syndrome; and (5) nicotine self-administration. Specific human topics include: (1) measurement of nicotine and carboxyhemoglobin concentrations, (2) effect of nicotine yield on smoking behavior, and (3) nicotine arousal.

82-0535. Lawson, J. S.; Blatch, J. C. **Early Progress Report of Practical Experience With Lifestyle Programmes in a Suburban Population.** *Medical Journal of Australia* 2(3): 119-121, August 8, 1981.

An experimental program aimed at reducing health risks in northern Sydney, Australia, achieved behavioral and attitudinal changes in the short term for a range of lifestyle problems with the exception of cigarette smoking and alcohol consumption. The five components of the program are: (1) a parenting program aimed at parents of infants and young children; (2) an early morning daily television program aimed at children aged 6 to 12 years; (3) a pilot project within schools, aimed at improving the health of infants and schoolchildren; (4) programs for adults within the individual areas of circulatory and respiratory disorders, weight control, alcohol abuse, relaxation and stress management, exercise, tobacco abuse, and personal growth and self-esteem; and (5) lifestyle programs for health care professionals. Seven smoking cessation pilot programs, each conducted over a 4-week period, were completed. The greatest difficulty was in getting participants, resulting in such small numbers that the group educational approach appeared irrelevant when compared to the size of the problem. A larger scale antismoking program directed at children is now being developed along the lines of the Swedish nonsmoking generation campaign which involves coordinated use of the media, schools, and health agencies.

82-0536. Lichtenstein, E.; Antonuccio, D. O. **Dimensions of Smoking Behavior.** *Addictive Behaviors* 6(4): 365-367, 1981.

The interrelationships among topography and frequency measures were used in an attempt to specify the structural dimensions of smoking behavior. Twenty-four male smokers with an average age of 23.9 years and an average consumption of 20.8 cigarettes a day were observed while smoking in two 45-minute sessions. Six measures of smoking behavior were obtained: cigarette frequency, inter-cigarette interval, puff frequency per cigarette, percentage of tobacco burned, cigarette duration, and puff duration. A principal components analysis subjected to varimax rotation resulted in three orthogonal factors, each factor essentially consisting of a cluster of two of the earlier measures. The three factors were inhalation (puff frequency and puff duration); cigarette rate (frequency and inter-cigarette interval); and amount smoked (percent of tobacco burned and cigarette duration). The factors accounted for 80 percent of the variance. Both frequency and topography measures were stable over the two smoking occasions. Puff volume, however, was not measured, and may be an important and possibly independent topographic variable. (Auth. Abs. Mod.)

82-0537. Litt, I. F. **Substance Abuse Among Adolescent Females.** *Focus on Women: Journal of Addiction and Health* 2(2): 61-67, Summer 1981.

The frequency of smoking, and of other habits such as alcohol consumption and drug abuse, has increased among female adolescents in the past decade, leading to possible health hazards. From 1969 to 1979, the number of adolescent females who smoked increased from 22 percent to 27 percent of 13- to 17-year-old females. In 1979, female adolescent cigarette smokers outnumbered male adolescent smokers for the first time. The adverse effects of maternal smoking on fetal size and perhaps on fetal intellectual and physical development may especially affect adolescent mothers. Adolescents, particularly those under 15 years of age, are more likely to give birth to small-for-date babies, even without maternal smoking. Smaller babies are generally at greater risk for all complications as well as death during the neonatal period. The implications of substance abuse among female adolescents may be qualitatively and quantitatively different from those among male adolescents and males in general.

82-0538. Marshall, M. **Tobacco Use in Micronesia. A Preliminary Discussion.** *Journal of Studies on Alcohol* 42(9): 885-893, September 1981.

Smoking is an established habit in Micronesian society, where tobacco, which has been available to Micronesians for over a century, and to some for possibly 450 years, appears to be considered an essential item, not a luxury. Smoking is more common among men than women, relatively rare among children, and influenced by cultural prohibitions and practices. Most of the tobacco consumed in Micronesia comes from outside the islands, imported from places such as the United States and Japan. In the U.S. Trust Territory of the Pacific Islands, the population of about 120,000 people spent over 1.5 million dollars on tobacco products in 1976. Import duties on these commodities made up almost a third of all import taxes collected. Trust Territory Annual Reports apparently show that heart diseases and malignant neoplasms led the list of known causes of death from 1971 to 1973. A variety of respiratory ailments were also prominent on the list, including influenza, pneumonia, bronchitis, emphysema, asthma, and tuberculosis. It is suggested that a large portion of the deceased may have been heavy smokers. Health education programs could perhaps reduce the number of young smokers in Micronesia, but information is needed on why tobacco use is considered fashionable among Micronesian cultures. In addition, as long as the governments rely on tobacco taxes to generate a significant portion of their revenues, assistance for educational programs may be difficult to obtain.

82-0539. Myers, M. L.; Iscoe, C.; Jennings, C.; Lenox, W.; Minsky, E.; Sacks, A. **Federal Trade Commission Staff Report on the Cigarette Advertising Investigation. Public Version.** Federal Trade Commission, Division of Advertising Practices, Bureau of Consumer Protection, May 1981, 293 pp.

Five major issues are addressed in this investigative report on cigarette advertising: (1) the known health consequences of smoking, (2) the information concerning the dangers of smoking present in cigarette advertising, (3) the public's knowledge of the dangers of smoking, (4) the effectiveness of the current health warning, and (5) what remedial actions are necessary. Results indicate that today more consumers are aware that smoking is a health hazard than in 1964, but many smokers do not know the extent of the danger; however, many who smoke have switched to low tar/nicotine cigarettes. Consumers, especially women, need more information about the specific hazards of smoking. Recommendations for legislation include variation in size, shape, and wording of the health warning in cigarette advertisements, and rotation of its location and specific message. Also, placing limitations on the use of imagery (tombstone advertising), and disclosing carbon monoxide levels are encouraged. Expanded educational efforts are needed to supplement the health warnings of advertisements. Samples of cigarette advertising, proposed warnings, and a rotational warning system are included as appendices.

82-0540. Newman, I. M.; Duryea, E. J. **Adult Tobacco Use in Nebraska.** *Nebraska Medical Journal* 67(1): 7-8, January 1982.

The prevalence of cigarette smoking among Nebraska adults, as measured in a random sample (n = 1,880), tends to be lower than the national average. Nationwide, the proportion of adult smokers (age 17 or older) has declined to 33.7 percent, down from 36.9 percent in 1970, with most of the decline among males. In the Nebraska survey, 29.8 percent of males and 27.2 percent of females aged 18 or older were regular smokers; the incidence of smoking was lower than the national sample in all age groups for males and all age groups for females except the 18- to 24-year-olds and those over 65. An increased individual consumption rate, however, was found nationwide and among Nebraska males. Nebraska females were more likely to smoke 15-24 cigarettes each day than the national sample but less likely to be heavy smokers (more than 24 cigarettes/day). The Nebraska adults showed a preference for filtered cigarettes. A total of 14.8 percent of the males and 16 percent of the females judged their type of cigarette to be more hazardous than others. More than 50 percent of both male and female smokers believed they would still be smoking after 5 more years, but 83 percent of the males and 85 percent of the females showed a desire to quit if an easy method were available. Among the male sample 7.1 percent smoked a pipe, 7.4 percent smoked cigars, and 8.2 percent chewed tobacco. It is concluded that better ways are needed to help young people resist pressures to begin smoking and to help those who wish to quit smoking.

82-0541. O'Connell, D. L.; Alexander, H. M.; Dobson, A. J.; Lloyd, D. M.; Hardes, G. R.; Springthorpe, H. J.; Leeder, S. R. **Cigarette Smoking and Drug Use in Schoolchildren. II. Factors Associated With Smoking.** *International Journal of Epidemiology* 10(3): 223-231, September 1981.

In 1979, as the first stage in the evaluation of an educational strategy to discourage children from smoking, over 6,000 10- to 12-year-old primary schoolchildren in the Hunter Health Region (New South Wales, Australia) were surveyed to assess the prevalence of smoking and to identify some of the social and personal factors associated with this phenomenon. Logistic regression was used to determine the relative importance of the key factors in relation to the proportion of smokers in the "recent" and "regular" smoking categories. Friends' smoking behavior was the best predictor of the child's smoking status followed in order by approval of advertising, siblings' smoking behavior, amount of money available to spend weekly, sex, age, and finally parents' smoking habits. Other factors such as size of family, position of the child in the family, country of birth of parents, father's occupation, having a single parent, and urban or nonurban location of schools were all significantly associated with smoking prevalence in these children. Linear modeling techniques used in the analysis showed girls and nonsmokers to have less favoring attitudes to smoking and tobacco advertising than boys and smokers. Significant differences in knowledge occurred between categories of smokers. Most children claiming not to smoke disapproved of advertising and did not intend to smoke in the future. Among current smokers, some with neutral or favoring attitudes were ambivalent about future smoking. The data suggest that intervention in the field of cigarette advertising may be the most direct and effective means of reducing prevalence of smoking in children. (Auth. Abs.)

82-0542. Pederson, L. L.; Baskerville, J. C.; Lefcoe, N. M. **Multivariate Prediction of Cigarette Smoking Among Children in Grades Six, Seven and Eight.** *Journal of Drug Education* 11(3): 191-203, 1981.

Current smoking by schoolchildren was found to be related to age, socioeconomic status, school achievement, attitude, knowledge, and parental and peer smoking. To identify characteristics associated with high risk for smoking, a study was conducted which related variables measured in 1975 to smoking status in 1978 using multivariate statistical procedures. Sociodemographic characteristics, smoking environment, school performance, attitude toward smoking, and knowledge about the effects of smoking were included. Results were available for 1,082 children who were tested in 1975 in grades four through six and again in 1978 when they were in grades six through eight. Cross-sectional analysis of variables measured in 1978 revealed that six variables accounted for 37 percent of the variance in smoking status using a multiple regression model. Five variables that were measured in 1975 in the longitudinal analysis accounted for 32 percent of the variance in 1978 smoking status. The finding of no association with sex supports recent data which suggest that boys and girls no longer differ with regard to smoking. In addition, peer smoking has been shown to have the strongest relationship to current smoking, and may explain why programs which use peer leaders and offer instruction in dealing with peer pressure appear to be effective. The variables which were found to predict smoking status may offer some clues to the type of educational programs which might be used effectively; the importance of peer and parental smoking suggests that social acceptability may be one of the most important elements in initiation of smoking, and creating a social climate hostile to smoking may do more to reduce the prevalence of smoking among the young than any intense effort to communicate material on health and disease. (Auth. Abs. Mod.)

82-0543. Powell, E. B. **The War Against Smoking.** *Rhode Island Medical Journal* 63(8): 287-288, August 1980.

Poor results of smoking cessation programs should not discourage further attempts, particularly in the area of prevention of smoking. A Rhode Island survey of cigarette smoking among public school children found that initiation of cigarette smoking is not solely an individual psychological phenomenon, but is also influenced by family, friends, jobs, and school grades. Of the 2,600 children in grades 6, 8, 10, and 12, interviewed between December 1978 and April 1979, 14 percent of male students and 21 percent of female students reported smoking. Teenage females began smoking earlier than teenage males. A smoking parent was found to increase the possibility that a child would smoke by 50 percent, while the presence of an older sibling who smoked could quadruple the possibility. The presence of friends who smoked could also increase a child's likelihood of beginning the habit. The report sheds new light on the magnitude of the problem; it is a challenge to public health agencies and educators to develop new and more sophisticated approaches to cope with smoking among public school children, rather than return to more "traditional" methods of discouraging smoking.

82-0544. Rogers, D. **Juvenile Smoking—A Complex Issue.** *Tobacco Reporter* 108(1): 44-46, November 1981.

Despite several years of antismoking publicity, there is little indication that the attitudes of young people toward smoking have changed; smoking among children is still increasing in many countries. Some children begin to smoke at age 5, and about 80 percent of those who smoke regularly continue to smoke as adults. One third of British 16-year-olds smoke up to 10 cigarettes a day, and a large proportion of 11- to 13-year-olds are regular smokers. A recent survey showed that 35 percent of the 15- to 21-year-olds classed themselves as smokers. Some 37 percent of smokers under 21 began before age 13. These juveniles constitute an impressive market; British youngsters age 11 to 17 spent 94 million pounds on cigarettes last year, compared to 152 million pounds on candy, 50 million pounds on books and comics, 71 million pounds on soft drinks, and 25 million pounds on ice cream. The 11- and 12-year-olds alone spent one million pounds on cigarettes, and by age 15 to 17, cigarette expenditures had overtaken candy expenditures. However, little is really known about why young people smoke. The British tobacco industry has adopted some constraints, and has an agreement with the Government Health Department that cigarette advertisements will not be designed to appeal specifically to young people, or to present smoking as glamorous. The industry has sometimes withdrawn (on request) questionable sales promotions that appealed to youth, such as the Black Cat brand, which featured a low price and picture cards, and a promotion which offered discounts on records, holidays, and sports gear.

82-0545. Shor, R. E.; Williams, D. C.; Canon, L. K.; Latta, R. M.; Shor, M. B. **Beliefs of Smokers and Never Smokers About the Motives That Underlie Tobacco Smoking.** *Addictive Behaviors* 6(4): 317-324, 1981.

College students (202 smokers and nonsmokers), questioned about their beliefs about the benefits of smoking, tended to view smoking as an aid to coping with negative experiences rather than as a source of any positive benefits. Based on replies to 22 agree—disagree type questions on smoking motivation, both smokers and nonsmokers tended to diverge from the midpoint answer as a group, except that nonsmokers felt that smoking helps smokers fit in with their friends—an idea with which smokers disagreed. Smokers and nonsmokers agreed that smoking (1) helps smokers settle their nerves; (2) helps smokers feel calm, soothed, and relaxed; (3) helps them avoid weight gain; (4) gives them something to do with their hands; and (5) helps prevent the craving for smoking. They did not agree that smoking (1) helps smokers concentrate, (2) helps keep their minds clear, (3) helps them be more attentive, (4) helps them avoid things worse than smoking, (5) helps them achieve social poise, or (6) helps them forget their troubles. It is suggested that since most smokers take up the behavior in their adolescent years and probably form their beliefs about benefits and the social meanings of smoking in their preadolescent and childhood years, a systematic investigation of believed benefits from childhood to adulthood would be a productive area of investigation.

82-0546. Shor, R. E.; Williams, D. C.; Latta, R. M.; Canon, L. K.; Shor, M. B. **Differences Between Smoking and Nonsmoking College Students in Their Attitudes on Tobacco Smoking.** *Journal of Drug Education* 11(3): 261-274, 1981.

Divergence was found only in the difference in strength of attitudes about smoking in this study of whether smokers and nonsmokers hold systematically different attitudes on tobacco smoking. Twenty-seven smoking-related Likert type attitude items were selected for analysis from a longer questionnaire that had been administered to 61 smoking and 246 nonsmoking college students at the University of New Hampshire. Both smokers and nonsmokers expressed highly similar unfavorable attitudes toward smoking but nonsmokers expressed those attitudes more strongly. With few exceptions the findings were consistent over a considerable heterogeneity of item content. The hypothesis was generated that if investigators seek to find differences between smokers' and nonsmokers' attitudes that diverge from neutrality or uncertainty in different directions, they will for the most part fail. However, if they seek to find differences in the strength of such attitudes that are in the same direction of divergence, they will for the most part succeed. (Auth. Abs.)

82-0547. Stepney, R. **Would a Medium-Nicotine, Low-Tar Cigarette Be Less Hazardous to Health?** *British Medical Journal* 283(6302): 1292-1296, November 14, 1981.

Smokers apparently adjust their smoking behavior to achieve a pharmacological dose of nicotine according to this study of medium-tar smokers; however, since many of the adverse health effects of smoking are caused not by nicotine but by tar, it was hoped that these smokers would find that a medium-nicotine, low-tar cigarette provided an adequate nicotine intake without the need for oversmoking, thus reducing tar and carbon monoxide exposure. Smoking behavior and exposure to carbon monoxide, nicotine, and tar were studied in 19 middle-tar smokers. All smoked their own brands for 3 weeks and then switched to either a conventional low-nicotine, low-tar brand (control) or a medium-nicotine, low-tar cigarette for a further 3 weeks, the order then being reversed. The medium-nicotine, low-tar brand also had a low delivery of carbon monoxide. With the medium-nicotine, low-tar cigarette, mouth-level delivery and intake of nicotine was similar to that with the smokers' usual brands, and significantly greater than with the control low-tar cigarette. Intake of carbon monoxide from the medium-nicotine, low-tar cigarette was significantly less than with either own or control brands. With both low-tar brands mouth-level exposure to tar was reduced relative to smokers' usual cigarettes. There was no evidence, however, that the reduction in tar exposure was greater with the medium-nicotine brand than with the control low-tar cigarette. Both low-tar brands were "oversmoked" relative to subjects' usual middle-tar cigarettes. The medium-nicotine, low-tar cigarette was marginally more acceptable than the control brand, and the particular design used in the study resulted in a lower intake of carbon monoxide. In terms of reducing mouth-level exposure to tar, however, the medium-nicotine, low-tar cigarette had no advantage over the control low-tar product. In part this was because the ratio of tar to nicotine delivery obtained by human smokers was not the same as that obtained by a smoking machine. (Auth. Abs. Mod.)

82-0548. Vandenbroucke, J. P.; Kok, F. J.; Matroos, A.; Dekker, E. **Rookgewoonten van Nederlandse Huisartsen Vergeleken met die van de Bevolking. [Smoking Habits of Dutch Family Practitioners Compared With Those of the General Population.]** *Nederlands Tijdschrift voor Geneeskunde* 125(1): 4-6, 1981, Dutch.

A comparison of the results of an enquiry into the smoking habits of Dutch males in general and of male general practitioners between the ages of 25 up to and including 64 years reveals a slightly larger proportion of smokers among the general practitioners. Smoking general practitioners, however, differ from the smoking general population in the nature and amount of smoking materials consumed. Among smoking general practitioners, there are fewer cigarette smokers and more pipe and cigar smokers. Cigarette smoking general practitioners smoke fewer cigarettes than cigarette smokers in the general population. (Auth. Abs.)

82-0549. van der Linde, J. H. **The Smoking Epidemic. (Letter).** *South African Medical Journal* 60(12): 449-450, September 19, 1981.

Nonsmokers are characterized as being more concerned about health than smokers in this response to an earlier article (*South African Medical Journal* 59(18): 617-618, April 25, 1981) which recommended that doctors associated with the insurance industry should advocate reduced premiums for nonsmokers and higher rates for smokers. Nonsmokers appear to be more careful about diet, exercise, driving habits, hazardous pastimes, and alcohol. Higher socioeconomic classes experience lower mortality, and the proportion of smokers appears to decrease as the level of education increases. Smoking habits, lifestyle, socioeconomic class, and perhaps other factors may interact. In spite of the evidence, most insurers are still reluctant to accept that if present "average" premium rates are reduced for nonsmokers, the rates for smokers should be increased, perhaps substantially. See also 81-1976.

82-0550. Walckiers, D.; Lafontaine, A. **Le Tabac a Priser: Substitut Acceptable Pour les Fumeurs de Cigarettes? [Snuff: An Acceptable Substitute for Cigarette Smoking?]** *Archives Belges de Medecine Sociale, Hygiene, Medecine du Travail et Medecine Legale* 38(5/6): 323-330, 1980, French.

In view of the renewed popularity of snuff use in Germany, England, and Belgium, its effects on human health are examined. Previously published data on nicotine toxicity and its absorption by the snuff user are reviewed and compared with comparable data for cigarette or cigar smokers. The possible carcinogenicity of tobacco-specific nitrosamines, their presence in snuff, as well as rapid and important absorption of nicotine from snuff, are the risk factors which must be weighed against certain advantages of snuff dipping over smoking. It is concluded that the use of snuff is an alternative to smoking, but not a solution. It is an acceptable substitute for heavy smokers of cigarettes and a possible intermediate step to giving up tobacco use.

82-0551. Wallack, L. M. **Mass Media Campaigns: The Odds Against Finding Behavior Change.** *Health Education Quarterly* 8(3): 209-260, Fall 1981.

The effects of planned media campaigns to discourage use of drugs, alcohol, and tobacco are discussed. Pro-use advertising and funds far outweigh those of the health field, and pro-health media efforts have been embarrassingly ineffective compared to corporate promotional efforts. Changing behavior solely by media efforts is difficult. It is easier to modify a behavior than eradicate it; for instance, smoking risk minimization efforts (getting smokers to switch to low tar/nicotine brands) have been more successful than smoking-cessation campaigns. Antismoking programs which rely only on the media do not fare as well as those accompanied by a grassroots community effort. Also, such campaigns can backfire; an early public health movement against tobacco chewing contributed to an explosive increase in cigarette consumption around 1910. Individual antismoking events, such as the 1964 Surgeon General's Report, have relatively small and transitory effects on consumption; however, the cumulative impact of years of antismoking publicity has reduced predictable per capita consumption by one-third to one-fifth. A history of antismoking campaigns and a discussion of attitude-behavior therapy and the media are included.

See also, 82-0366, 82-0449, 82-0566

SMOKING PREVENTION AND INTERVENTION

82-0552. Arkin, R. M.; Roemhild, H. F.; Johnson, C. A.; Luepker, R. V.; Murray, D. M. **The Minnesota Smoking Prevention Program: A Seventh-Grade Health Curriculum Supplement.** *Journal of School Health* 51(9): 611-616, November 1981.

Preliminary results of a study of Minnesota seventh grade students comparing four smoking prevention curricula and a standard health curriculum group showed that the standard curriculum group moved out of the nonsmoking category at a higher rate than any of the four experimental groups and had a higher experimental and weekly smoking onset rate than the other groups. Of the 3,206 students from eight junior high schools who participated in the September 1979 pretest, 2,848 were present for the posttest in May 1980. Two competing strategies, a Short-Term Influences Curriculum and a Long-Term Influences Curriculum were compared. Within these broad strategies, several other variables were also tested: the use of same age peer opinion leaders or health educators as teachers, the use of media supplements, and the elicitation of a public commitment not to become a regular smoker. Program effectiveness was evaluated using self-report measures confirmed by saliva thiocyanate analyses. Results also revealed that: (1) the Long-Term Influences Curriculum significantly reduced the experimental smoking onset rate even beyond the reduction achieved by the Short-Term Influences programs, (2) the Adult-Led Short-Term Influences program similarly reduced the weekly smoking onset rate beyond the reduction achieved by the other treatment conditions, and (3) no significant differences were observed for the rate at which baseline nonsmokers took up smoking and quit by the time of the posttest. On the average, the experimental programs reduced the experimental smoking onset rate by 37.3 percent and the weekly smoking onset rate by 58.8 percent for baseline nonsmokers. (Auth. Abs. Mod.)

82-0553. Benfari, R. C.; Sherwin, R. (Editors). **Forum: The Multiple Risk Factor Intervention Trial (MRFIT). The Methods and Impact of Intervention Over Four Years.** *Preventive Medicine* 10(4): 387-553, July 1981.

In the 12,866 men at high risk of coronary heart disease selected for the trial, the initial reduction in cigarette smoking was greater than expected, but serum cholesterol did not drop as much as anticipated. The hypertension risk factors came close to the original predictions. There has been further reduction in each risk factor of the special intervention (SI) subjects from year to year (although within the usual care group, reductions in risk factors also occurred, contrary to original assumptions). The anticipated reduction in smoking was 55 percent for light smokers, 40 percent for moderate smokers, and 25 percent for heavy smokers. The SI model was based on behavioral methods, including focal groups, systematic desensitization, contingency contracting, and dissonance reduction. Wives were encouraged to become involved. Quit rates were strongly associated with initial level of smoking, and at the end of 4 years were 40.3 percent overall. Thiocyanate levels were determined to adjust the reported quit rates. Most recidivism occurred immediately after initial cessation. A dose-reduction program was offered to those unable to quit completely. The high cessation rate reflects the effectiveness of the interpersonal factors emphasized in the SI program.

82-0554. British Medical Journal. **A New Year Gift From the BBC to All Smokers.** *British Medical Journal* 283(6307): 1631, December 19-26, 1981.

The British Broadcasting Company's new series of 10-minute television programs to help smokers stop smoking, titled *So You Want To Stop Smoking*, are scheduled for broadcast at 5:45 Sunday evenings starting January 3. The programs are designed for smokers who want to quit, with emphasis on the benefits of quitting rather than on the risks of continuing to smoke. Two pilot programs were shown in 11 cities to two audiences: a group of interested professionals, and smokers who wanted to stop. The final programs center on a young, newly married girl, a married woman in her thirties, and a working-class Londoner in his forties and his wife, all of whom are trying to quit smoking. Television viewers can write in for an information pack, which includes a booklet, *So You Want to Stop Smoking*, a tabloid newspaper, a "contract" the viewer can use with a friend or spouse, and a letter from the presenter. Advertising will include the use of tee-shirts, badges, posters, and commercial radio station trailers. The Give Up Smoking kit has also been sent to all general practitioners, in the event patients request additional help after watching the programs.

82-0555. Engs, R. C.; Mulhall, P. F. **Again—Let's Look Before We Leap: The Effects of Physical Activity on Smoking and Drinking Patterns.** *Journal of Alcohol and Drug Education* 26(2): 65-74, Winter 1981.

The effectiveness of physical activity as part of drug and alcohol prevention programs for schools and communities is questioned following a university program which included students involved in active endurance (jogging and conditioning exercises; n = 135) and passive participatory (riflery, archery, and billiards; n = 155) types of exercise. Students' smoking and drinking patterns were examined before and after a 15-week period of engaging in the activities three times a week at 40 minutes a day. Results indicated no change in either smoking or drinking patterns for either group at the end of the 15-week period. Factors other than just vigorous exercise may be important for changes in drinking and smoking patterns, such as a conscious commitment to changing one's lifestyle. The factor of age might also be a reason for no significant change, as studies have shown that it is generally older adults or clinic populations which usually gain more positive mental health and lifestyles after exercise, while young adults and university students with little anxiety show little change. It is suggested that young people may smoke and drink for a variety of reasons including peer pressure, the need to prove masculinity and adult status, and not just to reduce the tensions and anxieties of daily living. Further research is recommended before such programs are presented as a panacea for preventing substance abuse in schools.

82-0556. Florey, C. du V.; Leeder, S. R.; Colquhoun, J.; Cullen, K. **Children and Smoking. (Letters).** *Medical Journal of Australia* 2(5): 255-256, September 5, 1981.

A report on the beneficial effect of intervention in preventing smoking in 12- to 14-year-old schoolchildren in Busselton, Australia, (*Medical Journal of Australia* 1(11): 586-587, May 30, 1981) is criticized; a reply from the report's authors follows. One problem cited is interpretation of answers to the single smoking question given: "How many cigarettes did you smoke last week?". This question could not be used to separate the experimenters from the "regular" smokers, was not likely to find the regular smokers consistently, and used by itself, seems subject to abuse by respondents. The before and after design of the study was not suited for evaluating the intervention, since no data were obtained on smoking behavior trends among children not in the program or on the effect of the questionnaire itself. There was little description of the intervention program and none on how the sample of children was obtained. No information was given on the smoking status of 11 children missing at the 1 year followup. There was no exploration of the recidivism implied by an increase in the prevalence of smoking between the first and second followup questionnaires. The authors reply that they believed it was suitable to intervene on the entire 12- to 14-year-old group of schoolchildren in the Busselton area, since controlled trials within schools might result in cross-contamination between control and experimental groups. The before-and-after design was used because no reason was seen to reject as controls some schools not exposed to the program. All the missing children had left the district and were not followed up. The authors know of no studies that successfully demonstrate an effect of smoking questionnaires on subsequent smoking behavior in children or adults. As for the possibility of recidivism, it is agreed that the study may not have stopped children from smoking once they began the habit, but they may have deterred a significant number from beginning for at least 1-2 years. See also 81-1962.

82-0557. Hjalmarsson, A.; Svardsudd, K. **Health Consequences of Giving Up Smoking in a Prospective Population Study of Middle-Aged Swedish Men.** *Acta Medica Scandinavica* 210(1/2): 93-96, 1981.

The beneficial effect of quitting smoking was studied in a prospective study of 855 50-year-old men randomly selected from the general population of Gothenburg, Sweden. These men have been followed since 1963. For this report, data from the first 10 years of followup were used. The survivors from this period were divided into three groups: those who had never smoked, those who were smokers at the first examination but not since (ex-smokers), and those who were smokers at all three examinations. Compared to ex-smokers, the smokers had a steeper decrease of bronchial peak flow, more days of sick leave, more complaints, and poorer self-assessed life situation score. After adjusting for possible confounding factors and differences in morbidity prevalence between the groups at the start of the study, the smokers still had a more unfavorable health course than the ex-smokers. (Auth. Abs. Mod.)

82–0558. Hullemann, K.-D.; Weber-Falkensammer, H. **Rauchverzicht in der Klinik. Einstellungen von Patienten im Heilverfahren. [Giving Up Smoking in a Clinic. Attitude of the Patients in Therapy.]** *Munchener Medizinische Wochenschrift* 123(36): 1327-1328, September 4, 1981, German.

Answers to a questionnaire filled out by 309 inpatients (46.9 percent male and 52.4 percent female) at a West German clinic are analyzed. The smokers made up 18.8 percent of all patients and consumed an average of 13.5 cigarettes/day/smoker. The plurality (40 percent) of the smokers consumed 11 to 20 cigarettes/day. About 62 (over 80 percent) of the nonsmokers were in favor of smoking prohibition at the workplace and the clinic, respectively, vs. only about 10 (36.2 percent) of the smokers. About one-half of all smokers would be willing to stop smoking in the clinic, if the 15.5 percent of undecided smokers are counted to be in agreement with smoking prohibition. A total of 88 percent of all surveyed patients were in favor of smoking prohibition, if the nonanswering patients are excluded. One-half of the smoking patients would stop smoking if urged to do so by their physicians, one-fifth would wait for additional motivation, and 13.6 percent make up the hard core smokers. Three-quarters of all nonsmokers, but only one-half of the smokers, were in favor of an antismoking campaign. Among the smokers, 44.7 percent have tried once to cease smoking; one-third of the total sample were ex-smokers.

82–0559. Kunze, M.; Herberg, D.; Gredler, B. **Ohne Rauch Geht's auch. Konzept, Durchfuhrung und Evaluation einer Aufklarungskampagne. [One Can Live Without Smoking. Concept, Conduct, and Evaluation of an Information Campaign.]** *Munchener Medizinische Wochenschrift* 123(36): 1329-1331, September 4, 1981, German.

The 8-week long antismoking campaign launched in November 1980 by the Austrian Federal Ministry of Health and Environment Protection is described as by far the most intensive ever conducted in Austria and internationally. The campaign's goals are stated under the motto "one can live without smoking." The campaign was a component of a long-term plan based on the "reduction of smoking-associated damage" concept. The three major constituents of the campaign were: (1) an information campaign through mass media, (2) a smoking cessation action for consenting smokers, and (3) attendant research to determine efficiency of the campaign. The goal of the information campaign was to promote a positive image of nonsmokers by means of media mix. The smoking cessation action consisted of sending out printed materials (cessation kit) to the participants, and posters to physicians. The research included: surveys of the general population, participants, and physicians; analysis of the cessation kit demand cards and the structure of the participant population; and monitoring reports on smoking and the campaign in the mass media.

82–0560. Lancet. **Reluctant Smokers—Watch B.B.C.!** *Lancet* 1(8262): 59, January 2, 1982.

A smoking cessation television program series is described. Six 10-minute B.B.C. Give Up Smoking (GUS) programs were designed to assist smokers, through support and practical advice, to quit. Since it is thought that six out of ten smokers want to give up smoking, the programs monitor the progress of four subjects determined to wean themselves off the habit and offer the reluctant smoker the most successful plan of action, concentrating on the increasing benefits of withdrawal and coping with weight gain. Some additional techniques include: (1) guest appearances of well-known figures who have quit, (2) offer of antismoking materials upon request, (3) contracts available to be made out with a friend, (4) program advertising on commercial radio stations, and (5) antismoking messages, with official authorization of Action on Smoking and Health and the Health Education Council, on independent television. The response to the GUS campaign has been encouraging, with 2,000 general practitioners requesting quantities of antismoking materials for distribution among their patients who smoke.

82–0561. Lichtenstein, E. **Social Learning, Smoking, and Substance Abuse.** In: Krasnegor, N. A. (Editor). National Institute on Drug Abuse Research Monograph 25, Department of Health, Education, and Welfare, Public Health Service, DHEW Pub. No. (ADM) 79-839, June 1979, pp. 114-127.

Intervention studies have failed in: (1) continuing to emphasize stimulus control and/or anxiety management in spite of lack of evidence for stimulus control of smoking, (2) ignoring the role of nicotine in maintaining the smoking habit, and (3) focusing on cessation without helping in maintenance of the nonsmoking habit. Recent studies from the Oregon Smoking Control Program which demonstrated modeling effects in the smoking habit showed that social circumstances affect whether and when a cigarette is smoked, but not puff rate, puff duration, or the amount of tobacco smoked. Nicotine fading programs, where the smoker progressively switches to lower tar and nicotine brands for 3 weeks prior to quitting, appear promising. Even those who resumed or continued smoking after the program stayed with the low tar and nicotine brands they smoked during the last phase of the program. Relapse and maintenance studies indicate that buddy systems improve the abstinence rates of any cessation technique, while coping skills training was not found to be effective.

82-0562. Monismith, S. W.; Shute, R. E.; St. Pierre, R. W.; Alles, W. F. **Opinions of Seventh to Twelfth Graders Regarding the Effectiveness of Pro- and Anti-Smoking Messages.** *Journal of Drug Education* 11(3): 213-225, 1981.

Adolescent smokers and nonsmokers agreed that antismoking messages on television are effective. A total of 3,100 seventh to twelfth grade students were surveyed regarding the perceived effectiveness of various components of both prosmoking and antismoking messages. The volunteer subjects were enrolled in 28 Pennsylvania junior and senior high schools. Survey results indicated that most adolescents were exposed to antismoking information and materials in schools. The materials were perceived to be of good quality, and most students understood the information which was presented. Antismoking messages were supportive in helping nonsmokers maintain their abstinence, but only one-third of smokers reported that antismoking messages influenced their choice to smoke or not to smoke. Statistically significant differences existed between smokers and nonsmokers in their receptivity to antismoking messages. Nonsmokers found antismoking messages to be interesting and they wanted to know more about smoking, while smokers often found antismoking messages to be boring and useless. Students claimed that promotional smoking advertisements did not seem to exert a direct influence on the adoption of smoking during adolescence. However, promotional advertisements were very effective in depicting smoking as enjoyable or pleasant, and reinforced adolescent smokers' desire to smoke. Antismoking messages on television were judged very effective in making people aware of the dangers of smoking, and over half of all respondents indicated that these messages make people want to stop smoking. (Auth. Abs.)

82-0563. National Cancer Institute. **Smoking and Health. A Program To Reduce the Risk of Disease in Smokers. Status Report—December 1979.** Division of Cancer Cause and Prevention, National Cancer Institute, National Heart, Lung, and Blood Institute, National Institutes of Health, December 1979, 130 pp.

The status of the Smoking and Health Program, with emphasis on activities conducted during 1979, is summarized. This report provides an itemization of major accomplishments of this past year, followed by a general summary of the historical background indicating the environment in which the program evolved, of the health consequences of smoking, and of approaches to reducing tobacco-related diseases. The goals of the Smoking and Health Program, its activities, accomplishments, and current status are presented, including the following specific objectives: (1) to identify individuals at high risk of developing tobacco-related diseases; (2) to develop and evaluate pharmacologic intervention approaches to smoking cessation; and (3) to determine the hazardous components of tobacco products and develop methods for minimizing or eliminating these components from smoker-acceptable tobacco products. A description of program funding is also included.

82-0564. Pederson, L. L.; Stennett, R. G.; Lefcoe, N. M. **The Effects of a Smoking Education Program on the Behavior, Knowledge and Attitudes of Children in Grades 4 and 6.** *Journal of Drug Education* 11(2): 141-149, 1981.

An investigation of the short term impact of a smoking curriculum for children in grades 4 and 6, the age just prior to which many children begin to smoke, is reported. Students in four grade 4 and four grade 6 classrooms were pretested concerning their knowledge about the relationship between cigarette smoking and health, their attitudes toward smoking, and their current smoking behavior. Half of the classrooms from each grade were subsequently exposed to a smoking awareness program by the teacher. The remaining students were not systematically exposed to smoking education material. All children were then posttested, using the same questionnaire that had been used in the pretest. Grade 4 children were less likely to have tried cigarettes than grade 6 children. Although all children had negative attitudes toward smoking, grade 4 children were significantly more negative than grade 6 children. The attitudes of the experimental group tended to become less negative while the control group's attitude remained constant. The experimental group improved more in knowledge than the control group with grade 6 children making a greater gain from pre- to posttest than did the grade 4 children. The absolute gains in knowledge scores were, however, relatively small, indicating the need for further modifications in the curriculum material. (Auth. Abs.)

82-0565. Staroselsky, L. **A Russian Speaks: A Poison Industry?** *Tobacco Reporter* 108(4): 46, 48, April 1981.

Soviet officials and government organizations continue to address the issue of smoking and health, and are seeking ways to reduce the health hazards of tobacco. One suggestion is to increase the proportion of cigarettes produced with filters and to enhance the antinicotine characteristics of filters. Tobacco varieties are evaluated by a Test Smoking Commission. In some areas, bans have been placed on tobacco product advertising and on the selling of tobacco in or near health and children's institutions. Research is attempting to develop tobacco varieties that are low in nicotine as well as productive. Farming methods, such as the use of nitrogen fertilizer, can also influence nicotine content. It is suggested that the only effective way of responding to tobacco's harmful effects is to quit smoking.

82-0566. Wright, F. E.; Drake, E.; Poole, S. **Women, Smoking Cessation, and Cessation Maintenance.** Ottawa, Ontario, Canadian Council on Smoking and Health, October 1980, 99 pp.

Data was collected from 204 Ottawa women to determine why some women continue to smoke, others never started, and still others were able to quit. Of the 204 women, 39 percent were current smokers, 27 percent were ex-smokers, and 34 percent were nonsmokers. Forty-six percent of the ex-smokers succeeded on their first attempt to quit and reported having experienced few problems when they quit. Ex-smokers in general reported having smoked fewer than 16 cigarettes/day and having smoked for a relatively short period of time. In addition, they more often lived with persons who did not smoke and had few friends and co-workers (under 20 percent) who smoked. More ex-smokers also reported knowing someone with chronic heart disease, lung cancer, or emphysema/chronic bronchitis, and having smoking-related ailments themselves. Regardless of their smoking status, 40 percent of the women who were under the age of 26 reported that both of their parents smoked. Among women over the age of 26, nonsmokers tended to report that neither parent smoked and smokers tended to report that one or both parents smoked. Seventy-four percent of the smokers had made at least one unsuccessful attempt to quit smoking, and of these, 38 percent had tried to quit within the last year. Smokers in general stayed off cigarettes for shorter periods of time than did ex-smokers on their unsuccessful attempts. Smokers more often reported having strong cravings, and being irritable and nervous during their attempts to quit. They appeared to receive as much social support as did ex-smokers but more frequently reported slipping back gradually or having one cigarette and giving up. (Auth. Abs. Mod.)

82–0567. Zganec, V. **Protection and Promotion of Mental Health and the Control of Problems Associated With Drinking, Other Dependences and Smoking in the Commune of Tresnjevka, Zagreb.** *Alcoholism* 16(1/2): 89-93, 1980.

A variety of programs and organizations have been set up in the Commune of Tresnjevka, Zagreb, in recent years to combat problems associated with smoking, drinking, and other dependencies. In 1979, a Coordinating Committee for the Protection and Promotion of Mental Health and the Control of Problems Associated with Drinking, Other Dependencies and Tobacco Smoking was formed. Several commissions were envisaged to implement various measures, among them a Commission for controlling smoking and other dependencies. In a program of measures for 1980, special actions in particular against smoking were planned.

See also, **82–0465, 82–0530**

SMOKING CESSATION METHODS

82–0568. Hopkins, S. J. **Chicle de Nicotina. [Nicotine Chewing Gum.]** *Medicamentos de Actualidad* 17(3): 99-103, 1981, Spanish.

A chewing gum containing nicotine which is bound to an ion-exchange resin, was developed for use in the cessation of smoking. Chewing partially compensates for the oral activity of the smoker, and the nicotine is slowly released during the chewing. The rate of release (largely dependent on the frequency and vigor of the chewing) averages about 90 percent of the nicotine released within about 30 minutes. The gum contains a buffer to maintain the alkalinity of the mouth at about pH 8.5, as at that pH the nicotine is well absorbed from the bucca mucosa, and the peak plasma level of nicotine is reached within 15-30 minutes. When chewed slowly, the use of the gum reduces the symptoms of nicotine withdrawal and gives considerable assistance to the well-motivated individual to give up smoking; however, success depends largely on motivation. Indications for use; contraindications, as in pregnant women; adverse reactions which may be expected; and possible overdose effects are discussed. Most patients require a dose of about 10 pieces of 2 mg gum daily, but frequency of use should be gradually reduced as dependence on nicotine is brought under control.

82–0569. Lando, H. A. **Effects of Preparation, Experimenter Contact, and a Maintained Reduction Alternative on a Broad-Spectrum Program for Eliminating Smoking.** *Addictive Behaviors* 6(2): 123-133, 1981.

Formal smoking cessation programs have been notoriously unsuccessful; however, the development of aversive conditioning, when intrinsically related to the natural smoking response, has produced some encouraging results. The present investigation compared the successful two-stage treatment reported by Lando (*Journal of Consulting and Clinical Psychology* 45(3): 361-366, 1977) against an intensified version of a less successful three-stage program also reported by Lando (*Behavior Therapy* 9(4): 666-668, 1978). Minimal experimenter contact and maintained reduction for nonabstinent subjects were also assessed. Subjects were 44 males and 56 females. Although there was a consistent trend for two-stage treatment to produce better results than three-stage treatment, differences between these conditions were significant only at 12-month followup. Intensive contact was superior to minimal contact only at Week 1. The maintained reduction procedure was not successfully implemented. Significant interactions were evident between experimenter contact and treatment stages throughout the followup period, with two-stage subjects appearing to benefit from intensive contact and three-stage subjects actually doing better under minimal contact. Results supported the efficacy of the two-stage aversion and maintenance program. Results also contradicted the assumption that more complex is necessarily better and pointed to the inadequacy of simple post hoc alternatives for nonabstinent subjects. (Auth. Abs. Mod.) See also 78-0555 and 80-0825.

82-0570. Martin, J. E.; Prue, D. M.; Collins, F. L., Jr.; Thames, C. J. **The Effects of Graduated Filters on Smoking Exposure: Risk Reduction or Compensation?** *Addictive Behaviors* 6(2): 167-176, 1981.

Two experiments were conducted to determine the effects of graduated filters on smoking behavior and health risk. Experiment 1 assessed the effects of four filters on carbon monoxide (CO) uptake (boost) in a smoker consuming five different brands of cigarettes. Progressive reductions in CO boost were found across graduated filters in four brands of filtered cigarettes. A nonfiltered brand of cigarette did not show CO changes across the filters. Experiment 2 consisted of an evaluation of the effects of the graduated filters on smoking rate, topography, and CO levels in four current smokers. Clear reduction in smoking rate and CO levels was found. Also, no compensatory topography changes resulted from the use of filters. Results are discussed in terms of the potential for significantly reducing tobacco exposure and the consequent risk reduction through employment of graduated filters. (Auth. Abs.)

82-0571. Nash, J. D. **Taking Charge of Your Smoking.** Palo Alto, California, Bull Publishing Company, 1981, 247 pp.

A comprehensive program to help people become permanent nonsmokers is presented, which goes beyond just helping the person quit smoking and gives him the tools to help make this major lifestyle change a change for life. For those who use smoking as a means of coping with stress, specific stress management techniques are taught, as well as how to reorient thinking into more self-enhancing patterns. For those who are highly dependent on the stimulation of nicotine, the program is structured to eliminate or reduce withdrawal symptoms. For everyone, the development of a new self-concept as a nonsmoker is encouraged, which is ultimately the key to quitting smoking permanently. Specific objectives include: increasing motivation to quit, behavioral education, urge management techniques, and weight management. A leader's guide is also available.

82-0572. Olms, J. S. **How To Stop Smoking: Effective New Acupuncture Point Discovered.** *American Journal of Acupuncture* 9(3): 257-260, September 1981.

A newly discovered acupuncture point was found to be effective in programs for the cessation of smoking. The acupuncture point is between the lung and large intestine meridians on the wrist. Smoking therapy administered from January 1980 to February 1981 on a total of 245 women and 290 men resulted in a success rate of approximately 84 percent. The total number of cases treated with this acupuncture method during the past 10 years showed a success rate of 75 to 80 percent.

82-0573. Shipley, R. H. **Maintenance of Smoking Cessation: Effect of Follow-up Letters, Smoking Motivation, Muscle Tension, and Health Locus of Control.** *Journal of Consulting and Clinical Psychology* 49(6): 982-984, December 1981.

Sending encouraging or supportive letters to smoking cessation subjects during the posttreatment period had no main effect in promoting maintenance, but did help reduce smoking in subjects who smoked from habit, showed low levels of sensorimotor stimulation from smoking, or showed high muscle tension. A total of 44 subjects participated in the smoking cessation program, of whom half received encouraging letters at the rate of three a week for followup weeks 1 and 2, two a week for weeks 3-6, and one a week until 3 months after treatment. Followup measurements of smoking data were made at 3 and 6 months. The treatment resulted in an initial abstinence of 93 percent. The letters showed no maintenance effect by themselves. At 6 months, letter and control subjects had smoking levels of 49 percent and 48 percent of baseline, respectively. Abstinence levels were 20 percent and 30 percent, respectively. For those who received low scores from sensorimotor measurements, the smoking levels at 3 months were 24 percent for those receiving letters and 48 percent for those who did not. At 6 months, the respective figures were 31 percent and 60 percent. The abstinence data were consistent but not significant. Among high scorers, letters actually increased smoking rates at 3 months and 6 months, perhaps by increasing awareness of missed pleasures of smoking. For those subjects who were considered high habit smokers, letters decreased smoking only at 6 months (39 percent vs. 68 percent for baseline). The comparison for low habit subjects and the abstinence data were not significant. The increased awareness of smoking derived from the letters may have been helpful to those who smoked mostly from habit, with little awareness of reward. Letters were associated with greater reductions in smoking rate among tense subjects (29 percent vs. 49 percent of baseline) and decreased reductions among relaxed subjects, at 3 months only. Abstinence data were not significant. Maintenance rates were promoted if the subjects had a strong belief that one controls one's own health, or if the subjects had a low belief that chance does, regardless of letter receipt. Letters were more effective among subjects expressing belief in others' power over one's health.

TOBACCO PRODUCT ADDITIVES

82–0574. Arthur D. Little, Inc. **Development of a Flavor System for Acceptability to Smokers of Candidate Less Hazardous Cigarettes. Final Report—Phase I, II, III and IV.** Report to Enviro Control Inc., Contract Number ECI-SHP-75-116, January 1977, 112 pp.

Of 277 organic chemicals screened for inclusion in less hazardous cigarettes, 146 were selected as having potential for developing and refining more complex flavoring mixtures. All agents screened were commercially pure, and of known structure and properties. Inhalation toxicities, if known, were considered in the selection process. Each was evaluated and rated with respect to five characteristics of cigarette flavor, including amplitude, tobacco, woody and other aromatics, irritation, and presence or absence of a sensation of wet mouth. This screening information was entered into an APL computer and selected for combinations ranging from two to six components. Two six-component systems were chosen as the base formulas for flavor refinement. The criteria were: balance of the intensities of the tobacco and woody-related aromatics, reduction of flavors and fragrances inappropriate to cigarette smoke, reduction of irritation factors such as tongue sting and throat burn, and increase in overall fullness of cigarette flavor. Four seven-component and two eight-component systems were ultimately constructed and identified as meeting the flavor criteria, and were then analyzed as to manufacturing feasibility. The contribution of nicotine to flavor, both as a free agent and as the bound salt in an artificial tobacco substrate, was also studied.

82–0575. Corbet, H. C.; Schurer, D. **Apparatus for the Determination of the Burning Velocity of Cigarette Paper.** *Beitraege zur Tabakforschung International* 11(2): 96-98, September 1981.

An apparatus which makes it possible to rapidly determine the burning time and the burning velocity of cigarette paper is described. The time needed to burn a standard length of 250 mm bobbin paper is measured in seconds. A length of 50 mm is used for leaflet papers of roll-your-own cigarettes. In a test of the apparatus, the mean value for the burn time of 25 bobbins was 85.0 seconds, with a standard deviation of 1.70 seconds. Samples were tested over a 2-week period by different operators. The apparatus was considered to have good reproducibility and repeatability. The apparatus was also used to test the burning time and burning velocity for 15 cigarette papers of different origins. Differing results were obtained, with some commercial papers failing to burn. The burning time and velocity for four types of roll-your-own paper were also determined, and again differing results were obtained.

82–0576. Sprecker, M. A.; Vock, M. H.; Schmitt, F. L.; Vinals, J.; Kiwala, J. **Acetyl Hydrindacenes, Acetyl Indanes, Mixtures of Same, Processes for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,305,411, December 15, 1981, 25 pp.

Acetyl hydrindacenes and acetyl indanes are presented as active ingredients to alter, modify, augment, enhance, or impart flavors and/or aromas to tobacco, as well as to foodstuffs and perfumes. Addition of acetyl hydrindacenes and acetyl indanes, singly or mixed, appears to produce a sweet, floral, musk aroma to tobacco before smoking and in both mainstream and sidestream smoke during smoking. Satisfactory results may be obtained at a proportion by weight of the active ingredients to the smoking tobacco material of between 125 ppm and 1,500 ppm, or between 1,000 ppm to 10,000 ppm proportion by weight of active ingredients to flavoring material. The ingredients may be dissolved in a suitable solvent which may either be spread on the cured, cased, and blended tobacco material or the tobacco material may be dipped into the solution. Under certain circumstances the ingredients may be applied by a suitable applicator such as a brush or roller on the paper or leaf wrapper, or on the filter by either spraying, dipping, or coating. Only a portion of the tobacco (or substitute) need be treated. The treated tobacco can then be blended with other tobaccos before the final product is formed. The invention may be used in the manufacture of smoking tobacco or in other tobacco products formed from sheeted tobacco dust or fines. The ingredients can also be incorporated with filter tip materials, seam paste, or packaging materials.

TOBACCO MANUFACTURING AND PROCESSING

82–0577. Baskevitch, N.; Chatelain, E. **Enzymatic Determination of Citrate and Acetate in Cigarette Paper.** Presented at the 7th International Tobacco Scientific Congress, Manila, 1980, 20 pp.

The enzymatic method (specific method) was compared to two nonspecific methods, conductimetry and sodium determination by atomic absorption, for determining acetate and citrate in cigarette paper. An excellent correlation in data occurs between the enzymatic method and conductimetry. Higher results are obtained with conductimetry for citrate and acetate due to the presence of other combustion salts in cigarette paper. The differences in results observed between the enzymatic method and sodium determination by atomic absorption are more variable. This can be explained by the presence of sodium in nonimpregnated paper prior to the introduction of acetate salt. The enzymatic method appears excellent for research laboratories when exact quantities of combustion salt must be determined. Analysis is quick, determinations may be performed in a wide range of concentrations without dilution, the method is specific, and preparation and determination of samples is easy. The cost, however, is a drawback, and conductimetry remains the best method for routine quality control checks in mass production.

82-0578. Boegli, S.; Lebet, J.-P. **Method for Producing Filter Cigarettes.** United States Patent No. 4,303,080, December 1, 1981, 11 pp.

Standardization of air permeability in porous or perforated filter cigarettes is demonstrated in this method for producing a filter cigarette in which dilution of the smoke can be precisely calculated and the extent of the dilution kept within a very narrow tolerance range. The filter unit is provided with a porous or perforated covering strip, with a porous or perforated connecting strip which joins the tobacco part to the filter unit. Adhesive is used to attach the covering strip to the outside of the filter element and to attach the connecting strip to the outside of the filter unit and the tobacco. The adhesive is applied to the surfaces of the covering strip and of the connecting strip which are inwardly oriented on the filter cigarette, by means of coating tracks. The coating tracks extend so that for any given relative displacement between the two strips in the longitudinal direction of the filter unit, the adhesive coating tracks on the covering strip and on the connecting strip always intersect at an angle or at right angles in the radial direction with respect to the longitudinal axis of the filter unit. (Auth. Abs. Mod.)

82-0579. Burruss, R. P., Jr. **Device for Evaporation and Inhalation of Volatile Compounds and Medications.** United States Patent No. 4,303,083, December 1, 1981, 7 pp.

A device which uses controlled volatilization and evaporation to release the volatile compounds of tobacco and other smoking materials is described. The smoking materials may be any organic material with suitable volatile ingredients, such as tobacco, licorice, eucalyptus, teas, flower petals, or other herbs. The volatile compounds are defined as constituents with a vapor pressure of 1 atmosphere at a temperature less than 250°C. The device includes a main structural body resembling an ordinary, hand-held tobacco pipe, with a heat-generating electrical resistance element located inside the "bowl" or cavity, but not in direct contact with the cavity walls. The volatile compounds, when placed on a heated surface in direct thermal contact with the heat-generating element, are vaporized. The compounds may be applied to the heated surface by a squeeze tube, eye dropper, syringe, or application rods; and air passages allow the vaporized compounds to enter an airstream to be inhaled by the user. The device includes an electrical power cord, although power requirements may be reduced to the point where batteries can be used. The desired volatile compounds may thus be inhaled while minimizing the toxic hazards associated with ordinary smoking methods.

82-0580. Keritsis, G. D. **Method for Selective Denitration of Tobacco.** United States Patent No. 4,301,817, November 24, 1981, 10 pp.

A process for extracting nitrate ions from an aqueous solution of tobacco material to reduce the delivery of various gas phase components from tobacco products is described. Tobacco materials are contacted with an aqueous solution, yielding an aqueous extract and an insoluble fibrous portion. Ion-extraction methods are then used to selectively extract the nitrate ions from the aqueous extract without substantially reducing potassium ion content. Avoiding potassium removal allows a proportionately greater reduction in nitrogen oxide delivery relative to the amount of nitrate removed than would be achieved if potassium ions were also removed. Ionic extraction methods include membrane electrodialysis, ion exchange, and Donnan dialysis. The treated extract can then be recombined with the insoluble fibrous tobacco portion. Tobacco products treated in this manner show a reduced delivery of nitrogen oxides, hydrogen cyanide, and carbon monoxide, during combustion. The process may be used with whole cured tobacco leaf, cut or chopped tobacco, tobacco filler, reconstituted tobacco, and tobacco stems.

82-0581. Lowe, R. H.; Bush, L. P.; Hamilton, J. L. **Chemical Modification of Burley Tobacco by Curing Regime.** In: Sullivan, S. (Editor). *Proceedings of the Fifth Workshop Conference.* Presented by the Tobacco and Health Research Institute, University of Kentucky, Lexington, November 1-2, 1979. 1980, pp. 57-81.

The effects of three different methods of curing Burley tobacco on the chemistry of the cured leaf and some smoke constituents were measured. The curing methods tested were the conventional, relatively slow air-curing process; an accelerated primed leaf curing procedure; and a homogenized leaf curing procedure (HLC). The results suggested that rate of drying, drying temperature, and level of oxygen might have contributed to an increase in nitrosonornicotine. Generally, total nitrogen, protein, and nitrate were higher under conditions where the tobacco cured more rapidly. The notable exception was nitrate in HLC tobacco. Total alkaloids and free amino acids were lower in the more rapidly cured tobacco. Smoke from reconstituted tobacco was lower in total particulate matter (TPM) and nicotine and higher in oxides of nitrogen than natural leaf. Adding oxygen during accelerated curing caused an increase in both gas phase and particulate phase cyanide of cigarettes made from these tobaccos. Static burn rate was lower and the nitrogen oxides higher per puff in accelerated-cured than in stalk-cured tobacco. (Auth. Abs. Mod.)

82-0582. Mattina, C. F. **Method of Making Reconstituted Tobacco Having Reduced Nitrates.** United States Patent No. 4,308,877, January 5, 1982, 3 pp.

A method for making reconstituted tobacco with reduced nitrates is described in which natural tobacco is extracted with water to produce a soluble extract and an insoluble fibrous residue. The aqueous extract is contacted by microorganisms, such as bacteria or fungi, capable of converting nitrate in the extract to nitrogen. Contacting may be done by adding a microorganism culture to a batch of the extract, or by passing the aqueous extract through a supply of the microorganiams. In either case, the contacting should take place in a substantially anaerobic environment. The culture may be produced by cycling a quantity of tobacco extract through a filtering medium to promote the growth of microorganisms occurring naturally in tobacco. Carbohydrate and/or protein is added to the denitrated extract to replace compounds depleted during the denitrating procedure. Ultimately, the denitrated extract is recombined with the fibrous tobacco residue. (Auth. Abs.)

82-0583. Schepartz, A. I.; Mottola, A. C.; Schlotzhauer, W. S.; Dejong, D. W.; Lam, J. J. **Effect of Ozone Treatment of Tobacco on Leaf Lipids and Smoke PAH: A Pilot-Plant Trial.** *Tobacco International* 183(25): 39-41, December 11, 1981.

Based on previous laboratory experience with the ozonization of tobacco at the Athens laboratory, a pilot-plant scale trial was conducted at the Tobacco Research Laboratory, Oxford, North Carolina. Analytical procedures used for lipid determinations consisted of direct hydrolysis of the tobacco followed by silicic acid column chromatography for fractionation of lipid components. Final quantitation was accomplished by gas chromatography. The lipids determined were solanesol, C_{18} unsaturated fatty acids, major sterols, and neophytadiene. Using a 100-pound slurry of SC-58 tobacco, ozonization produced noteworthy decreases in solanesol, C_{18} unsaturated acids, and sterols. These decreases, in turn, resulted in reductions in polynuclear aromatic hydrocarbons (PAH) of the tobacco pyrolyzate and cigarette smoke condensate. These findings showed that ozonization of tobacco on a pilot-plant scale is feasible for the production of cigarettes yielding reduced amounts of smoke PAH. (Auth. Abs. Mod.)

82-0584. Schmidt, O. K.; Hoge, W. H. **Tobacco Sheet Reinforced With Hardwood Pulp.** United States Patent No. 4,306,578, December 22, 1981, 6 pp.

A process for reinforcing reconstituted tobacco sheet with unrefined hardwood pulp is described, in which the tobacco sheet is prepared from an aqueous slurry with a high solids composition. Unrefined short fiber pulp such as hardwood pulp can reinforce the sheets when incorporated at relatively low levels, and is lower in apparent viscosity characteristics than softwood pulp refined to the same fiber length. The unrefined short fiber can therefore be used in higher solid slurry preparation, resulting in a more economical and efficient process. Delignified wood pulps predominating in hardwood species such as oak, gum, or poplar may be used, at levels as low as 2 to 12 percent by weight without refining in combination with 75 percent or more by weight tobacco. Other short fiber pulps such as unbleached and bleached bamboo, Esparto grass, bagasse, rice straw, and wheat straw may also be used. An adhesive agent or binder is included which is soluble (or dispersible) in water. The adhesive agent may be any of those conventionally used, such as the film-forming polysaccharide adhesive gums and their derivatives, the cellulose ethers and derivatives, polyuronides, algins and their derivatives, or others. The process is intended for use with tobacco, reconstituted tobacco, and tobacco waste such as stems or fines, as well as for various tobacco substitutes.

TOBACCO ECONOMICS

82-0585. Altschuler, D. E. **The Economics of Smoking.** *Financial Planner* 10(10): 111, 114, 116, October 1981.

Several economic factors which could be used, in addition to health concerns, as arguments for quitting or not beginning the smoking habit are presented, including the large sums spent on smoking; money which, it is suggested, could be better spent in other ways. Each year, Americans spend over 9 billion dollars on tobacco products, making domestic tobacco companies economically successful. A financial analysis of the benefits derived for a pack-a-day smoker, who quits and invests the savings for 30 years, shows a possible income of over 20,000 dollars per year for life starting 31 years after quitting—a strong inducement for quitting. Smoking is described as economically wasteful for employers, also: workers who smoke are more prone to accidental injury, cost a company more in medical care and health benefits, are more prone to premature death, and have lower productivity due to smoking on the job. Smoking can also result in higher office cleaning and repair costs, and lower the morale and productivity of nonsmoking workers exposed to smoke. Since many people are known to react negatively when told how harmful their actions are, positive suggestions relative to the economic benefits of not smoking may offer an alternative approach.

82-0586. Burry, R. D. **A Survey of Cigarettes in 1981.** *Tobacco International* 183(21): 18-22, October 16, 1981.

Economic data indicate that the domestic cigarette industry will remain a large and growing business during 1981. Monthly production and shipment figures available up to June 1981 suggest that 1981 will be the third straight year of sales increases and demand; products produced for export during the first 5 months of 1981 declined 3.5 percent from the same 1980 period, despite a strong foreign demand. Domestic production increased sharply in the most recent available month, with output going into both exports and U.S. markets. Price increases by cigarette producers have resulted in an acceleration in manufacturer prices. An April price advance instituted by producers was passed on by major cigarette wholesalers to retailers. Leaf tobacco price comparisons slowed significantly during late 1979 and early 1980 from the immediately preceding period. During the second half of 1980 and to date in 1981, raw material cost inflation accelerated slightly, although the rate of increase remained below that of 1979. Domestic media spending increased sharply in 1979 and 1980, but Philip Morris is the only major cigarette product producer continuing to increase domestic media expenditures. Media spending for domestic tobacco products increased 34 percent for the first 3 months of 1981. Industry leader R. J. Reynolds reduced media expenditures in late 1980 below the prior-year level, the reduction continuing into early 1981, with media outlays decreasing 19 percent during the first quarter. In 1980, American Brands increased domestic media spending by 9 percent; however, during the first 3 months of 1981, there was a 13 percent reduction in total outlays.

82-0587. Business Week. **How Cigarette Makers Aim To Fire Up Sales.** *Business Week* 2717: 65, 68, 70, December 7, 1981.

Cigarette manufacturers in the United States are responding to slow growth by targeting customers according to age, sex, and geography, and by increasing support for special promotions such as concerts and sports events. Despite 1 billion dollars spent on advertising for 1981, sales increased only 1 percent. Increased consumption between 1975 and 1979 has been primarily among females, both adolescents and adults. New male smokers increased 3.4 percent compared with 7.8 percent for female smokers, although male smokers still outnumber female smokers. During the same period, smoking among male adolescents dropped 25 percent while among females there was an increase of 40 percent. The fastest growing domestic brand for 1981, R. J. Reynolds' More, relies on women for 75 percent of its sales. The oldest women's brand, Virginia Slims by Philip Morris, had a 10.1 percent sales increase in 1981. Manufacturers continue to promote "masculine" brands, however, such as Reynolds' Winston and Camel. Manufacturers have also introduced family brand advertising where a few brands are presented in similar settings and are sometimes shown together. Promotional events are also being used to boost sales: Brown & Williamson promoting Kool through a Kool Jazz Festival; the Philip Morris Merit Report, an opinion poll on national issues, now conducted in a total of 60 cities; and the Camel Sports Scoreboard, already being offered to newspapers across the country. Other companies are introducing new brands and selective distribution to increase sales, and revamping advertising strategies for more effective marketing.

82-0588. Greenberg, D. **Washington's Smoky Politics.** *New Scientist* 92(1275): 194-195, October 15, 1981.

Tobacco continues to be a major national industry, far larger than the size of antismoking efforts. At present, the United States spends 1 billion dollars annually on tobacco advertising and a similar amount on cancer research. The Federal Government has cut the annual 3 million dollar antismoking effort in half during recent budget reductions. Cigarette sales have generally experienced annual growth, having now reached 621 billion units. A recent Gallup Poll indicates tht a large majority of smokers are aware of and believe in the warnings of the health hazards of smoking, but are opposed to a major crackdown. Although tobacco advertising was banned from television, the print media now receive more than 500 million dollars a year in tobacco advertisements.

82-0589. Maxwell, J. C., Jr. **How the Brands Ranked. Maxwell International Estimates.** *World Tobacco* 74: 105-106, October 1981.

Further worldwide economic statistics on cigarette market trends (a continuation of statistics presented in *World Tobacco* 73: 61-66, July 1981) give estimates for 1978-1980, including market division by types and the position of leading brands in Britain, Italy, France, South Korea, Turkey, Finland, and Norway. Estimates for Belgium are for 1979-1980, and the Iceland figures list only the position of some leading brands (and import sources) for 1980. Market shares of nations surveyed increased for filter cigarettes, while the nonfilter share decreased (no figures available for Iceland). Leading brands, by country, were: Britain, Benson & Hedges, with a 12.3 percent market share in 1980; Italy, M. S. Filter (35.0 percent); France, Gauloises group (42.6 percent); South Korea, Geobugseon 84 mm (40.0 percent); Turkey, Samsun 85 mm (43.4 percent); Belgium, Belga (27.3 percent); Finland, Marlboro (34.2 percent); Iceland, Winston king size (32.2 percent); and Norway, Prince 85 mm (17.5 percent). See also 81-2029.

82-0590. Sangenito, P. P. **Latin American Tobaccos: It's Up...It's Down...It's the Same.** *Tobacco Reporter* 108(10): 30-33, 44-62, October 1981.

Summary status reports of the Latin American tobacco industry are presented, divided into the following areas: (1) South America (Argentina, Brazil, Chile, Colombia, Ecuador, Paraguay, Peru, Uruguay, and Venezuela); (2) Central America (Costa Rica, El Salvador, Guatemala, Honduras, Mexico, Nicaragua, and Panama); and (3) the West Indies (Dominican Republic, Haiti, and Jamaica). Data on total tobacco production, grower prices, imports and exports, tobacco products, and manufacturing are included. Upswing factors present in some countries include better than average quality tobacco products, good international demand, and a tight domestic supply. In other countries, downslide is caused by credit availability, increased production costs, and the general economy. Antismoking and advertising ban campaigns in Venezuela and Argentina could effectively impact on sales, though not as effectively as the depressed economy in Guatemala which has converted many smokers into nonsmokers. Forecasts on the tobacco industries are also included.

82–0591. Tabak Journal International. **Lower Per Capita Income Countries Value Their Tobacco Consumption More.** *Tabak Journal International* 5: 418, October 1981.

United Nations 1975-1977 data on national disposable income per capita and consumption expenditures in 43 countries appear to indicate that, overall, the countries with lower per capita incomes tend to spend a larger share of disposable income on tobacco than do higher per capita income countries. The countries were divided into nine groups with Group 1 having the lowest per capita incomes and Group 9 the highest. As the per capita income rose, the share of expenditures on food, beverages, and tobacco taken together decreased. Variations within groups for percent shares of tobacco consumption were greater than those for food, beverages, and tobacco taken together. Although an inverse relationship was seen between per capita income and tobacco consumption, there were also significant national differences or exceptions; for example, the United Kingdom (Group 6) spent a larger share of disposable income on tobacco products than most of the lower income countries surveyed. The higher than expected percent share of tobacco expenditures in Group 9 may have been due to relative stability in consumption despite extremely high excise tax rates.

82–0592. Tabak Journal International. **Statistics.** *Tabak Journal International* 5: 422-428, October 1981.

Worldwide cigarette and cigar production statistics are presented. Listed are the annual average cigarette production of over 100 countries for 1971-1975 and for each year from 1976 to 1980, the percent of total yearly filter cigarette production for over 50 countries from 1971 to 1980, yearly cigar production in several countries for 1977 to 1981, and yearly cigar imports and exports of selected countries for 1977 to 1981. Total cigarette production for Europe, North America, Africa, South America, Asia, Oceania, and worldwide has increased since 1971. The percentage share of filter cigarettes has increased since 1971 in most of the countries listed. Statistics for cigar production, exports, and imports, while incomplete, showed mixed trends.

82–0593. Tobacco Institute. **Tobacco Facts: Tobacco Industry Profile 1980.** *Tobacco Reporter* 108(5): 48, 50, 52, May 1981.

Data on the U.S. tobacco industry, covering calendar year 1979, are presented. The output of cigarettes from U.S. factories totaled 704.4 billion units. Of this, 12.7 billion cigarettes were shipped to overseas forces, 1.2 billion to Puerto Rico and other U.S. possessions, and 79.7 billion to other countries. In the United States an estimated 19 billion dollars was spent on tobacco products. Total production was about 703,290 metric tons, out of a world total of 5.6 million metric tons. Total U.S. harvest was the smallest in 36 years due to a smaller planted area, a very wet growing season, and a leaf fungus. About 95 percent of the nation's tobacco is sold at auction in 157 markets. Tobacco was the sixth largest cash crop, behind corn, soybeans, all types of hay, wheat, and cotton. The Government continued its tobacco programs. Receipts from taxes on tobacco included excises, as well as Federal, State, and local taxes. A total of 143 factories with Federal permits manufactured tobacco products in 20 States. Some 69,900 persons were employed in tobacco manufacturing in 1979. An estimated 1.4 million retail outlets, 1,520 primary tobacco wholesalers, and another 1,054 miscellaneous wholesalers distribute tobacco products. The United States is the leading exporter of tobacco and the third largest tobacco importer. In 1979, exports of leaf tobacco and manufactured products totaled almost 2.2 billion dollars, while imports were about 463 million dollars, showing a positive contribution to the U.S. balance of payments of over 1.7 billion dollars. Over 99 percent of all leaf tobacco exported was shipped from the East.

82–0594. Tobacco International. **Loose-Leaf Lively at Lorillard, as Chewing Market Widens Nationally.** *Tobacco International* 183(21): 15, 17, October 16, 1981.

Loose-leaf chew accounts for about 70 percent of sales and is a major component of the growing chewing tobacco market, which also includes plug and twist/roll, that share a relatively small and shrinking percentage of the chewing market. The smokeless market also includes dry, moist, and fine-cut snuff. The dry snuff portion of the market is also shrinking. Loose-leaf chew is popular chiefly among younger males, who may prefer it because it is smokeless and can be used in no-smoking areas; it doesn't need handling, as do cigarettes, cigars, and pipes; and it is relatively free of the smoking and health controversy. Brand leaders in the loose-leaf market are: (1) Red Man, manufactured by Liggett & Myers' subsidiary, Pinkerton Tobacco Co., with 33 percent; (2) Conwood Corp.'s Levi Garrett with 23 percent; (3) Lorillard's Beech-Nut at 17 percent; and (4) Work Horse, manufactured by R. J. Reynolds, at 5 percent. Lorillard brands also include Big Red and Havana Blossom. Although the most recent reports show a decline in loose-leaf sales, it is generally thought that loose-leaf has a promising future. Usage continues to grow in work areas such as the San Joaquin Valley in California, lumbering areas in the Northwest, the North Central States, and the Northeast; areas which are outside the traditional market stronghold of the South.

82–0595. Tobacco International. **Sales Gains for Snuff and Smoking Tobacco.** *Tobacco International* 183(21): 24, October 16, 1981.

During the second quarter of 1981, sales increased for domestic pipe tobacco, roll-your-own smoking tobacco, and snuff, while chewing tobacco sales dropped. American-made pipe tobacco for domestic consumption rose 0.4 percent for the second quarter of 1981 compared with 1980. Snuff sales showed an 8 percent gain for the first and second quarters of 1981. The drop in chewing tobacco sales in the second quarter was <0.1 percent, the first since 1973. The shortfall was attributed to loose-leaf type chewing tobacco, which is apparently experiencing a pause for regrouping. There were gains in both plug and fine-cut types. Chewing tobacco sales for the first half of the year were 0.2 percent higher than during the same period in 1980, due to the major advance in the sale of plug tobacco.

82–0596. Tobacco International. **Hectarage, Production Rise From Slump; U.S. Brands May Be on Way.** *Tobacco International* 183(21): 5-11, October 16, 1981.

In 1980, Korea's tobacco planting area and tobacco production (farm weight) dropped to their lowest levels since 1971, but economic targets for 1981 call for a 17 percent increase over the 1980 target. Imports are expected to reach 15,307 MT in 1981, a 35 percent increase. Leaf export targets for 1981 call for an 18 percent increase over 1980, while the 1981 planned production of tobacco products (72.3 billion pieces) is almost the same as the 1980 level (71.1 billion pieces). In Korea, leaf is purchased by the Office of Monopoly (OM), which sets prices and extends agricultural supplies on credit. Most domestic tobacco products are sold through OM-licensed retail stores. Despite reduced production and imports of leaf in 1980, stocks were fairly high because of decreased consumption of domestic and foreign leaf. Korean leaf exports to the United States in 1980 were 11,223 MT compared to imports of U.S. leaf of 4,720 MT (42 percent of total imports compared with 60 percent in 1979). Tobacco product sales in the domestic sales showed a 75 percent increase, principally filtered cigarettes. The 1980 net income from tobacco products was equivalent to 761.2 million dollars, with a net income for 1981 set at 1 billion dollars. Several U.S. tobacco companies have approached the OM with proposals to license the manufacture of U.S. cigarette brands in Korea. Should agreement be reached, U.S. leaf imports are expected to increase significantly.

82–0597. Tobacco Reporter. **Spotlight—United Kingdom: A Time of Testing for the British Tobacco Trade.** *Tobacco Reporter* 108(12): 34-37, December 1981.

Smokers in the United Kingdom have compensated for high cigarette prices by switching brands, cutting back consumption, or by quitting altogether. Over the past 8 years, 28 percent of men and 14 percent of women have given up the smoking habit in Britain. Cigarette sales have slumped due to two massive tax increases. Sales dropped from 137 billion units in 1974 to 121.5 billion in 1980. Taxes form 74 percent of the final recommended retail price. Advertising bans, antismoking pressures by the health lobby, and the taxes imposed led to British smokers cutting down on consumption, and manufacturers have been unable to pull sales back to an earlier level. The industry has complained of discrimination, but this has not changed the Government's fiscal or pro-health stance. Manufacturers also face a more complete ban on advertising, making it difficult for them to build brand loyalty, or to introduce new brands, and thus to alter smoking patterns in favor of low-tar cigarettes.

82–0598. Tobacco Reporter. **Cigars and Cigarettes: A Look at 1980/81 Sales Trends.** *Tobacco Reporter* 108(12): 49-50, December 1981.

Cigarette sales were up 3 percent in the United States, in a period which saw the introduction of 17 new brands or spinoffs. Low tar markets continue to grow steadily. World cigarette production rose 1.4 percent; this slow growth rate is probably due to antismoking campaigns, increased excise taxes, higher retail prices, and in some countries, reduced incomes. The increases occurred mainly as smokers switched from roll-your-own to factory-made cigarettes. The Soviet Union, Japan, France, and Italy recorded declines. World cigarette trade rose 4 percent to an all-time high in 1980. Consumption of large cigars and cigarillos in the United States is expected to drop only fractionally from the 1980 total of four billion units. Cigar sales in Europe also remained relatively steady, with a decline of 0.4 percent. West Germany had the largest decline (1.5 percent), but continues to lead European sales; France showed a 5.2 percent increase from the year before. Denmark remains the highest per capita consumer of cigars and cigarillos (141 pieces in 1980), though sales dropped by 5.1 percent. Sales volume in Ireland and Italy remains small, though both showed increases of 7.5 and 7.2 percent, respectively.

82–0599. Tobacco Reporter. **Japanese Tobacco: A Front-Runner in Technology.** *Tobacco Reporter* 108(7): 30-31, 34, July 1981.

The Japan Tobacco and Salt Public Corporation (JTS), the country's only tobacco business entity, is considered to have some of the world's finest tobacco manufacturing techniques. The JTS has extended technical assistance, and sometimes tobacco manufacturing machinery as well, to overseas countries. About 35 million Japanese smoke 300 billion cigarettes annually, plus cigars and pipe tobacco. The JTS has 37 manufacturing plants, 12 raw material processing plants, and 5 sheet production facilities. The JTS holds the opinion that the health effects of smoking are not yet fully determined. The JTS does, however, disclose the tar and nicotine content of tobacco products, places caution labels on packs, regulates advertising and sales promotion, and supports expanded medical research. The JTS has made an effort to reduce the tar and nicotine content of its products. The weighted average index of all its cigarette brands has dropped from 17.6 mg of tar and 1.28 mg of nicotine in 1975 to 15.9 mg of tar and 1.05 mg of nicotine in 1980. The JTS restricts the use of radio and television in announcing new brands and in providing information about products.

82–0600. Wood, P. **German Tobacco: Prospects Far From Clear.** *Tobacco Reporter* 108(7): 36-38, July 1981.

Data are given on the tobacco industry in both West Germany and East Germany. A total of 161 billion cigarettes were manufactured in West Germany in 1980, up 3 billion from 1979. West German manufacturers and importing companies supplied over 6 billion packs, or 127.1 billion units in 1980, up 3.5 billion units from 1979. Smokers spent the equivalent of 8.7 billion dollars. The tax revenue on tobacco products and cigarette packs was about the equivalent of 5.2 billion dollars in terms of stamp duty. Three cigarette companies increased their supplies to the domestic market in 1980: Philip Morris (Munich), R. J. Reynolds, and BAT (Hamburg). Of 245 brands available in the total German market, 50 accounted for 97 percent of the market. Filter cigarette sales rose. There were 20 new cigarette introductions. West German cigarette imports showed only a slight rise in 1980, while exports declined. Future prospects for the industry are not clear. In East Germany (GDR), production is only 5,300 tons. The tobacco and cigarette industry is heavily dependent on imports. In 1979, the GDR imported 6.1 billion cigarettes and 19,900 tons of fermented and smoking tobaccos. The GDR population smokes heavily with an estimated 4.2 million men and 1.5 million women considered habitual smokers, each smoking between 5,500 and 6,000 cigarettes annually. The GDR health authorities have been attempting to combat these trends. Cigarette and tobacco advertising is banned, and smoking is forbidden in various public places.

82–0601. World Tobacco. **Change the Price-Support Program or Abandon It.** *World Tobacco* 74: 77-83, October 1981.

Dissatisfaction with the tobacco price support program is found in the Federal Government, and even among tobacco farmers; but disagreement persists over what, if any, modifications should be made. The general public appears to favor cuts in Federal spending and regulation in general, which makes the tobacco program particularly vulnerable since it is both highly regulatory and involves a significant government expense. Tobacco pricing was established in 1960, based on an index of all farm costs. Since tobacco technology has experienced significant cost-saving advances during the ensuing years, the level of support has risen far above actual production cost, making some U.S. tobacco less competitive with foreign tobacco. There has also been an increase in rentals of tobacco allotments to growers, and in the excess use of nitrogen by such growers to ensure the production of enough tobacco to cover rental cost. Although this excess nitrogen increases tobacco yield, it also lowers leaf quality. In addition, the tobacco may not have ripened by harvest time, making curing difficult. Suggestions for overcoming the obvious problems range from ending the program to modifying and updating it, to make prices more responsive to market forces.

See also, **82–0538**

LEGISLATION

82–0602. Lyons, M. S.; Taylor, M. W. **Farm Politics in Transition: The House Agriculture Committee.** *Agricultural History* 55(2): 128-146, April 1981.

The influence of farming interests (including tobacco farming) in the formation of farm policy in the House Agriculture Committee and Congress in general has declined in recent years. There appear to be two major reasons for this. First, the farm population has steadily declined during the past 3 decades, resulting in reduced farm representation in Congress. There has also been a shift in regional and commodity-group representation, wherein the importance of cotton and tobacco has diminished in the House Agriculture Committee in particular, while wheat, corn, soybeans, and livestock have gained in importance in committee considerations. Second, consumer awareness of and influence on congressional farm policy has increased. An illustrative example of the trend was the handling of a price support bill, H.R. 4296, reported by the House Agriculture Committee and passed by Congress in 1975. Although the bill was described as being very moderate and the result of compromise, it was successfully vetoed as unnecessary and wasteful by President Ford. The shift in the balance of power in farm politics, with farmers now an isolated minority, was seen in the 1976 election, in which President Ford still carried every traditionally Republican farm state. It is suggested that a return to previous policies is unlikely in the 1980s, but that farmer and consumer interests may be able to work together.

82–0603. Tobacco International. **Ban on Cigarette Ads in U.K. Now Unlikely.** *Tobacco International* 183(25): 29, 31, December 11, 1981.

The British tobacco industry is taking some comfort in the appointment of a new Minister of Health who, unlike his predecessor, will not be likely to ban cigarette ads in the United Kingdom. Dr. Gerald Vaughn, instead, is in favor of continuing voluntary arrangements rather than legislation. Sports sponsorship also seems to have escaped curtailment. The Advertising Association has spoken out against restrictions saying that an advertising ban would have no effect on consumption since advertising is used to provide a choice of brands and information, especially concerning low-tar products, for those who are already users of legal products. Examples are cited of failure to curtail consumption in countries where bans have been introduced.

See also, **82–0539, 82–0601**

GENERAL

82–0604. British Medical Journal. **Antismokers Under Attack.** *British Medical Journal* 283(6302): 1281, November 14, 1981.

Although smoking remains a major health hazard, the British Government appears to be undermining the efforts of antismoking officials. New leadership in the Department of Health and Social Security (DHSS) has stated that the government does not support regulatory or legislative means for controlling the commercial freedom of tobacco companies; and the grant by DHSS to Action on Smoking and Health, a leading antismoking organization, may be reduced. One doubtful, unproven concept introduced into public debate is the idea of a "safer" cigarette. However, it remains problematical whether such a cigarette can actually be developed. Public attitudes may be relaxing on smoking issues, but smoking remains the largest preventable cause of death in Britain.

82–0605. Schmidt, F. **Health Damage Through Forced Smoking.** Report on the Fourth World Conference on Smoking and Health, Stockholm, Sweden, June 18-21, 1979, 22 pp.

"Forced smoking" is used as a more appropriate term than "passive smoking" to describe the constraints placed on nonsmokers by smokers. Forced smoking is a real danger to health. More than 40 carcinogenic substances and at least a dozen cocarcinogens have been identified in tobacco smoke. The effects of carbon monoxide and nicotine are well documented. Also hazardous to forced smokers is the fine dust and particulate matter, and the inability of ventilation systems to remove these toxins from indoor air compounds the problem. Forced smoking may be considered a health hazard from acute effects alone—headache, nausea, allergic reactions, and burning eyes. Parliamentarians and industrial physicians are schizophrenic about this danger: less toxic substances such as cyclamates and butter yellow are restricted or banned, often at great financial burdens, while the harm of smoking in the workplace is often ignored, even though preventive measures would cost nothing. "However one looks at it: whoever advocates permission to smoke at work becomes an advocate of environmental pollution."

82–0606. South African Medical Journal. **MASA: Statement on Smoking.** *South African Medical Journal* 59: 471, March 21, 19[illegible].

The Medical Association of South Africa (MASA) believes smoking to have harmful medical and social effects, leading it to recommend several legislative actions and to seek the support of important influence groups. The recommended legislative actions include a ban on tobacco media advertising, a clear health warning on all purchases, a ban on cigarette-vending machines, the removal of tobacco products from open display shelves in supermarkets, no tax relief on tobacco promotion, a ban on cigarette sales to minors, and the establishment of maximum permitted nicotine and tar yields. The influence groups whose support MASA seeks include doctors and other health professionals; the Department of Health, Welfare, and Pensions; sportsmen and entertainers; insurance companies; and industry and commerce.

AUTHOR AND ORGANIZATIONAL INDEX

Aaro, L. E., 82-0507
Adams, J. D., 82-0314
Agnati, L. F., 82-0326, 82-0327
Albert, R. E., 82-0423
Alexander, H. M., 82-0541
Alexandersson, R., 82-0419
Alles, W. F., 82-0562
Altschuler, D. E., 82-0585
American Cancer Society., 82-0369
American Heart Association., 82-0437
Ames, R. G., 82-0404
†AMF Inc., 82-0584
Amin, S., 82-0312
Andersson, K., 82-0326, 82-0327
Angeloff, L. G., 82-0370
Antonuccio, D. O., 82-0536
Aravanis, C., 82-0458
Arch, P. J., 82-0470
Arkin, R. M., 82-0552
Arnott, M. S., 82-0348
Arrendale, R. F., 82-0322
Arthur D. Little, Inc., 82-0574
Asmal, A. C., 82-0441
Atkins, J. P., Jr., 82-0384
Attfield, M. D., 82-0404
Attiyat, A. S., 82-0298
Auerbach, O., 82-0438

Babaknia, A., 82-0480
Baccetti, S., 82-0334
Bachman, J., 82-0349
Bachman, J. G., 82-0531
Back, D. J., 82-0329
Baettig, K., 82-0508
Bagga, O. P., 82-0395
Baghurst, K. I., 82-0521
Bailey, A., 82-0477
Baker, R. R., 82-0299
Balls, L. D., 82-0483
Barancik, J. I., 82-0365
Bartsch, H., 82-0397
Bashir, K. R., 82-0492
Baskerville, J. C., 82-0542
Baskevitch, N., 82-0577
Basu, D., 82-0355
Bates, S., 82-0522
Baum, H., 82-0414
Baum, R. H., 82-0501
Bazhanov, N. O., 82-0493
Beaglehole, R., 82-0439, 82-0446
Becker, C. G., 82-0494
Bedenko, V., 82-0312, 82-0341, 82-0346
Begin, C., 82-0512
Benfari, R. C., 82-0509, 82-0553
Bensenberg, M., 82-0515
Berggreen, S., 82-0510
Bergler, R., 82-0511
Berglund, E.-L., 82-0507
Bergstrom, J., 82-0495
Bernard, P.-M., 82-0512
Bertram, J. F., 82-0405
Betts, T. E., 82-0406
Bevan, D. R., 82-0300, 82-0328
Bhide, S. V., 82-0301
Biener, K., 82-0513
Bing, R. J., 82-0440
Birkett, D. J., 82-0340
Biswas, N. M., 82-0496
Blackburn, H., 82-0458
Blatch, J. C., 82-0535
Blot, W. J., 82-0394
Blum, A. L., 82-0504
Boegli, S., 82-0578
Boger, E., 82-0341
Bolduc, P., 82-0407
Bone, R. C., 82-0514
Boreham, J., 82-0477
Bourke, G., 82-0452
Boyarskas, Yu. L., 82-0464
Bradley, R. F., 82-0441
Brasser, L. J., 82-0411
Brasseur, L., 82-0424
Breckenridge, A. M., 82-0329
Bridges, R. B., 82-0363
British Medical Journal., 82-0371, 82-0554, 82-0604
Brown, P. R., 82-0305
Bruhn, J. G., 82-0366
Bruninghaus, H., 82-0471
Brunnemann, K. D., 82-0302
Buiatti, E., 82-0334
Bull, T. B., 82-0416
Bulmer, D. R., 82-0492
Burch, J. D., 82-0372
Burruss, R. P., Jr., 82-0579
Burry, R. D., 82-0586
Busbee, D. L., 82-0348, 82-0373, 82-0390
Buscajoni, M., 82-0425
Bush, L. P., 82-0303, 82-0581
Busick, E. J., 82-0441
Business Week., 82-0587
Buzina, R., 82-0458

Caggiula, A., 82-0498
Callis, J. B., 82-0298
Calverley, P.M.A., 82-0408
Calvert, G. D., 82-0521
Campbell, W. B., 82-0453
Canon, L. K., 82-0545, 82-0546
Cantrell, E. T., 82-0348, 82-0373
Carmichael, I. A., 82-0309
Carrol, S. F., 82-0517
Casey, K. R., 82-0374
Castonguay, A., 82-0379
Caton, J. E., 82-0429
Cavelier, C., 82-0428
Chadha, M. S., 82-0301
Chakarvarti, S. K., 82-0330
Chaplin, J. F., 82-0322
Chassin, L., 82-0515
Chatelain, E., 82-0577
Chau, N., 82-0428
Chemistry and Industry., 82-0478
Chen, C.-H.B., 82-0311
Cheng, Y. C., 82-0332
Chinn, S., 82-0502
Chortyk, O. T., 82-0323
Chou, C. C., 82-0320
Chouroulinkov, I., 82-0351
Chowdhury, P., 82-0514
Chretien, J., 82-0409, 82-0410
Christensen, B., 82-0439
Christian, G. D., 82-0298
Christlieb, A. R., 82-0441
Chuttani, H. K., 82-0395
†Cie and Baumgartner Papier S.A., 82-0578
Clarke, G. C., 82-0502
Clarke, N. G., 82-0497
Classen, H. G., 82-0500
Clemmesen, J., 82-0375
Cluette, J. E., 82-0461
Clyne, C., 82-0470
Cohen, A. J., 82-0331

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Cole, P. V., 82–0470
Colley, J.R.T., 82–0411
Collins, F. L., Jr., 82–0570
Colquhoun, J., 82–0556
Copeland, G.K.E., 82–0309
Corbet, H. C., 82–0575
Corrao, G., 82–0337
Corty, E., 82–0515
Coulson, A. H., 82–0412
Crawford, F., 82–0329
Cross, K., 82–0329
Croteau, R., 82–0304
Crowe, M. W., 82–0483
Cucco, J. A., 82–0305
Cudak, C., 82–0345
Cullen, K., 82–0556
Cummins, R. O., 82–0516
Cushner, I. M., 82–0479

Dai, W. S., 82–0498
Daly, L., 82–0452
Davis, D. L., 82–0306
Davis, J. M., 82–0342
Dawley, H. H., Jr., 82–0517
De Haas, J. H., 82–0518
De Haas-Posthuma, J. H., 82–0518
Dejong, D. W., 82–0583
Dekker, E., 82–0548
Dement, J. M., 82–0386
Detels, R., 82–0412
Diamond, L., 82–0445
Digenis, G. A., 82–0332
Director, J., 82–0533
Divers, W. A., Jr., 82–0480
Djordjevic, B. S., 82–0458
Dmitriev, M. T., 82–0333
Dobbs, S. D., 82–0519
Dobson, A. J., 82–0541
Dolara, P., 82–0334
Dontas, A. S., 82–0458
Doody, M. C., 82–0358
Drage, C., 82–0418
Drake, E., 82–0566
Drath, D. B., 82–0413
Dube, M. F., 82–0313
Ducot, B., 82–0499
Dull, R. T., 82–0520
DuMouchel, W. H., 82–0385
Duryea, E. J., 82–0540
Duverger, A., 82–0351
Dwyer, T., 82–0521
Dysken, M. W., 82–0342

Eaker, E., 82–0509
Eckmann, F., 82–0471
Edreva, A., 82–0307
Ehrly, A. M., 82–0459
Eikmann, Th., 82–0335
Einbrodt, H. J., 82–0335
Eisenstadt, E., 82–0353
Ejiofor, A., 82–0414
Ekstrom, G., 82–0308
Elo, J., 82–0462
Elson, L. A., 82–0406
Ely, D. L., 82–0362
Eneroth, P., 82–0326, 82–0327
Enger, S. C., 82–0455
Engs, R. C., 82–0555
Erikssen, J., 82–0463
Ernst, E., 82–0442

†F. J. Burrus, 82–0578
Faden, A., 82–0415
Fagerstrom, K.-O., 82–0522
Fairweather, F. A., 82–0309
Falkeborn, Y., 82–0336
Falvo-Gerard, L., 82–0498
Fidanza, F., 82–0458
Fischer, G., 82–0459
Fischetto, G., 82–0425
Flanagan, S., 82–0461
Flenley, D. C., 82–0408
Florey, C. du V., 82–0502, 82–0556
Flynn, F., 82–0415
Forss, M., 82–0485
Fortic, B., 82–0376
Fortson, P. J., 82–0323
Foster, J. R., 82–0443
Fox, B., 82–0416
Frank, R., 82–0523
Fraumeni, J. F., Jr., 82–0394
Freed, B. R., 82–0332
Freeman, A. S., 82–0310
Friedman, P. J., 82–0417
Fuxe, K., 82–0327

Gallup Report., 82–0524
Ganda, O. P., 82–0441
Garfinkel, L., 82–0438
Genda, A., 82–0476
Gilbert, H. S., 82–0444
Gillespie, M. N., 82–0445
Gilli, G., 82–0337
Gingell, R., 82–0345
Girach, Z., 82–0346
Glueck, C. J., 82–0439, 82–0446
Going, R. E., 82–0501
Goldbourt, U., 82–0447
Goldman, H., 82–0481
Goldman, R. H., 82–0364
Gonvers, J. J., 82–0504
Gopalachari, N. C., 82–0297
Graham, I., 82–0452
Grandjean, P., 82–0338
Greden, J. F., 82–0525
Gredler, B., 82–0559
Greenberg, B. S., 82–0526
Greenberg, D., 82–0588
Greenhalgh, R. M., 82–0448
Gregson, R. L., 82–0339
Griffin, A. C., 82–0348, 82–0390
Grinkevichene, O. K., 82–0464
Gritz, E. R., 82–0527
Grossarth-Maticek, R., 82–0377
Grygiel, J. J., 82–0340
Gustavsson, H., 82–0308
Gutai, J. P., 82–0498
Gwynn, G. R., 82–0322

Haba, T., 82–0476
Hadzhiyska, E., 82–0307
Haines, C. M., 82–0449
Hakulinen, T., 82–0528
Halama, P., 82–0529
Halatek, D. G., 82–0363
Hamilton, J. L., 82–0581
Hamman, R. F., 82–0365
Hammer, R. E., 82–0481
Hankins, D., 82–0418
Hankinson, J. L., 82–0404
Hardes, G. R., 82–0541
Harford, M. A., 82–0533
Harris, J. E., 82–0385, 82–0450
Haskell, W. L., 82–0451
Hauknes, A., 82–0507
Hayashi, Y., 82–0482
Hearl, F. J., 82–0404
Hecht, S. S., 82–0311, 82–0312, 82–0314, 82–0341, 82–0379
Heckman, R. A., 82–0313
Hedenstierna, G., 82–0419
Heinonen, O. P., 82–0469
Heiss, G., 82–0451
Helgeland, A., 82–0455, 82–0456
Herberg, D., 82–0559
Herning, R. S., 82–0349
Hickey, N., 82–0452
Hicks, R., 82–0342
Hillerdal, O., 82–0392
Hillis, L. D., 82–0453
Hirayama, T., 82–0378, 82–0385, 82–0401
Hirsch, R. S., 82–0497
Hirsh, P. D., 82–0453
Hjalmarsson, A., 82–0557

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Hjermann, I., 82–0454, 82–0455, 82–0456
Hoffmann, D., 82–0302, 82–0311, 82–0312, 82–0314, 82–0341, 82–0346, 82–0379
Hoge, W. H., 82–0584
Hojnacki, J. L., 82–0461
Holck, S. E., 82–0380
Hollnagel, H., 82–0338
Holme, I., 82–0454, 82–0455, 82–0456
Hopkins, S. J., 82–0568
Hoshino, K., 82–0482
Howard, P. H., 82–0355
Howe, G. R., 82–0372, 82–0381
Hsu, S. C., 82–0501
Huber, G. L., 82–0413
Huchon, G., 82–0410
Hugod, C., 82–0457
Huhti, E., 82–0382
Hullemann, K.-D., 82–0558
Hund, M., 82–0321
Huong, N. D., 82–0428
Hurtig, J. B., 82–0492
Hutchison, D.C.S., 82–0414
Hyatt, B., 82–0345

Idle, M., 82–0477
Insull, W., 82–0439
†International Flavors and Fragrances Inc., 82–0576
Isaac, R. S., 82–0347
Iscoe, C., 82–0539
Ito, S., 82–0476
Iverson, D. C., 82–0530
Iwainsky, H., 82–0315

Jacobs, D., 82–0446
Jacoby, E. H., 82–0488
Jarvis, M. J., 82–0470
Jennings, C., 82–0539
Johansson, S.G.O., 82–0506
Johnson, A. W., 82–0322
Johnson, C. A., 82–0552
Johnston, L. D., 82–0531
Jones, R., 82–0407
Jones, R. T., 82–0349
Jones, S. T., 82–0324

Kaelin, M. A., 82–0383
Kametani, T., 82–0476
Kameyama, Y., 82–0482
Kanaya, H., 82–0476
Kaprio, J., 82–0532
Karvonen, M. J., 82–0458
Kastenbaum, M. A., 82–0385
Kaszubowski, P., 82–0343
Kauffmann, F., 82–0420
Kaufman, D. W., 82–0472
Kay, A. B., 82–0421
Keane, W. M., 82–0384
Keeler, R. F., 82–0483
Keritsis, G. D., 82–0580
Kew, R. R., 82–0461
Keys, A., 82–0447, 82–0458
†Kimberly-Clark Corp., 82–0582
Kimura, N., 82–0458
Kissinger, D. G., 82–0484
Kiwala, J., 82–0576
Knott, V. J., 82–0492
Koenig, C., 82–0500
Koizumi, J., 82–0476
Kok, F. J., 82–0548
Kolmodin-Hedman, B., 82–0419
Kornegay, H. R., 82–0385
Korte, A., 82–0344
Koskenvuo, M., 82–0532
Kottke, T. E., 82–0469
Kotze, T.J. van W., 82–0433
Kozlowski, L. T., 82–0533
Krolewski, A. S., 82–0441
Kronenberg, R., 82–0418
Kuller, L. H., 82–0402, 82–0475, 82–0498
Kumar, R., 82–0534
Kung, S. D., 82–0316
Kunze, M., 82–0559
Kuszynski, C., 82–0345
Kuwata, K., 82–0317
Kuzma, J. W., 82–0484
Kwiterovich, P., 82–0439

Lader, M., 82–0534
Lafontaine, A., 82–0550
Lakowicz, J. R., 82–0300, 82–0328
Lal, N., 82–0330
Lam, J. J., 82–0583
Lancet., 82–0387, 82–0560
Landgraf, H., 82–0459
Lando, H. A., 82–0569
Langenbach, R., 82–0345
Langley, D., 82–0353
LaPorte, R. E., 82–0498
Larsson, C., 82–0336
Latta, R. M., 82–0545, 82–0546
LaVoie, E. J., 82–0312, 82–0341, 82–0346
Lawson, J. S., 82–0535
Lazarus, L., 82–0342
Leavitt, S., 82–0345
Lebet, J.-P., 82–0578
Lebowitz, M. D., 82–0523
Lee, P. N., 82–0401
Leeder, S. R., 82–0541, 82–0556
Lefcoe, N. M., 82–0542, 82–0564
Leggett, R.J.E., 82–0408
Lehr, R. E., 82–0341
Lehtovirta, P., 82–0485
Leikauf, G., 82–0423
Leitch, A. G., 82–0421
Leitch, D. R., 82–0521
Lemen, R. A., 82–0386
Lenaerts, H., 82–0335
Lenox, W., 82–0539
Lepper, M., 82–0400
Leren, P., 82–0454, 82–0455, 82–0456
Lesser, J., 82–0342
Levy, R. I., 82–0460
Lewis, D. J., 82–0422
Lichtenstein, E., 82–0536, 82–0561
Liebow, A. A., 82–0417
Lilienfeld, A. M., 82–0365
Lindemeyer, R. G., 82–0501
Linn, S., 82–0486
Lippmann, M., 82–0423
Litt, I. F., 82–0537
Litzinger, E. F., 82–0306
Liu, S., 82–0400
Lloyd, D. M., 82–0541
Long, R. C., 82–0318
Loranskiy, D. N., 82–0388
Love, A.H.G., 82–0491
Lowe, R. H., 82–0581
Lubawy, W. C., 82–0347
Luepker, R. V., 82–0552
Lumb, E. M., 82–0421
Lund-Larsen, P. G., 82–0455, 82–0456
Lynn, D., 82–0313
Lyons, M. S., 82–0602

Mabuchi, H., 82–0476
MacDonald, E. J., 82–0385, 82–0401
Machado, L., 82–0506
Machalski, M., 82–0343
MacIver, M., 82–0329
MacMahon, B., 82–0389
Makey, A. R., 82–0416
Malick, L., 82–0345
Maliza, C., 82–0400
Manchester, D. K., 82–0487, 82–0488
Mantel, N., 82–0385
Marshall, M., 82–0373, 82–0538
Marshall, M. H., 82–0348

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Marshall, M. V., 82–0348, 82–0390
Martin, B. R., 82–0310
Martin, J. E., 82–0570
Martin, R. R., 82–0348, 82–0373, 82–0390
Massey, F. J., Jr., 82–0412
Matroos, A., 82–0548
Mattina, C. F., 82–0582
Mattina, C. F., 82–0306
Maxwell, J. C., Jr., 82–0589
Maxwell, W. A., 82–0519
Mayaux, M.-J., 82–0499
Mazzoli, S., 82–0334
McCann, J., 82–0391
McCulloch, M. W., 82–0356
McCullough, B., 82–0429
McDuffie, K. L., 82–0322
McLemore, T. L., 82–0348, 82–0373, 82–0390
McManus, I. C., 82–0349
McQuinn, R. L., 82–0332
Meltzer, L. T., 82–0350
Mendoza, E., 82–0415
Menotti, A., 82–0458
Meyer-Bisch, C., 82–0428
Michiels, F., 82–0351
Middleton, K., 82–0503
Miettinen, O. S., 82–0472
Milani, L., 82–0425
Miller, A. B., 82–0372
Mines, A. H., 82–0349
Minsky, E., 82–0539
Mitchell, J. A., 82–0481
Miyamoto, S., 82–0476
Moatamed, F., 82–0374
Moavero, N. E., 82–0424
Mohr, U., 82–0397
Monismith, S. W., 82–0562
Monson, R. R., 82–0486
Montgomery, L., 82–0345
Morris, L. E., 82–0394
Morrison, J. A., 82–0446
Morrison, J. E., 82–0517
Mostardi, R. A., 82–0362
Mottola, A. C., 82–0583
Mueller-Lissner, S. A., 82–0504
Mulcahy, R., 82–0452
Mulhall, P. F., 82–0555
Mulligan, J. J., 82–0461
Mur, J. M., 82–0428
Muramatsu, M., 82–0319
Murphy, P. A., 82–0475
Murray, D. M., 82–0552
Myers, M. L., 82–0539

Nagel, B., 82–0321
Nagpaul, K. K., 82–0330
Naik, S. R., 82–0395
Nakayama, A., 82–0476
Nash, J. D., 82–0571
National Cancer Institute., 82–0563
Nedeljkovic, S., 82–0458
Nemery, B., 82–0424
Nesnow, S., 82–0345
New Scientist., 82–0489
Newman, I. M., 82–0540
Nicolosi, R. J., 82–0461
Nissinen, A., 82–0462
Nitter-Hauge, S., 82–0463
Nordberg, A., 82–0336
Nou, E., 82–0392

Oakley, C. M., 82–0473
Obe, G., 82–0344
O'Connell, D. L., 82–0541
Ohta, M., 82–0476
Oiwake, H., 82–0476
Olms, J. S., 82–0572
Olsen, N. B., 82–0338
Olshavsky, R. W., 82–0515
Olson, A., 82–0483
O'Malley, P. M., 82–0531
Orlandelli, E., 82–0425
Orme, M. L'E., 82–0329
Orzechowska-Juzwenko, K., 82–0352
Osterman, K., 82–0506
O'Sullivan, J. P., 82–0406

Palisano, P., 82–0393
Pare, J.A.P., 82–0398
Patra, P. B., 82–0496
Paul, O., 82–0400
Pawlik, G., 82–0440
Pearle, J. L., 82–0426, 82–0427
Pederson, L. L., 82–0542, 82–0564
Peloquin, A., 82–0398
Perdrizet, S., 82–0420
Perper, J., 82–0475
Peter, P., 82–0504
Peters, J. M., 82–0364
Petkyavichyus, R. V., 82–0464
Pham, Q. T., 82–0428
†Philip Morris Incorporated, 82–0580
Philips, B. U., Jr., 82–0366
Phillips, G. F., 82–0309
Phillips, J. R., 82–0514
Phillips, T. C., III., 82–0490
Piade, J. J., 82–0314
Pizzone, G., 82–0425
Polarz, H., 82–0352
Poole, S., 82–0566
Popova, Ye. B., 82–0388
Pottern, L. M., 82–0394
Powell, E. B., 82–0543
Prajsnar, D., 82–0335
Prasad Rao, J.A.V., 82–0297
Pratap, A. I., 82–0301
Prentice, D. E., 82–0339
Presson, C. C., 82–0515
Procter, A., 82–0525
Prue, D. M., 82–0570
Puddu, V., 82–0458
Pukkala, E., 82–0528
Pung, T. C., 82–0320
Punsar, S., 82–0458
Puska, P., 82–0462, 82–0469
Putzrath, R. M., 82–0353

Rackl, A., 82–0440
Raman, S., 82–0492
Rana, M. W., 82–0361
Rand, M. J., 82–0356
Rawbone, R., 82–0416
Rawson, G., 82–0465
Raynor, W. J., Jr., 82–0400
Reddy, M. S., 82–0395
Redmond, C. K., 82–0402
Reese, M. E., 82–0466
Reger, R. B., 82–0404
Reid, L., 82–0407
Rhomberg, H. P., 82–0467
Richter-Reichhelm, H. B., 82–0397
Rickert, W. S., 82–0325
Riemer, S. C., 82–0300, 82–0328
Ripeckyj, A., 82–0342
Rivenson, A., 82–0379
Rivers, J. M., 82–0313
Robinson, J. C., 82–0325
Rochat, R. W., 82–0380
Roe, F.J.C., 82–0331, 82–0396
Roemhild, H. F., 82–0552
Rogers, A. W., 82–0405
Rogers, D., 82–0544
Rogers, W. R., 82–0429
Rokaw, S. N., 82–0412
Rom, W. N., 82–0374
Romano, C., 82–0354
Rona, R. J., 82–0502
Rosecrans, J. A., 82–0350
Rosenberg, L., 82–0472
Rosi, D., 82–0334
Roskamm, H., 82–0468
Rosner, B., 82–0486
Rossof, A. H., 82–0400
Rousseau, N., 82–0512

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Rowe, P. H., 82-0329
Russell, M.A.H., 82-0470
Rutishauser, M., 82-0430
Ryan, K. J., 82-0486
Rycroft, R.J.G., 82-0503

Sabadie, N., 82-0397
Sacks, A., 82-0539
Saeger-Lorenz, K., 82-0459
Salonen, J. T., 82-0462, 82-0469
Saloojee, Y., 82-0470
Salyayev, V. N., 82-0493
Sangenito, P. P., 82-0590
Santodonato, J., 82-0355
Saracci, R., 82-0397
Sarantos-Laska, C., 82-0356
Sardella, D. J., 82-0341
Sarma, J.S.M., 82-0440
Sarna, S., 82-0532
Sasseville, K., 82-0345
Satyanarayana Murty, B.V.V., 82-0297
Sayre, J. W., 82-0412
Schepartz, A. I., 82-0583
Schlesinger, R. B., 82-0423
Schlotzhauer, W. S., 82-0583
Schmid, P., 82-0504
Schmidt, F., 82-0605
Schmidt, O. K., 82-0584
Schmitt, F. L., 82-0576
Schoenbaum, S. C., 82-0486
Schoeny, R., 82-0321
Schraufnagel, D., 82-0398
Schrott, H., 82-0451
Schurer, D., 82-0575
Schwalb, H., 82-0471
Scursatone, E., 82-0337
Seldrup, J., 82-0357
Semenciw, R., 82-0372
Severson, R. F., 82-0322
Shachkute, A. A., 82-0464
Shaper, A. G., 82-0516
Shapiro, L., 82-0399
Shapiro, S., 82-0472
Shekelle, R. B., 82-0400
Shephard, B. C., 82-0497
Sherman, S. J., 82-0515
Sherwin, R., 82-0553
Shipley, R. H., 82-0573
Shivapurkar, N. M., 82-0301
Shor, M. B., 82-0545, 82-0546
Shor, R. E., 82-0545, 82-0546
Shorey, J. M., 82-0413
Shryock, A. M., 82-0400
Shupe, J. L., 82-0483
Shute, R. E., 82-0562
Sipahimalani, A. T., 82-0301
Slone, D., 82-0472
Smiddy, B. A., 82-0321
Smith, E., 82-0329
Smith, J. C., 82-0380
Smith, L. C., 82-0358
Smith, N. P., 82-0503
Snook, M. E., 82-0323
Soeldner, J. S., 82-0441
Sokoloff, J., 82-0417
Solberg, L. A., 82-0455
Sonin, B. V., 82-0359
Sonnenberg, A., 82-0504
South African Medical Journal., 82-0606
Spears, A. W., 82-0324
Spektor, D., 82-0423
Spira, A., 82-0499
Sprecker, M. A., 82-0576
Springthorpe, H. J., 82-0541
Squitieri, C., 82-0425
St. Pierre, R. W., 82-0562
Stack, D. J., 82-0461
Stamler, J., 82-0400
Stanescu, D. C., 82-0424
Staroselsky, L., 82-0565
Statland, B. E., 82-0360
Steiner, R. E., 82-0473
Stellman, S. D., 82-0403
Stennett, R. G., 82-0564
Stepney, R., 82-0547
Sterkowicz, S., 82-0474
Stok, E. T., 82-0503
Stoll, J. G., 82-0509
Stolley, P. D., 82-0472
Story, D. F., 82-0356
Strickler, D. P., 82-0519
Strohmeyer, G., 82-0504
Strong, J. P., 82-0455
Stubblefield, P. G., 82-0486
Sugrue, D. D., 82-0473
Sullivan, J. M., 82-0361
Svardsudd, K., 82-0557
Sypczynski, A., 82-0474
Szamosi, A., 82-0419

Tabak Journal International., 82-0591, 82-0592
Takeda, R., 82-0476
Takehira, Y., 82-0482
Talbott, E., 82-0475
Tamir, I., 82-0439
Tashkin, D. P., 82-0412
Tatami, R., 82-0476
Taylor, H. L., 82-0446, 82-0451, 82-0458
Taylor, M. W., 82-0602
Thames, C. J., 82-0570
Thaulow, E., 82-0463
Thompson, G. R., 82-0473
Thompson, W., 82-0491
Tilbury, R. S., 82-0332
Tobacco Institute., 82-0593
Tobacco International., 82-0594, 82-0595, 82-0596, 82-0603
Tobacco Reporter., 82-0597, 82-0598, 82-0599
Tollqvist, J., 82-0419
Tompa, A., 82-0345
Trayner, I. M., 82-0473
Tremblay, J., 82-0512
Trichopoulos, D., 82-0389
Trost, D. C., 82-0439
Tsai, H. T., 82-0320
Tso, T. C., 82-0316
Tsokos, C. P., 82-0401
Tulley-Freiler, L., 82-0346
Tuomilehto, J., 82-0462
Turchi, A., 82-0334

Uebori, M., 82-0317
Ueda, K., 82-0476
Ueda, R., 82-0476
Umemura, S., 82-0319

Vaddadi, K. S., 82-0505
Van Buchen, F.S.P., 82-0458
Vandenbroucke, J. P., 82-0548
van der Linde, J. H., 82-0549
Van Hamont, N., 82-0494
Vannucci, V., 82-0334
Van Vunakis, H., 82-0477
Vatne, K., 82-0463
Velve Byre, K., 82-0454
Victor, B., 82-0525
Vidas, M., 82-0384
Vinals, J., 82-0576
Vir, S. C., 82-0491
Vock, M. H., 82-0576
Vogel, E., 82-0504
Vogt, T. M., 82-0367

Wagner, H. M., 82-0344
Wagner, M., 82-0494
Wagoner, J. K., 82-0386
Walckiers, D., 82-0550
Wald, N. J., 82-0477
Wale, C. J., 82-0516
Walker, M., 82-0516
Wallack, L. M., 82-0551
Wang, N.-S., 82-0398

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

Wanner, A., 82–0431
Ward, G. W., 82–0449
Warram, J. H., 82–0441
Warren, C., 82–0321
Warren, C. W., 82–0380
Warshawsky, D., 82–0321
Weber-Falkensammer, H., 82–0558
Wehner, A. P., 82–0432
Weinberg, G. B., 82–0402
Weishaar, R., 82–0440
Wetmore, R., 82–0384
Weybrew, J. A., 82–0318
Wicht, C. L., 82–0433
Wilde, W., 82–0434
Wilkes, M. M., 82–0480
Willerson, J. T., 82–0453
Williams, D. C., 82–0545, 82–0546
Williams, F. P., III., 82–0520
Williams, O. D., 82–0446
Wingerson, L., 82–0368
Winkel, P., 82–0360
Winsel, K., 82–0315
Woebkenberg, N. R., 82–0362
Wood, P., 82–0600
Wood, P. D., 82–0451
World Tobacco., 82–0601
Worstell, D., 82–0362
Wray, N. P., 82–0348, 82–0373
Wright, F. E., 82–0566
Wu, M.-C., 82–0435
Wu, M.-M., 82–0412
Wu, S. C., 82–0320
Wyatt, R. J., 82–0363
Wynder, E. L., 82–0403
Wysocki, J., 82–0343

Yamazaki, Y., 82–0317
Yen, S., 82–0389
Yen, S.S.C., 82–0480
Young, J. C., 82–0325
Young, R., 82–0311

Zaborskis, A. A., 82–0464
Zamel, N., 82–0418
Zetterstrom, O., 82–0506
Zganec, V., 82–0567
Ziegler, R. G., 82–0394
Zwilling, B. S., 82–0436

---

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

SUBJECT INDEX

ABORTION
coffee consumption and smoking relationship, 82–0486

ADENOCARCINOMA
bronchioloalveolar carcinoma comparison, 82–0398

ADOLESCENTS
complex factors in initiation of smoking, 82–0543
effectiveness of smoking and antismoking messages, 82–0562
females, increased smoking, 82–0537
Great Britain, smoking prevalence and expenditures, 82–0544
health education and the smoking habit, 82–0530
high-density lipoprotein cholesterol levels, sucrose intake and smoking effects, 82–0439
high school seniors, cigarette and drug use, 1975-1980, 82–0531
Lithuania, atherosclerosis precursors in schoolchildren in Kaunas, 82–0464
Minnesota seventh grade health curriculum programs, 82–0552
Norway, smoking habit in boys vs. girls ages 12-15, 82–0507
predicting intentions to smoke, 82–0515
pulmonary disorders in juvenile smokers, 82–0430
stop-smoking efforts emphasizing dependency, 82–0514

ADVERTISING
adolescent smoking, reinforcement effectiveness, 82–0562
Federal Trade Commission investigation on cigarette advertising, recommendations for legislation, 82–0539
Great Britain, legislative ban for cigarettes unlikely, 82–0603
Great Britain, tobacco industry constraints related to youth, 82–0544
media use in modifying smoking behavior, 82–0551
shift from television to print media, 82–0588
smoking initiation, maintenance, and cessation relationship, 82–0511
targeting customer populations, 82–0587

AGE FACTORS
adolescents and young adults, effect of sucrose and smoking on high-density lipoprotein cholesterol, 82–0439
bronchial carcinoma in younger female smokers in Sweden, 82–0392
menopause, smoking effect, 82–0527
myocardial infarction in young age groups, smoking relationship, 82–0468

AGING
geriatric medication treatments, smoking effects, 82–0342
plasma testosterone level association with smoking, 82–0498

AGRICULTURAL PRACTICES
Burley tobacco constituent modification by curing, 82–0581
crop rotation effect on polyphenolic constituents of flue-cured tobacco, 82–0297
maleic hydrazide treatment of tobacco, N-nitrosodiethanolamine content, 82–0302

AIR POLLUTION
carboxyhemoglobin and methemoglobin levels in industrial and nonindustrial areas, 82–0362
N-nitrosamine content measurements, 82–0305
particulate matter effects on BaP metabolism in macrophages, mutagenicity of extracts, 82–0321
passive smoking as morbidity factor, 82–0333
respiratory disease and smoking relationship, 82–0412
respiratory disease, occupational exposure and smoking as risk factors, 82–0410
World Health Organization, respiratory indices in children, 82–0411

ALCOHOL CONSUMPTION
anesthesia dosage requirements in patients, high tobacco relationship, 82–0492
blood lead levels and smoking habit relationship, 82–0338
esophageal neoplasm risk factor, synergistic effect with smoking, 82–0394
Finland, relationship in twins to smoking and physical activity, 82–0532
laryngeal cancer, causal role compared to tobacco, 82–0372
marihuana and tobacco use relationship, 82–0520
mitotic index elevation in hamsters, smoke exposure effects, 82–0344
pregnant California women, drinking and smoking patterns, 82–0484
social class, town of residence, and smoking relationship, 82–0516
televised incidents on top-rated shows, 82–0526
Yugoslavia, control programs, 82–0567

155

ALDEHYDES
phenols and/or phenol ether mixtures, 82–0304

ALKALOID CONTENT
glass capillary gas chromatographic analysis, 82–0322
mass spectra for tobacco-derived nitrosamines, metabolites, and related compounds, 82–0311
senescence and curing, chemical transformations, 82–0318

ALKALOIDS, TOBACCO
content reduction in rapidly cured tobacco, 82–0581
nicotine and arecoline in dorsal hippocampus and mesencephalic reticular formation, 82–0350
nicotine levels in low-tar cigarettes, 82–0324
physiology and biosynthesis, 82–0303, 82–0306

ALLERGY
contact dermatitis in tobacco workers, 82–0503
occupational, frequency among smokers, 82–0506

ANABASINE
biosynthesis, 82–0303
glass capillary gas chromatographic analysis, 82–0322
mass spectra for tobacco alkaloid-derived nitrosamines, metabolites, and related compounds, 82–0311

ANTIBODIES
cocoa, coffee, ragweed, and tobacco as cross-reacting allergens, 82–0494
higher sputum/serum ratio of IgE in smokers, 82–0421
serum IgE, increased level in smokers, 82–0506

ANTIGEN-ANTIBODY REACTIONS
cocoa, coffee, ragweed, and tobacco as cross-reacting allergens, 82–0494
serum IgE, increased level in smokers, 82–0506

ANTIGENS
cocoa, coffee, ragweed, and tobacco as cross-reacting allergens, 82–0494
higher sputum/serum ratio of IgE in smokers, 82–0421
serum IgE, increased level in smokers, 82–0506

ANTISMOKING CAMPAIGNS
adolescents, survey of effectiveness, 82–0562
Australia, intervention program for schoolchildren, 82–0556
Australia, lifestyle improvement program, 82–0535
Austrian national campaign, 82–0559
budget cuts, 82–0588
Great Britain, decreasing government support, 82–0604
Great Britain, televised smoking cessation series, 82–0554, 82–0560
mass media, effects on smoking behavior, 82–0551
Medical Association of South Africa proposals, 82–0606
Soviet Union programs, 82–0565
Yugoslavia, Commune of Tresnjevka, 82–0567

ANTISMOKING MATERIALS
information kit for viewers of British antismoking television program, 82–0554, 82–0560

ANXIETY
reduction, effectiveness of physical activity in smoking prevention programs, 82–0555

AORTA
cholesterol influx, carbon monoxide effect, 82–0440

ARECA CHEWING
salivary secretions and flow rates in healthy vs. oropharyngeal cancer subjects, 82–0395

ARECOLINE
dorsal hippocampus and mesencephalic reticular formation, 82–0350

AROMATIC HYDROCARBON CONTENT
carcinogenic risk assessment for multiple exposures, 82–0355
ozonization of tobacco and smoke effect, 82–0583
spiked cigarettes to obtain biologically monitorable quantities, 82–0298

AROMATIC HYDROCARBONS
activation in specific organ cells from rats, 82–0345
activity in tumorous and normal lung tissue from lung cancer patients, 82–0397
benzo(*a*)pyrene teratogenic effect in fetal mice, relationship, 82–0482
benzofluoranthenes, carcinogenicity on mouse skin, 82–0312
cigarette tar yield vs. lung cancer, 82–0382
fluorescence spectroscopy in determining particle-assisted cellular uptake, 82–0300
nitrogen analogs identification, 82–0323
particle effects on rates of membrane uptake, 82–0328
pulmonary metabolism, inhibition by smoke exposure, 82–0347
tobacco smoke effect on biotransformation processes, 82–0315
tumorigenesis of dibenzo(*a, i*)pyrene and dibenzo(*a, h*)pyrene in mouse skin, 82–0341

ARRHYTHMIA
carbon monoxide effects on ischemic heart, 82–0443

ARTERIAL DISEASES
arterial reconstruction failure, smoking related, 82–0448
intermediate-density lipoprotein cholesterol in smokers, 82–0476

premature ischemic heart disease risk factors, 82–0463
thrombosis, reduced risk in smokers, 82–0442

ARTERIES
dose-related smoke effects on fibrous thickening of arterial walls, 82–0438
intermediate-density lipoprotein cholesterol in smokers, 82–0476
spasmatic activity of tobacco smoke components on coronary arteries, 82–0445

ARTERIOSCLEROSIS
carbon monoxide factor, 82–0440
coronary atheroma patterns, familial hypercholesterolemia vs. smoking effect, 82–0473
Oslo, Norway, study of raised lesions in coronary and cerebral arteries, 82–0455
precursors in schoolchildren in Kaunas, 82–0464
rabbit myocardia exposure to tobacco gas phase constituents, 82–0457
sudden cardiac death in women, smoking factor, 82–0475

ARYL HYDROCARBON HYDROXYLASE
inducibility in PAMs and lymphocytes, lung cancer correlation, 82–0373
pulmonary metabolism, inhibition by smoke exposure, 82–0347

ASBESTOS
abnormalities in exposed shipyard workers, smoking comparison, 82–0427
construction workers' exposure, 82–0419
exposure and smoking, 82–0374
fiber-enhanced membrane uptake of polynuclear aromatic hydrocarbons, 82–0328
inhalation effects in hamsters, smoking relationship, 82–0432
laryngeal cancer, causal relationship, 82–0372
lung cancer risk in smokers vs. nonsmokers, 82–0386
respiratory function effects among shipyard workers who smoke, 82–0426

ASTHMA
genetic factors identified in twins raised apart, 82–0418
respiratory function before and after smoking, 82–0425

ATTITUDES
adolescents, factors promoting initiation of smoking, 82–0543
adolescents, predicting intentions to smoke, 82–0515
children, characteristics associated with smoking initiation, 82–0542
college students, perceived benefits of smoking among smokers and nonsmokers, 82–0545
college students, smokers vs. nonsmokers, 82–0546
elementary smoking curriculum for grades 4 and 6, impact, 82–0564
Gallup Poll 1981, awareness of health hazards as factor in changes, 82–0524
hospital role models promoting smoking behavior, 82–0517
nonsmokers, lifestyle factors in reduced insurance premiums, 82–0549
West German clinic smoking prohibition, patients' opinion, 82–0558

AUSTRALIA
cholesterol levels in male service recruits, smoking factor, 82–0521
lifestyle improvement program, 82–0535
schoolchildren, prevention of smoking, 82–0556
social and personal smoking factors in children, 82–0541

AUSTRIA
antismoking campaign, 82–0559
coronary risk factor survey, 82–0467

AVERSIVE THERAPY
two-stage vs. three-stage treatment models, 82–0569

BEHAVIOR, ANIMAL
nicotine as a discriminative cue in the rat, 82–0354
nicotine effects, 82–0508, 82–0534
nicotine tolerance in rats, 82–0336
slug reactions to cigarette smoke exposure, 82–0434

BEHAVIOR, HUMAN
adolescents, factors promoting initiation of smoking, 82–0543
adolescents, predicting intentions to smoke, 82–0515
advertising relationship to smoking initiation, maintenance, and cessation, 82–0511
college students, perceived benefits of smoking among smokers and nonsmokers, 82–0545
comprehensive quit-smoking program, 82–0571
disposable income vs. tobacco consumption, worldwide, 82–0591
Gallup Poll 1981, changing U.S. smoking patterns, 82–0524
health intervention methods, 82–0509
hospital role models promoting smoking, 82–0517
letters during smoking cessation followup, effect, 82–0573
nicotine compensation in dependent smokers, 82–0522
nonsmokers, lifestyle factors in reduced insurance premiums, 82–0549
smoking despite health warnings, 82–0588
theories and suggestions for smoking intervention programs, 82–0561

BENZO(*A*)ANTHRACENES
- metabolism by human lymphocytes and PAMs, 82–0348

BENZO(*A*)PYRENE
- enzyme activity correlation in placenta, 82–0487
- kinetics of transfer between human lipoproteins, 82–0358
- lung cancer, high-tar vs. low-tar cigarettes, 82–0382
- lung preneoplastic lesions in rat embryo, correlation, 82–0351
- metabolism by human lymphocytes and pulmonary alveolar macrophages, 82–0348
- mutagenicity and metabolism in liver, lung, kidney, and bladder cells from rats, 82–0345
- mutagenicity of BaP-treated macrophage extracts from isolated perfused lungs, 82–0321
- pulmonary metabolism, inhibition by smoke exposure, 82–0347
- teratogenic susceptibility of fetal mice, 82–0482
- trace metal effects on metabolism in PAMs, 82–0390
- tumorous vs. normal lung tissue metabolism in lung cancer patients, 82–0397

BIDI SMOKING
- head and neck cancer relationship, 82–0384

BIOASSAY
- nicotine receptors in the rat, techniques for behavioral, pharmacological, biochemical studies, 82–0354
- phenazone metabolism in smokers vs. nonsmokers, 82–0352
- spiked cigarettes to obtain biologically measurable quantities of aromatic hydrocarbons, 82–0298

BIRTH DEFECTS
- benzo(*a*)pyrene effect in mice, 82–0482
- coffee consumption and smoking relationship, 82–0486
- congenital deformities in calves fed *nicotiana glauca*, 82–0483
- zinc status association and tobacco use, 82–0491

BIRTH WEIGHT
- adolescent maternal smoking effects, 82–0537
- benzo(*a*)pyrene effect in mice, 82–0482
- coffee consumption and smoking relationship, 82–0486
- maternal smoking effects, 82–0479, 82–0489, 82–0490
- relative vs. attributable risk from maternal smoking, 82–0387

BLADDER NEOPLASMS
- environmental factors, with smoking, 82–0403
- risk factors, 82–0381

BLOOD
- carboxyhemoglobin levels in smokers, 82–0335
- deformability of red blood cells through chronic inhalation of cigarette smoke, 82–0459
- lymphocytes in smokers and nonsmokers, 82–0343
- polycythemia, smoking as a possible factor, 82–0444

BLOOD CHEMICAL ANALYSIS
- lifestyle effect, 82–0360

BLOOD CHOLESTEROL
- acute inhalation of cigarette smoke, effects in pigeons, 82–0461
- coronary disease risk factor, 82–0469
- decreased serum levels, reduced coronary heart disease incidence, 82–0454
- physical activity relationship, 82–0451
- premature ischemic heart disease, risk factor, 82–0463
- sucrose intake and smoking associations, 82–0439
- weight, height, and ponderal indexes relationship, 82–0446

BLOOD CIRCULATION
- gingival flow reduction, epinephrine and nicotine effect, 82–0497
- uterine flow reduction, nicotine injection effects in rats, 82–0481
- uteroplacental blood flow, acute smoking effects in normotensives vs. hypertensives, 82–0485

BLOOD COAGULATION
- tobacco smoke exposure effects, 82–0466

BLOOD LIPIDS
- acute inhalation of cigarette smoke, effects in pigeons, 82–0461
- decreased serum levels, reduced coronary heart disease incidence, 82–0454
- physical activity relationship, 82–0451

BLOOD PRESSURE
- Finland, hypertension control program, 82–0462
- normotensive vs. hypertensive pregnant women, acute smoking effects, 82–0485
- premature ischemic heart disease, risk factor, 82–0463
- public knowledge and behavior trends, 82–0449

BODY HEIGHT
- children, parental smoking effects, 82–0502, 82–0510

BODY TEMPERATURE
- hypothermia in rats, nicotine effect, 82–0493

BODY WEIGHT
- asbestos and cigarette smoke exposure, effects in hamsters, 82–0432
- high-density lipoprotein cholesterol, smoking relationship, 82–0446
- reduction through tobacco smoke in rats, 82–0413

BONE DISEASES
- congenital deformities in calves fed *nicotiana glauca*, 82–0483

BRAIN
  nicotine and arecoline effect, 82–0350

BRONCHIAL DISEASES
  air pollution and smoking effects, 82–0412
  mediators of hypersensitivity, 82–0421

BRONCHIAL EPITHELIUM
  recovery in ex-smokers vs. current smokers, 82–0405

BRONCHIAL NEOPLASMS
  Sweden, smoking habit determinants in males vs. females, 82–0392

BRONCHITIS
  carboxyhemoglobin reduction of exercise tolerance, 82–0408
  reduced tracheal mucus transport, 82–0431

CADMIUM
  pregnancy, possible toxicity, 82–0478

CAFFEINE
  high level consumers, tobacco consumption relationship, 82–0525

CALIFORNIA
  smoking and drinking patterns among pregnant women, 82–0484

CANADA
  Gallup Poll 1981, smoking patterns and attitudes, 82–0524
  Quebec schoolchildren, smoking habits, 82–0512
  smoking initiation and cessation among women, survey, 82–0566

CANNABIS
  tetrahydrocannabinol recovery and quantification, 82–0310

CARBAMATES
  nitrosoureas and nitrosocarbamates, metabolic disposition, 82–0332

CARBOHYDRATES
  chewing tobacco content, oral cariogenic microorganism growth support, 82–0501

CARBON DIOXIDE
  formation mechanisms in cigarettes, 82–0299

CARBON MONOXIDE
  atherosclerosis relationship, 82–0440
  coronary disease, role in etiology of, 82–0470
  graduated filters in reduction of CO and smoking rate, 82–0570
  industrial and nonindustrial areas, 82–0362
  intra-individual variability of indices in firemen, 82–0428
  ischemic, arrhythmogenic effects, 82–0443
  mathematical models for CO delivery and butt length, 82–0325
  rabbit myocardia exposure effects, 82–0457
  reduced exercise tolerance in bronchitis and emphysema patients, 82–0408

CARBON MONOXIDE CONTENT
  Britain, cigarette yield decrease, 82–0309
  formation mechanisms in cigarettes, 82–0299

CARBOXYHEMOGLOBIN LEVELS
  arterial reconstruction failure, smoking related, 82–0448
  concentrations in pipe, cigar, and cigarette smokers, 82–0477
  coronary heart disease, possible synergistic effects of nicotine or other tobacco constituents, 82–0470
  correlation with smoking, 82–0335
  industrial and nonindustrial areas, 82–0362
  mathematical model to determine smoking effect, 82–0337
  polycythemia, tissue hypoxia associated with smokers, 82–0444
  reduced exercise tolerance in bronchitis and emphysema patients, 82–0408

CARCINOGENESIS
  environmental cancer education proposal, 82–0383
  environmental factors, 82–0396
  head and neck, epidemiologic considerations, 82–0384
  mechanisms of initiation and promotion, 82–0378
  occupational and smoking effects, worldwide literature review of, 82–0388
  polynuclear aromatic hydrocarbons in four cell types from rats, 82–0345
  risk assessment for multiple exposures to polynuclear aromatic hydrocarbons, 82–0355
  risk factors and cures, 82–0393
  tumorigenic activity of dibenzo(*a, i*)pyrene and dibenzo(*a, h*)pyrene in mouse skin, 82–0341
  urine studies in smoking vs. nonsmoking chemical workers, 82–0334

CARCINOGENS
  benzofluoranthenes, 82–0312
  lung treatments with benzo(*a*)pyrene or cigarette smoke condensate in rat embryo, 82–0351
  N-nitrosodiethanolamine, maleic hydrazide relationship, 82–0302
  nitrosoureas and nitrosocarbamates, metabolic disposition, 82–0332
  passive smoking, health hazards, 82–0605
  tobacco nitrosamines in hamsters, 82–0379

CARCINOMA, BRONCHOGENIC
  adenocarcinoma vs. bronchioloalveolar carcinoma, 82–0398

CARCINOMA, EPIDERMOID
- males, smoking as risk factor, 82–0376

CARDIOVASCULAR DISEASES
- declining death rates, smoking reduction relationship, 82–0460
- ethnic factors in the Old Order Amish, 82–0365
- incidence among smokers, women vs. men, 82–0527
- prostaglandin imbalance, smoking effect, 82–0453
- rabbit myocardia exposure to tobacco gas phase constituents, 82–0457
- risk factors in smoking mental patients, 82–0471
- smoking as risk factor in young patients, 82–0468

CARDIOVASCULAR SYSTEM
- ethanol and/or nicotine effect on chick embryo development, 82–0359

CATECHOLAMINES
- dopamine, increased turnover and reduced concentration from nicotine administration in rats, 82–0327
- epinephrine and nicotine, gingival blood flow reduction, 82–0497
- fetal adrenergic activity, maternal smoking effects, 82–0480
- lifestyle effect on clinical determination, 82–0360
- nerve terminal systems in hypothalamus, nicotine effects, 82–0326
- noradrenaline release in presence of tetrodotoxin, 82–0356
- norepinephrine levels in nicotine-injected rats, 82–0361

CELLS
- mitochondrial respiratory function, carbon monoxide effect, 82–0440
- particle-assisted uptake of aromatic hydrocarbons, fluorescence spectroscopy determination, 82–0300

CELLS, EPITHELIAL
- airway mitotic activity in rats, tobacco smoke effect, 82–0407
- tobacco smoke-induced laryngeal lesions in rats and hamsters, 82–0422

CENTRAL AMERICA
- tobacco industry economics by country, 82–0590

CENTRAL NERVOUS SYSTEM
- nicotine and arecoline effect, 82–0350
- nicotine effects in animals and humans, 82–0534

CEREBROVASCULAR DISORDERS
- risk factors, 82–0455

CESSATION OF SMOKING
- comprehensive behavior-change program, 82–0571
- coronary heart disease incidence and mortality reduction, relationship, 82–0450, 82–0454
- criticism and suggestions in smoking intervention programs, 82–0561
- economic arguments against smoking, 82–0585
- health consequences, 82–0557
- letters of encouragement during followup, 82–0573
- lung function improvement in ex-smokers, 82–0424
- lung neoplasm reversal effects, 82–0391
- mass media campaigns, effects, 82–0551
- MRFIT intervention trial for coronary disease risk reduction, 82–0553
- National Cancer Institute program for smoking-related disease risk reduction, 82–0563
- nicotine dependence, early daily onset of smoking as factor, 82–0533
- obstacles among chronic ischemic heart disease patients, 82–0474
- secondary prevention recommendations for infarction survivors, 82–0437
- sex ratio, reasons, methods, and problems, 82–0513
- taxation factors in Britain, 82–0597
- understanding habit factors in treatment, 82–0514
- women, factors affecting success or failure, 82–0566
- women, success rates, 82–0527

CESSATION OF SMOKING, METHODS
- acupuncture, success rate, 82–0572
- aversive therapy, two-stage vs. three-stage models, 82–0569
- behavioral interventions, review, 82–0509
- British television program series, 82–0554, 82–0560
- nicotine chewing gum, 82–0568
- nicotine fading programs, 82–0561

CHEWING TOBACCO
- decreased sales for second quarter 1981, 82–0595
- loose-leaf market trends, 82–0594
- nitrosamine content, Indian variety, 82–0301
- oral cariogenic microorganism growth, effect on, 82–0501

CHILDREN
- Australia, prevention of smoking, 82–0556
- Australia, social and personal factors in smoking habit, 82–0541
- body height, parental smoking effects, 82–0502, 82–0510
- characteristics associated with smoking initiation, 82–0542
- elementary educational smoking curriculum, short term impact, 82–0564
- Great Britain, smoking prevalence and expenditures, 82–0544
- high-density lipoprotein cholesterol levels, sucrose intake and smoking effects, 82–0439
- Lithuania, atherosclerosis precursors in schoolchildren in Kaunas, 82–0464

maternal smoking during pregnancy, effects on mental and physical development, 82–0489
Norway, smoking habit in boys vs. girls ages 12-15, 82–0507
respiratory indices and air pollution, 82–0411
school health program, primary grades for smoking education, 82–0512
stop-smoking efforts emphasizing dependency, 82–0514

CHROMIUM
neutron activation analysis of cigarette tobacco, 82–0320

CHROMOSOMES
mitotic index elevation in smoke-exposed hamsters, 82–0344

CHRONIC BRONCHOPULMONARY DISEASE
reduced tracheal mucus transport, 82–0431
sex ratios in South African smokers, 82–0433

CIGAR SMOKE
air-cured and fermented tobacco, health hazards in rats, 82–0406

CIGAR SMOKING
head and neck cancer relationship, 82–0384
serum cotinine levels, comparison with pipe and cigarette smokers, 82–0477

CIGARETTE PAPER
apparatus for determining burning time and velocity, 82–0575
enzymatic method for determination of citrate and acetate, 82–0577

CIGARETTE SMOKE
analysis of parsley, marihuana, and tobacco samples, 82–0310
chrysotile asbestos, combined effects in hamsters, 82–0432
flue-cured Virginia tobacco, health hazards in rats, 82–0406
N-nitrosamine content measurements, 82–0305
N-nitrosodiethanolamine content, maleic hydrazide relationship, 82–0302
nicotine effects on lung function, 82–0315
nitrogen analogs of polynuclear aromatic hydrocarbons, identification, 82–0323
product formation mechanisms, 82–0299

CIGARETTES
mathematical models for relationship between chemical deliveries and butt length, 82–0325
Maxwell international estimates of market trends, 82–0589
neutron activation analysis of trace elements, 82–0320
worldwide production trends, 82–0592

CIGARETTES, HIGH-NICOTINE
compensation behavior of smokers, 82–0522

CIGARETTES, LESS HAZARDOUS
Britain, yield decrease, 82–0309
chemical additive flavoring systems, 82–0574
denitration of tobacco by ionic extraction, 82–0580
manufacturing method for standard smoke dilution, 82–0578
smoke effects of high tar/nicotine vs. low tar/nicotine cigarettes on arterial walls, 82–0438
Soviet research, 82–0565
tar/nicotine ratio relationship to smoking habit, 82–0547

CIGARETTES, LOW-NICOTINE
Britain, yield decrease, 82–0309
compensatory smoking behavior, 82–0349, 82–0522
Soviet research, 82–0565
tar levels and compensatory smoking relationship, 82–0547

CIGARETTES, LOW-TAR
Britain, yield decrease, 82–0309
market share growth, 82–0324
nicotine levels and compensatory smoking relationship, 82–0547
possible mortality reduction, 82–0382

CIGARETTES, NONTOBACCO
analysis of parsley and marihuana samples, 82–0310

CIGARS
worldwide production trends, 82–0592

CILIARY ACTIVITY
slug reactions to cigarette smoke exposure, 82–0434
ultrastructural abnormalities in smokers vs. nonsmokers, 82–0416

COFFEE CONSUMPTION
caffeine vs. tobacco consumption, 82–0525
pancreatic cancer, smoking relationship, 82–0371, 82–0389
pregnancy, effect with smoking, 82–0486

COMBUSTION TEMPERATURE
cigarette product formation mechanisms, 82–0299
tobacco char oxidation analysis, 82–0319

CONTRACEPTIVES, ORAL
cardiovascular disease, interactive risk factor with smoking, 82–0527
myocardial infarction in women, 82–0472
steroid therapy, smoking relationship, 82–0329

CORONARY DISEASE
arteriosclerosis patterns, familial hypercholesterolemia vs. smoking effect, 82–0473

Austrian risk factor survey, 82–0467
cholesterol levels in male service recruits, smoking as factor, 82–0521
cholesterol, lipids, and smoking as risk factors, 82–0447
chronic ischemic heart disease patients, smoking habits, 82–0474
diet and smoking in all-causes death rate, 82–0458
dose-related smoke effects of fibrous thickening of arterial walls, 82–0438
Finland, risk factor clustering in men vs. women, 82–0469
Ireland, trends in cigarette smoking, 82–0452
MRFIT intervention trial, reduction of smoking risk factor, 82–0553
nicotine and carbon monoxide as causal factors, 82–0470
premature ischemic heart disease risk factors, 82–0463
reduced mortality among older males vs. decreased prevalence of smoking, 82–0450
reduced serum lipids and cessation of smoking effect, 82–0454
risk among pipe, cigar, and cigarette smokers, 82–0477
risk factor with smoking in deaths in diabetics, 82–0441
risk factors, 82–0455
social class and physical activity as risk factors, 82–0456
spasmatic activity of tobacco smoke components on coronary arteries, 82–0445
sudden cardiac death in women, smoking as factor, 82–0475

COST BENEFITS ANALYSIS
economic arguments against smoking, 82–0585

COTININE
serum levels in pipe, cigar, and cigarette smokers, 82–0477

COUNSELING
behavioral intervention methods, 82–0509
health education, risk intervention trial, 82–0465

CYANIDES
mathematical models for relationship between hydrogen cyanide and butt length, 82–0325
rabbit myocardia exposure effects, 82–0457

DEMOGRAPHIC FACTORS
health and social characteristics, smoking habit relationship, 82–0366
Norway, influences on smoking habit, 82–0507
social class and residence, effects on smoking and alcohol consumption, 82–0516
variables influencing children's initiation of smoking, 82–0542

DENMARK
blood lead levels, smoking habit and alcohol consumption relationship, 82–0338
tobacco sales trends, 1980-1981, 82–0598

DENTAL DISEASES
oral cariogenic microorganism growth, smokeless tobacco effect, 82–0501
plaque accumulation, smoking influence, 82–0495

DIABETES
coronary and hypertension deaths, smoking factors, 82–0441

DIET
cholesterol, lipids, and smoking as risk factors in coronary disease, 82–0447
high saturated fat intake, coronary risk factor, 82–0469
polynuclear aromatic hydrocarbons exposure, 82–0355
vitamin A, in lung cancer risk reduction, 82–0400

DISTRICT OF COLUMBIA
esophageal neoplasm among black males, risk factors, 82–0394

DNA
mitotic index elevation in smoke-exposed hamsters, 82–0344

DRUG ABUSE
geriatric patients, risks in multiple drug usage, 82–0342
high school seniors, 1975-1980, 82–0531
pharmacology and toxicology of nicotine as a drug, 82–0331
televised incidents on top-rated shows, 82–0526

DRUG METABOLISM
clomipramine treatment, smoking effects, 82–0357
geriatric medication treatments, smoking effect, 82–0342
mental patients, drug treatment and cardiac risk factors, 82–0471
niacin/nicotine reactions, 82–0505
oral contraceptive steroid therapy, smoking relationship, 82–0329
phenazone elimination in smokers vs. nonsmokers, 82–0352
sodium thiopental requirements in heavy drinkers and heavy smokers, 82–0492
theophylline clearance in smokers and nonsmokers, 82–0340

DUST
occupational and environmental disease detection, 82–0364

ECONOMICS
- advertising strategies for domestic market, 82–0587
- annual growth in cigarette sales, 82–0588
- arguments against smoking, 82–0585
- chewing, pipe, roll-your-own, and snuff tobacco sales, 82–0595
- Congress, declining influence of farming interests, 82–0602
- disposable income vs. tobacco consumption, worldwide, 82–0591
- domestic cigarette market trends in 1981, 82–0586
- Japan Tobacco and Salt Public Corporation, 82–0599
- Korea, increased production targets for 1981, 82–0596
- Latin America, tobacco industry status reports, 82–0590
- loose-leaf chewing tobacco market trends, 82–0594
- Maxwell international estimates of cigarette market trends, 1978-1980, 82–0589
- Micronesia, tobacco consumption patterns, 82–0538
- sales trends in cigarettes, cigars, cigarillos, 1980-1981, 82–0598
- tobacco industry 1979 data, 82–0593
- tobacco price support program, proposed modifications, 82–0601
- West Germany and East Germany, tobacco trends, 82–0600
- worldwide cigarette and cigar production trends, 82–0592

EMPHYSEMA
- asbestos exposure and smoking effects, 82–0427
- carboxyhemoglobin reduction of exercise tolerance, 82–0408
- human leukocyte elastase inhibition by cigarette smoke, 82–0414
- initiated by cigarette smoke, 82–0436

ENZYME ACTIVITY
- *O*-deethylase, correlation with benzo(*a*)pyrene, 82–0487
- emphysema generation, 82–0436
- human leukocyte elastase inhibition by cigarette smoke, 82–0414
- lecithin-cholesterol acyltransferase, smoking effect, 82–0461
- lifestyle effect on clinical determination, 82–0360
- phenazone metabolism in smokers vs. nonsmokers, 82–0352
- placental monooxygenase sensitivity to maternal smoking, 82–0488

ENZYMES
- elastolytic and antiproteases, 82–0436
- human leukocyte elastase, 82–0414
- lecithin-cholesterol acyltransferase, 82–0461
- monooxygenase, 82–0488

ERYTHROCYTES
- filterability influenced by smoking, 82–0459
- lifestyle effect on clinical determination, 82–0360
- polycythemia, smoking as a possible factor, 82–0444

ESOPHAGEAL NEOPLASMS
- epidemiology risks and contributing factors, 82–0378
- Washington, D.C. black males, risk factors, 82–0394

ETHNIC GROUPS
- health and social characteristics, smoking habit relationship, 82–0366
- Mexican American women, lung cancer mortality and smoking habits, 82–0380
- Old Order Amish, mortality patterns, 82–0365
- Washington, D.C. black males, esophageal neoplasm risk factors, 82–0394

EUROPE
- tobacco sales trends, 1980-1981, 82–0598

EX-SMOKERS
- bladder cancer risk factors, 82–0381
- epithelial recovery, 82–0405
- health consequences of quitting, 82–0557
- lung function improvement following smoking cessation, 82–0424
- percentage increase in Dutch survey, 82–0518
- reduced coronary heart disease mortality among older males, 82–0450
- success factors among women, 82–0566
- women vs. men, reasons, methods, and problems of stopping, 82–0513

FATTY ACIDS
- prostaglandin imbalance relationship to cardiovascular disease, smoking effect, 82–0453

FEDERAL REPUBLIC OF GERMANY
- economic tobacco trends, 82–0598, 82–0600

FERTILITY
- testicular varicocele in fertile and infertile males, smoking relationship, 82–0499

FETAL GROWTH
- adolescent maternal smoking effects, 82–0537
- maternal smoking hazards, 82–0479, 82–0490

FETAL MORTALITY
- benzo(*a*)pyrene effect in mice, 82–0482
- maternal smoking hazards, 82–0479

FETUS
- adrenergic activity, maternal smoking effects, 82–0480
- maternal smoking hazards, 82–0490

FILTERS
adhesive application method for standardizing smoke dilution, 82–0578
aromatic hydrocarbon retention in spiked cigarettes, 82–0298
graduated filters in cessation of smoking, brand assessment, 82–0570
nitrosamine removal by cellulose acetate, 82–0314

FINLAND
coronary disease risk factor clustering, men vs. women, 82–0469
estimated future lung cancer incidence, smoking relationship, 82–0528
North Karelia, hypertension control program, 82–0462
twins, smoking, alcohol, and physical activity relationship, 82–0532

FLUORENES
mutagenicity comparison of methylfluorenes and methylbenzofluorenes, 82–0346

FORMALDEHYDE
indoor air pollution, health hazards, 82–0523

FRANCE
tobacco sales trends, 1980-1981, 82–0598

GAS PHASE, TOBACCO SMOKE
alveolar macrophages' metabolism and phagocytic ability, effect on, 82–0413
rabbit myocardia exposure effects, 82–0457

GASTROINTESTINAL DISEASES
duodenal ulcers, smoking effect on healing, 82–0504

GENETICS, PLANTS
fraction 1 protein in tobacco, 82–0316
nicotine levels in tobacco leaf, 82–0303
tobacco leaf chemistry, 82–0306

GERMAN DEMOCRATIC REPUBLIC
economic tobacco trends, 82–0600

HEAD AND NECK NEOPLASMS
smoking relationship, 82–0384

HEALTH EDUCATION
Australian lifestyle improvement program, 82–0535
environmental cancer information proposal, 82–0383
Finland, hypertension control program, 82–0462
Great Britain, smoking cessation televison series, 82–0554
high blood pressure, trends in public knowledge and behavior, 82–0449
Minnesota curriculum, smoking supplements, 82–0552
risk factors, screening and intervention trial, 82–0465
school-based health promotion programs, 82–0530
schoolchildren, optimum age for antismoking program, 82–0512

HEALTH PRACTICES
environmental cancer education proposal, 82–0383

HEART DISEASES
carbon monoxide effects on ischemic heart, 82–0443
Ireland, trends in cigarette smoking, 82–0452
reduced serum lipids and cessation of smoking effect, 82–0454
screening for risk indices and intervention, 82–0465

HEMATOCRIT
polycythemia, smoking as a possible factor, 82–0444

HEMODYNAMICS
filtration rates in smokers vs. nonsmokers, 82–0459
gingival blood flow, epinephrine and nicotine effects, 82–0497

HEMOGLOBINS
lifestyle effect on clinical determination, 82–0360

HEREDITY
Finland, twins smoking, alcohol, and physical activity relationship, 82–0532
mitotic index elevation in smoke-exposed hamsters, 82–0344

HETEROCYCLIC NITROGEN COMPOUNDS
cigarette flavor, 82–0313

HISTAMINE
increased levels in smokers' sputum, 82–0421

HORMONES
lifestyle effect on clinical determination, 82–0360
nicotine-induced increased levels of serum prolactin, GH, and LH, 82–0326
plasma testosterone levels in middle-aged men, 82–0498

HYPERCHOLESTEREMIA
arteriosclerosis factor, carbon monoxide effect, 82–0440
coronary atherosclerosis patterns, smoking effect comparison, 82–0473

HYPERCHOLESTEROLEMIA
clustering effect with smoking, fatty diet, and hypertension, 82–0469

HYPERLIPIDEMIA
arteriosclerosis factor, carbon monoxide effect, 82–0440

HYPERTENSION
clustering effect in men with smoking, hypercholesterolemia, and fatty diet, 82–0469

Finland, control program, 82–0462
mortality in diabetics, risk factor with smoking, 82–0441
public knowledge and behavior trends, 82–0449
uteroplacental blood flow, acute smoking effects in normotensives vs. hypertensives, 82–0485

IMMUNE SYSTEM
cocoa, coffee, ragweed, and tobacco as cross-reacting allergens, 82–0494
complement protein levels in smokers, 82–0363
higher sputum/serum ratio of IgE in smokers, 82–0421
pulmonary response to tobacco smoke in rats, 82–0339
serum IgE, increased level in smokers, 82–0506
tobacco smoke effects, 82–0409

IMMUNITY
complement protein levels in smokers, 82–0363

IMMUNOGLOBINS
bronchial levels, immunohistochemical assay in rats, 82–0339

INDIA
chewing tobacco, nitrosamine content, 82–0301

INFANT MORTALITY
maternal smoking hazards, 82–0479, 82–0490

INTELLECTUAL DEVELOPMENT
characteristics associated with smoking initiation in children, 82–0542
maternal smoking during pregnancy, children's development effect, 82–0489

IRELAND
coronary patients, trends in cigarette smoking, 82–0452
tobacco sales trends, 1980-1981, 82–0598
zinc concentration in hair and serum of pregnant women in Belfast, 82–0491

ITALY
tobacco sales trends, 1980-1981, 82–0598

JAPAN
tobacco economic data, 82–0598, 82–0599

KOREA
production predictions for 1981, 82–0596

LABELING
Federal Trade Commission recommendations for health warning, 82–0539

LACTATION
nicotine content in mothers who smoke, 82–0308

LARYNGEAL NEOPLASMS
tobacco, alcohol, asbestos, and nickel in etiology of, 82–0372

LARYNX
tobacco smoke-induced lesions in rats and hamsters, 82–0422

LEAD
blood levels in smokers, alcohol consumption effects, 82–0338

LECITHINS
depression of activity in pigeons, cigarette smoke induced, 82–0461

LEGISLATION
Congress, declining influence of farming interests, 82–0602
Federal Trade Commission investigation on cigarette advertising, recommendations, 82–0539
Great Britain cigarette advertising ban unlikely, 82–0603
Great Britain, decreasing government support for tobacco regulation, 82–0604
Great Britain, taxation, advertising bans, pro-health campaigns, effect on cigarette consumption, 82–0597
Medical Association of South Africa proposals, 82–0606
passive smoking, health hazards, and occupational smoking restrictions required, 82–0605
tobacco price support program, proposed modifications, 82–0601

LEUKOCYTES
human elastase inhibition by cigarette smoke, 82–0414

LEUKOPLAKIA
smokeless tobacco as risk factor, 82–0399

LIPOPROTEINS
acute inhalation of cigarette smoke, effects in pigeons, 82–0461
cholesterol levels for ages 6 to 25, smoking and sucrose intake association, 82–0439
cholesterol levels in Australian male service recruits, smoking factor, 82–0521
coronary artery disease in smokers, 82–0476
kinetics of benzo(*a*)pyrene transfer between human lipoproteins, 82–0358
physical activity relationship, 82–0451
weight, height, and ponderal indexes relationship, 82–0446

LIVER
ethanol and/or nicotine effect on chick embryo development, 82–0359

LUNG DISEASES
asbestos and cigarette smoke inhalation effects in hamsters, 82–0432
asbestos exposure and smoking, 82–0374
eosinophilic granuloma, 82–0417
incidence among smokers, women vs. men, 82–0527

LUNG FUNCTION
airways in healthy and asthmatic subjects, 82–0425
asbestos exposure and smoking among construction workers, 82–0419
asbestos exposure and smoking among shipyard workers, 82–0426
carboxyhemoglobin reduction of exercise tolerance in bronchitis and emphysema patients, 82–0408
cessation of smoking effect, 82–0424
Chinese asymptomatic normal smoking adults, 82–0435
cigar vs. cigarette smoke effects in rats, 82–0406
coal miners exposed to diesel emissions, smoking effects, 82–0404
nicotine effects on clearance mechanisms, 82–0315
passive smoking effects, 82–0420
pulmonary disorders in juvenile smokers, 82–0430
Sweden, cessation of smoking by middle-aged men, 82–0557
World Health Organization, respiratory indices in children vs. air pollution, 82–0411

LUNG NEOPLASMS
aryl hydrocarbon hydroxylase inducibility, correlation, 82–0373
asbestos exposure, smokers vs. nonsmokers, 82–0374, 82–0386
association with cigarette tar yield, 82–0382
benzo(*a*)pyrene or cigarette smoke condensate inductions in rat embryo, 82–0351
epidemiology risks and contributing factors, 82–0378
Finland, estimated future incidence vs. smoking, 82–0528
mortality among Mexican American women vs. Anglo women, smoking relationship, 82–0380
nonsmoking wives of smokers, risk, 82–0385, 82–0401
Pennsylvania, incidence vs. smoking habits, 82–0402
physical and psychosocial risk factors, 82–0377
rate increases, smoking-related, 82–0393
relative vs. attributable risk related to smoking, 82–0387
reversal with cessation of smoking, 82–0391
Slovenia, Yugoslavia, smoking as risk factor, 82–0376
toxicity of tobacco smoke, 82–0409
tumorous vs. normal lung tissue metabolism of Benzo(*a*)pyrene, 82–0397
vitamin A intake, reduced risk, 82–0400

LUNGS
baboons, smoke particulates retention, 82–0429
nucleic acid experiments with nicotine and tar, 82–0370

LYMPHOCYTES
aryl hydrocarbon hydroxylase inducibility, lung cancer correlation, 82–0373
metabolism of benzo(*a*)pyrene, 82–0348
smokers vs. nonsmokers, 82–0343

MACROPHAGES
asbestos exposure effects, 82–0432
mutagenicity of BaP-treated macrophage extracts from isolated perfused lungs, 82–0321

MAMMARY NEOPLASMS
operational epidemiology, 82–0378

MARIHUANA SMOKING
alcohol and tobacco usage relationship, 82–0520

MASS MEDIA
alcohol and tobacco consumption on top-rated television programs, 82–0526
British television smoking cessation series, 82–0554, 82–0560
tobacco companies, spending patterns in 1981, 82–0586

MATERNAL-FETAL EXCHANGE
possible cadmium toxicity, 82–0478
uterine blood flow and intrauterine oxygen tension in rats, nicotine effect, 82–0481
uteroplacental blood flow, acute smoking effects in normotensive vs. hypertensive women, 82–0485

MATERNAL SMOKING
adolescents, pregnancy effects, 82–0537
fetal adrenergic activity, effects, 82–0480
fetal and infant health effects, 82–0369
fetal health hazards, 82–0490
mental and physical development of children, 82–0489
mother's milk, nicotine concentrations, 82–0308
perinatal risks, 82–0479
placental monooxygenase activities, fluorometric determination, 82–0487
placental monooxygenase activity sensitivity, 82–0488
possible cadmium toxicity, 82–0478
pregnancy effects with coffee consumption, 82–0486
relative vs. attributable risk for infant growth retardation, 82–0387

MATHEMATICAL MODELS
carboxyhemoglobin ratios, determination in smokers, 82–0337
chemical deliveries of cigarettes in relation to butt length, 82–0325
Finland, estimated future lung cancer incidence, 82–0528

MESOTHELIOMA
- asbestos exposure, 82–0386

METALS, METAL ISOTOPES
- neutron activation analysis of cigarette tobacco, 82–0320
- trace metal toxicity on PAMs from smokers, 82–0390

MICHIGAN
- demographic study of smoking habits and reported illness in Tecumseh, 82–0366

MICRONESIA
- tobacco consumption patterns, 82–0538

MINNESOTA
- seventh grade health curriculum programs, 82–0552

MORBIDITY
- demographic studies, smoking habits relationship, 82–0366
- Finland, estimated future lung cancer incidence, 82–0528
- health risk assessment methods, 82–0367
- National Cancer Institute program to reduce smoking-related disease risk, 82–0563
- occupational and environmental disease detection, 82–0364
- passive smoking as factor, 82–0333
- physical and psychosocial risk factors, 82–0377
- relative vs. attributable risk, 82–0387
- Sweden, cessation of smoking by middle-aged men, 82–0557
- women vs. men, 82–0527

MORTALITY
- cancer, environmental factors, 82–0396
- cancer, 1982 estimates, 82–0369
- cholesterol, lipids, and smoking as factors, 82–0447
- declining cardiovascular disease death rates, 82–0460
- diet and smoking in all-causes death rate, 82–0458
- ethnic factors in the Old Order Amish, 82–0365
- Finland, estimated future lung cancer incidence, 82–0528
- health risk assessment methods, 82–0367
- lung cancer death rate among Mexican American women vs. Anglo women, smoking relationship, 82–0380
- physical and psychosocial risk factors, 82–0377
- reduced coronary heart disease deaths among older males, 82–0450
- relative vs. attributable risk, 82–0387
- sudden cardiac death in women, smoking as factor, 82–0475
- women vs. men, 82–0527

MOTIVATION
- adolescents, factors promoting initiation of smoking, 82–0543
- adolescents, predicting intentions to smoke, 82–0515
- college students, perceived benefits of smoking among smokers and nonsmokers, 82–0545
- Gallup Poll 1981, awareness of health hazards as factor in changes, 82–0524
- hospital role models promoting smoking behavior, 82–0517
- letters of encouragement during smoking cessation followup, 82–0573
- nonsmokers, lifestyle factors in reduced insurance premiums, 82–0549

MOUTH NEOPLASMS
- salivary secretions in healthy vs. cancerous subjects, tobacco/areca chewing relationship, 82–0395
- smokeless tobacco as risk factor, 82–0399
- smoking relationship, 82–0384

MUTAGENESIS
- carcinogenic chemical detection in body fluids, smokers vs. nonsmokers, 82–0334
- nitrosoureas and nitrosocarbamates, metabolic disposition, 82–0332

MUTAGENS
- biological activity in smokers' urine, 82–0353
- methylfluorenes and methylbenzofluorenes, mutagenicity comparison, 82–0346
- nitrosoureas and nitrosocarbamates, metabolic disposition, 82–0332

MYOCARDIAL INFARCT
- chronic ischemic heart disease patients, smoking habits, 82–0474
- oral contraceptive users, 82–0472
- physical and psychosocial risk factors, 82–0377
- secondary prevention, cessation of smoking recommendations, 82–0437
- young patients, smoking as risk factor, 82–0468

NEBRASKA
- adult smoking compared to national figures, 82–0540

NEONATE
- congenital deformities in calves fed *nicotiana glauca*, 82–0483
- maternal smoking hazards, 82–0490

NEOPLASMS
- cancer registers, research value, 82–0375
- environmental factors, 82–0396
- ethnic factors in the Old Order Amish, 82–0365
- incidence among smokers, women vs. men, 82–0527
- lung and spleen nucleic acid experiments with nicotine and tar, 82–0370

167

mutagens in body fluids relationship, 82–0353
smoking as risk factor, 82–0369
tobacco nitrosamines' carcinogenic activity in hamsters, 82–0379

NETHERLANDS
nonsmokers vs. smokers, ratio, 82–0518
physicians vs. general population, smoking survey, 82–0548

NICKEL
laryngeal cancer, causal relationship, 82–0372

NICOTINE
blood coagulation, effect on, 82–0466
coronary heart disease, role in etiology of, 82–0470
dopamine, increased turnover and reduced concentration in rats, 82–0327
dorsal hippocampus and mesencephalic reticular formation as mediators, 82–0350
gingival blood flow reduction, 82–0497
hypothermia in rats, effect on, 82–0493
lung clearance mechanisms effects, 82–0315
mathematical models for relationship between delivery and butt length, 82–0325
mother's milk, concentrations in, 82–0308
nucleic acids of rat lung and spleen, effect on, 82–0370
peripheral nerves, toxic effects, 82–0415
physical dependency relative to smoking habit, 82–0514
recovery and measurement from tobacco, 82–0310
role in the smoking habit, 82–0508
skin temperature changes in smokers vs. nonsmokers, 82–0500
spasmatic activity on coronary arteries, 82–0445
tolerance in rats, 82–0336
toxic effects on chick embryos, 82–0359
uterine blood flow and intrauterine oxygen tension effect on rats, 82–0481

NICOTINE CONTENT
glass capillary gas chromatographic analysis, 82–0322
ozonization of tobacco effect, 82–0583

NICOTINE DERIVATIVES
mass spectra from tobacco alkaloids, metabolites, and related compounds, 82–0311

NICOTINE METABOLISM
excretion changes in smokers under stress, 82–0519
physiology and biosynthesis, 82–0303

NICOTINE PHARMACOLOGY
behavioral and central nervous system effects in humans and animals, 82–0534
dorsal hippocampus and mesencephalic reticular formation as mediators, 82–0350
nerve terminal systems in the rat, effect on, 82–0326
niacin flushing in schizophrenics, 82–0505
nicotine receptors, behavioral, biochemical, and pharmacological studies in the rat, 82–0354
noradrenaline release in presence of tetrodotoxin, 82–0356
norepinephrine levels in nicotine-injected rats, 82–0361
testicular degeneration in rats, 82–0496
toxicological data on nicotine as a drug, 82–0331

NICOTINE REDUCTION
compensatory smoking behavior, 82–0522
puffing behavior, indication of nicotine addiction, 82–0349

NITRATES
denitration of tobacco by ionic extraction, 82–0580
methemoglobin levels in industrial and nonindustrial areas, 82–0362
nitrosamines in tobacco products relationship, 82–0314

NITROGEN CONTENT
Burley tobacco curing method effect, 82–0581
nitrogen analogs of polynuclear aromatic hydrocarbons in cigarette smoke condensate, 82–0323
senescence and curing, chemical transformations, 82–0318
tobacco yield and quality effect, 82–0601

NITROGEN OXIDES
denitration of tobacco by ionic extraction, 82–0580
indoor tobacco smoke exposure, health hazards, 82–0523
methemoglobin levels in industrial and nonindustrial areas, 82–0362
rabbit myocardia exposure effects, 82–0457

NITROSAMINE CONTENT
ambient air and cigarette smoke measurements, 82–0305
Indian chewing tobacco, 82–0301
N-nitrosodiethanolamine in maleic hydrazide-treated tobacco, 82–0302

NITROSAMINES
analysis of tobacco products, 82–0314
carcinogenic activity in hamsters, 82–0379
mass spectra from tobacco alkaloids, metabolites, and related compounds, 82–0311

NONSMOKERS
lifestyle factors in reduced insurance premiums, 82–0549
lymphocyte studies, 82–0343
percentage increase in Dutch survey, 82–0518

NORNICOTINE
biosynthesis, 82–0303

Burley tobacco curing method relationship to nitrosonornicotine level, 82-0581
glass capillary gas chromatographic analysis, 82-0322
Indian chewing tobacco levels, 82-0301
mass spectra for tobacco alkaloid-derived nitrosamines, metabolites, and related compounds, 82-0311
nitrosamines in tobacco products, 82-0314

NORWAY
mortality associated with coronary risk factors, social class, and physical activity, 82-0456
Oslo study of raised lesions in coronary and cerebral arteries, smoking association, 82-0455
smoking survey of schoolchildren ages 12-15, 82-0507

OBESITY
premature ischemic heart disease, risk factor, 82-0463

OBSTRUCTIVE AIRWAY DISEASES
asbestos exposure and smoking effects, 82-0427
genetic factors identified in twins raised apart, 82-0418
peripheral nerve dysfunction, nicotine toxicity, 82-0415

OCCUPATIONAL DISEASES
asbestos-related, 82-0386
detection procedures, 82-0364
respiratory disease risk factors, 82-0410
worldwide literature review of smoking relationship, 82-0388

OCCUPATIONS
asbestos workers, 82-0374, 82-0386
chemical workers, 82-0334
coal miners, 82-0399, 82-0404
coffee roastery workers, 82-0506
construction workers, 82-0419
executive women, 82-0368
firemen, 82-0428
pharmaceutical factory workers, 82-0506
physicians, 82-0548
shipyard workers, 82-0426, 82-0427
soldiers, 82-0529
steel workers, 82-0424
tobacco industry workers, 82-0503

OXYGEN TRANSPORT
uterus, nicotine effects, 82-0481

PANCREATIC NEOPLASMS
coffee drinking and smoking relationship, 82-0371, 82-0389

PARENTAL SMOKING
children's body height effect, 82-0502, 82-0510
influence on smoking initiation by children, 82-0542

PASSIVE SMOKING
baboons, smoke particulates retention, 82-0429
blood coagulation, effect on, 82-0466
cancer risks in nonsmoking wives of smokers, 82-0385, 82-0401
children's body height vs. parental smoking relationship, 82-0502, 82-0510
disease risk factor, 82-0333
health hazards and occupational smoking restrictions required, 82-0605
human placental monooxygenase activity sensitivity, 82-0488
indoor air pollution, health hazards, 82-0523
respiratory function effects, 82-0420

PATENTS
acetyl hydrindacenes and acetyl indanes as flavor and aroma enhancers, 82-0576
denitration of tobacco by ionic extraction, 82-0580
evaporation of volatile compounds from smoking materials, 82-0579
manufacturing method for standardizing smoke dilution of filter cigarettes, 82-0578
reconstituted tobacco with reduced nitrates, 82-0582
tobacco sheet, hardwood pulp reinforcement, 82-0584

PEER GROUPS
children, influence on smoking initiation, 82-0542
hospital role models promoting smoking behavior, 82-0517

PENNSYLVANIA
adolescents, survey of smoking and antismoking messages effects, 82-0562
Allegheny County, lung cancer incidence vs. smoking habits, 82-0402
demographic study of smoking habits and reported illness in Roseto, 82-0366

PEPTIC ULCER
duodenum, smoking effect on healing, 82-0504
incidence among smokers, women vs. men, 82-0527
women, increasing incidence and smoking relationship, 82-0368

PERIODONTAL DISEASES
gingival circulation, epinephrine and nicotine effect, 82-0497

PERIPHERAL NERVE DISEASES
impairment of sensory and motor nerves, nicotine factor, 82-0415

PESTICIDES
insecticide residues in tobacco, 82-0324

PHAGOCYTOSIS
- alveolar macrophages, effect of gas phase of tobacco smoke, 82–0413

PHARYNGEAL NEOPLASMS
- bidi smoking relationship, 82–0384

PHENOL CONTENT
- crop rotation effects, 82–0297
- ozonization of tobacco effect, 82–0583
- thin-layer chromatographic method for identification, 82–0307

PHENOLS
- determination in tobacco smoke and auto exhaust by reversed-phase liquid chromatography, 82–0317
- phenazone metabolism in smokers vs. nonsmokers, 82–0352
- urine excretion level in smokers vs. nonsmokers, 82–0335

PHYSICAL ACTIVITY
- Finland, relationship in twins to smoking and alcohol, 82–0532
- plasma HDL cholesterol relationship, 82–0451
- social class and coronary risk factor associations, 82–0456
- university drug and alcohol prevention program, effectiveness, 82–0555

PIPE SMOKING
- bladder cancer risk factors, 82–0381
- head and neck cancer relationship, 82–0384
- serum cotinine levels, pipe vs. cigar vs. cigarette smokers, 82–0477

PIPE TOBACCO
- increased sales for second quarter 1981, 82–0595

PLACENTA
- *O*-deethylase activity, correlation with benzo(*a*)pyrene, 82–0487
- human monooxygenase activity sensitivity to maternal smoking, 82–0488
- possible cadmium toxicity, 82–0478

PRECANCEROUS CONDITIONS
- induction with benzo(*a*)pyrene or cigarette smoke condensate in rat embryo, 82–0351

PREGNANCY
- benzo(*a*)pyrene correlation with enzyme activity, 82–0487
- coffee consumption and smoking effects, 82–0486
- congenital deformities in calves fed *nicotiana glauca*, 82–0483
- drinking and smoking patterns among California women, 82–0484
- serum and hair zinc levels and congenital malformations, 82–0491
- uteroplacental blood flow, acute smoking effects in normotensives vs. hypertensives, 82–0485

PREMATURITY
- coffee consumption and smoking relationship, 82–0486

PREVENTION OF SMOKING
- advertising as effective intervention tool, 82–0541
- Australia, intervention program for schoolchildren, 82–0556
- complex factors in adolescent smoking habit, 82–0543
- curriculum for grades 4 and 6, short term impact, 82–0564
- Medical Association of South Africa, 82–0606
- physical activity as part of university educational program, 82–0555
- schoolchildren, optimum age for education program, 82–0512
- seventh grade health curriculum programs, 82–0552
- Soviet Union programs, 82–0565
- United Kingdom, decreasing government support, 82–0604
- Yugoslavia, Commune of Tresnjevka, 82–0567

PROTEIN CONTENT
- Burley tobacco curing method effect, 82–0581
- fraction 1 properties, 82–0316
- senescence and curing, chemical transformations, 82–0318

PROTEINS
- complement protein levels in smokers, 82–0363

PSYCHIATRIC DISORDERS
- cardiac risk factors, drug treatment and smoking, 82–0471
- schizophrenics, smoking effect on treatment, 82–0505
- sudden cardiac death in women, smoking and psychiatric history as factors, 82–0475
- withdrawal effects on soldiers in stress situations, 82–0529

PULMONARY ALVEOLAR MACROPHAGES
- activity in smoke-exposed rats, 82–0339
- aryl hydrocarbon hydroxylase inducibility, lung cancer correlation, 82–0373
- metabolism of benzo(*a*)pyrene, 82–0348
- response to gas phase of tobacco smoke, 82–0413
- trace metal toxicity, smoking effects, 82–0390

PULMONARY ALVEOLI
- emphysema generation activity with cigarette smoke, 82–0436

PULMONARY CLEARANCE
- sulfuric acid inhalation tests, 82–0423

PYRIDINES
- aza-arenes in cigarette smoke condensate, identification, 82–0323
- cigarette flavor role, 82–0313

RADIATION
- indoor tobacco smoke exposure, health hazards, 82–0523
- occupational and environmental disease detection, 82–0364

RECIDIVISM
- buddy system prevention strategy, 82–0561
- nicotine dependence as factor, 82–0533
- prevention through use of letters of encouragement, 82–0573

REDUCTION OF SMOKING
- aversive therapy, two-stage vs. three-stage models, 82–0569
- decreased coronary heart disease incidence, 82–0454
- graduated filters use, 82–0570
- mass media campaigns, effects, 82–0551
- MRFIT intervention trial for coronary disease risk reduction, 82–0553
- nicotine fading programs, 82–0561
- snuff use by heavy smokers, 82–0550

RESPIRATORY FUNCTION TESTS
- air pollution and smoking effects, 82–0412
- airways in healthy and asthmatic subjects, 82–0425
- asbestos exposure and smoking among construction workers, 82–0419
- asbestos exposure and smoking among shipyard workers, 82–0426
- asymptomatic normal smoking Chinese adults, 82–0435
- carboxyhemoglobin effects, reduction of exercise tolerance in bronchitis and emphysema patients, 82–0408
- coal miners exposed to diesel emissions, smoking effects, 82–0404
- comparison in smokers, ex-smokers, and nonsmokers, 82–0424
- diffusion capacity abnormalities in asbestos-exposed smokers, 82–0427
- intra-individual variability of indices determination in firemen, 82–0428
- lower mucus transport rate in smokers, 82–0431
- maximal expiratory flow-volume curves for juvenile smokers, 82–0430
- passive smoking effects, 82–0420
- sulfuric acid inhalation exposure vs. mucociliary clearance, 82–0423
- Sweden, cessation of smoking by middle-aged men, 82–0557
- twins raised apart, 82–0418
- World Health Organization, clinical indices in children vs. air pollution, 82–0411

RESPIRATORY MUCOSA
- lower mucus transport rate in smokers, 82–0431
- smoke exposure effects in the slug, 82–0434
- sulfuric acid inhalation tests, 82–0423

RESPIRATORY SYMPTOMS
- coal miners exposed to diesel emissions, smoking effects, 82–0404

RESPIRATORY SYSTEM
- cilia ultrastructural abnormalities in smokers vs. nonsmokers, 82–0416
- eosinophilic granuloma in the lung, 82–0417
- mitotic activity in airway epithelium in rats, tobacco smoke effect, 82–0407
- sulfuric acid inhalation exposure vs. mucociliary clearance, 82–0423

RESPIRATORY TRACT DISEASES
- air pollution and smoking effects, 82–0412
- asbestos exposure and smoking, 82–0374
- eosinophilic granuloma in the lung, 82–0417
- ethnic factors in the Old Order Amish, 82–0365
- occupational exposure and smoking as factors, 82–0410
- sex differences, chronic bronchitis in South African smokers, 82–0433
- tobacco smoke-induced laryngeal lesions in rats and hamsters, 82–0422
- toxicity of tobacco smoke, 82–0409

RESPIRATORY TRACT NEOPLASMS
- bronchioloalveolar vs. adenocarcinoma, 82–0398

REVERSE SMOKING
- cancer of the palate relationship, 82–0384

SALIVA
- secretions and flow rates in healthy vs. cancerous subjects, tobacco/areca chewing relationship, 82–0395

SELENIUM
- toxicity on PAMs, benzo(*a*)pyrene metabolite effects, 82–0390

SEX RATIO
- cessation of smoking, reasons, methods, and problems, 82–0513
- chronic bronchitis in South African smokers, 82–0433
- lung neoplasm incidence, smoking as factor, 82–0376
- smoking habits and related problems, women vs. men, 82–0527
- Sweden, bronchial neoplasms and smoking, 82–0392

SKIN
- temperature changes in smokers vs. nonsmokers, 82–0500

SKIN DISEASES
- erythema, urticaria, and mild eczema in tobacco workers, 82–0503

SMALL AIRWAYS DISEASE
- prevalence among juvenile smokers, 82–0430

SMOKE INHALATION, ANIMAL
- cigarette vs. cigar smoke, health hazards in rats, 82–0406
- laryngeal lesions in rats and hamsters, 82–0422

SMOKE STREAMS
- analysis of parsley, marihuana, and tobacco cigarettes, 82–0310
- blood coagulation, exposure effects, 82–0466
- cigarette product formation mechanisms, 82–0299
- indoor air pollution, health hazards, 82–0523
- pulmonary metabolism effects in rabbit lungs of benzo(*a*)pyrene, 82–0347

SMOKING
- cancer registers and smoking data, research value, 82–0375

SMOKING ARTICLES
- evaporation of volatile compounds from smoking materials, 82–0579

SMOKING CHARACTERISTICS
- inhalation relationship to bladder cancer, 82–0403
- nicotine compensation behavior of dependent smokers, 82–0522
- puffing behavior as indication of nicotine addiction, 82–0349
- skin temperature changes, relationship, 82–0500
- stress relationship to puff rate and amount smoked, 82–0519
- tar/nicotine ratio relationship, 82–0547
- time of daily smoking onset as indication of nicotine dependence, 82–0533
- topography and frequency measures of smoking behavior, 82–0536

SMOKING CONTROL PROGRAMS
- Australia, intervention program for schoolchildren, 82–0556
- Australia, lifestyle improvement program, 82–0535
- Austrian national antismoking campaign, 82–0559
- Medical Association of South Africa proposals, 82–0606
- National Cancer Institute program for disease risk reduction in smokers, 82–0563
- seventh grade health curriculum programs, 82–0552
- Soviet Union, 82–0565
- United Kingdom, decreasing government support, 82–0604
- West German clinic, patients' survey, 82–0558
- Yugoslavia, Commune of Tresnjevka, 82–0567

SMOKING HABIT
- adolescent females, 82–0537
- adolescents, antismoking messages effect, 82–0562
- advertising relationship to initiation, maintenance, and cessation, 82–0511
- Australian children, social and personal factors, 82–0541
- blood chemical analysis, effect on, 82–0360
- British middle-aged men, 82–0516
- British youth, smoking prevalence and expenditures, 82–0544
- illnesses among populations with different systems of social support, relationship, 82–0366
- initiation among schoolchildren, related factors, 82–0542
- lung cancer incidence in white males, relationship, 82–0402
- Micronesians, 82–0538
- Nebraska adults vs. national average, 82–0540
- nicotine effects on human and animal smoking behavior, 82–0534
- nicotine's role, 82–0508
- Norwegian boys vs. girls ages 12-15, 82–0507
- zinc concentration in hair and serum of pregnant women, relationship, 82–0491

SMOKING SURVEYS
- adolescents, effectiveness of smoking and antismoking messages, 82–0562
- alcohol, tobacco, and marihuana usage in a general population, 82–0520
- Australian children, social and personal factors, 82–0541
- Austrian, for coronary risk factors, 82–0467
- Bellville, South Africa, 82–0433
- British, middle-aged men, 82–0516
- chronic ischemic heart disease patients, 82–0474
- college students, attitudes of smokers vs. nonsmokers, 82–0546
- college students, perceived benefits of smoking, 82–0545
- Dutch, increase in nonsmokers, 82–0518
- Dutch males, physicians vs. general population, 82–0548
- Gallup Poll 1981, changing U.S. smoking patterns, 82–0524
- high school seniors, 1975-1980, 82–0531
- Nebraska adults vs. nationwide average, 82–0540
- Norwegian children ages 12-15, 82–0507
- Ottawa, Canada, women, 82–0566

pregnant California women, drinking and smoking patterns, 82–0484
Quebec, Canada, schoolchildren, 82–0512

SNUFF
domestic market trends, 82–0594
increased sales for second quarter 1981, 82–0595
nasal cancer relationship, 82–0384
oral cariogenic microorganism growth, effect on, 82–0501

SNUFF-DIPPING
cigarette substitute for heavy smokers, 82–0550
oral cancer risks, 82–0399

SOCIOECONOMIC STATUS
class and town of residence, effect on alcohol consumption and smoking, 82–0516
physical activity and coronary risk factor associations, 82–0456
premature ischemic heart disease, risk factor, 82–0463

SOUTH AFRICA
Bellville population smoking habits, 82–0433
Medical Association antismoking proposals, 82–0606

SOUTH AMERICA
tobacco industry economics by country, 82–0590

SPERMATOZOA
nicotine exposure in rats, 82–0496
testicular varicocele in fertile and infertile males, smoking relationship, 82–0499

SPLEEN
nucleic acid experiments with nicotine and tar, 82–0370

STATISTICAL ANALYSIS
cancer, 1982 estimates of prevalence, 82–0369
cross-sectional survey for changes in lung function after smoking cessation, 82–0424
Finland, estimated future lung cancer incidence vs. smoking incidence, 82–0528
HDL-cholesterol relationship to strenuous physical exercise, 82–0451
health risk assessment methods, 82–0367
high-density lipoprotein cholesterol and triglycerides, body mass relationship, 82–0446
histology comparison with pack-years of cigarette consumption, 82–0405
lung cancer incidence in white males, Pennsylvania, 82–0402
multivariate prediction of smoking in children, 82–0542
twins raised apart, smoking effect on respiratory function tests, 82–0418
women, increase in smoking-associated diseases, 82–0368

STEROLS
ozonization of tobacco effect, 82–0583

STRESS
consumption increase among anxious smokers, 82–0519
military, nicotine withdrawal effects on soldiers, 82–0529
psychosocial risk factors, 82–0377
reduction, effectiveness of physical activity in smoking prevention programs, 82–0555

STUDENTS
attitudes on tobacco smoking, smokers vs. nonsmokers, 82–0546
characteristics associated with smoking initiation, 82–0542
cigarette and drug use among high school seniors, 1975-1980, 82–0531
health education and the smoking habit, 82–0530
perceived benefits of smoking, 82–0545
physical activity in prevention of smoking program, 82–0555
social and personal factors in smoking habit in Australian primary schools, 82–0541

SULFUR COMPOUNDS
cigarette flavor role, 82–0313

SWEDEN
asbestos exposure and smoking among construction workers, 82–0419
bronchial carcinoma and smoking habits in Uppsala, 82–0392
cessation of smoking by middle-aged men, health consequences, 82–0557

SWITZERLAND
women vs. men ex-smokers, reasons, methods, and problems of stopping, 82–0513

TAIWAN
maximal expiratory flow and volume in asymptomatic normal smoking adults, 82–0435

TAR CONTENT
Burley tobacco curing method effect, 82–0581

TARS, CIGARETTE
baboons, smoke particulates retention, 82–0429
mathematical models for relationship between delivery and butt length, 82–0325
ozonization of tobacco effect, 82–0583

TARS, TOBACCO
nucleic acids of rat lung and spleen, effect on, 82–0370

TAXATION
British cigarettes, consumption effects, 82–0597

TEETH
plaque accumulation, smoking influence, 82–0495

TERATOGENESIS
benzo(*a*)pyrene effect in fetal mice, 82–0482

TERPENES
biosynthesis, 82–0304

TESTES
nicotine exposure, degeneration in rats, 82–0496
plasma testosterone levels in middle-aged men, 82–0498
varicocele in fertile and infertile males, smoking relationship, 82–0499

TEXAS
lung cancer mortality, Mexican vs. Anglo women, 82–0380

THIOCYANATES
blood serum concentration in smokers, 82–0337

THROMBOSIS
reduced risk in smokers, 82–0442

TOBACCO
allergic reactions in industry workers, 82–0503

TOBACCO ADDITIVES
acetyl hydrindacenes and acetyl indanes as flavor and aroma enhancers, 82–0576
additional risk factors for carcinogenesis, 82–0369
flavor systems for less hazardous cigarettes, 82–0574
hardwood pulp reinforcement of tobacco sheet, 82–0584

TOBACCO AGING
chemical changes, 82–0306

TOBACCO AROMA
acetyl hydrindacenes and acetyl indanes, 82–0576

TOBACCO CHEWING
oral cancer risks, 82–0399
salivary secretions and flow rates in healthy vs. oropharyngeal cancer subjects, 82–0395

TOBACCO CONTAMINANTS
insecticide residues, 82–0324
maleic hydrazide, N-nitrosodiethanolamine content, 82–0302

TOBACCO CURING
Burley constituent modification, 82–0581
chemical changes, 82–0306, 82–0318
fermented cigar smoke, reduced health hazards in rats, 82–0406

TOBACCO FLAVOR
acetyl hydrindacenes and acetyl indanes, 82–0576
chemical additive systems for less hazardous cigarettes, 82–0574
chemical and physical properties, 82–0324
leaf and smoke constituents relationship, 82–0306
nonvolatile leaf precursors in smoke, 82–0313
product safety concerns, 82–0304
senescence and curing, chemical transformations, 82–0318

TOBACCO INDUSTRY
advertising strategies for domestic market, 82–0587
annual growth in cigarette sales, 82–0588
chewing, pipe, roll-your-own, and snuff tobacco sales, 82–0595
domestic cigarette economic trends in 1981, 82–0586
domestic economic 1979 data, 82–0593
Japan, economic data, 82–0599
Korea, increased economic targets for 1981, 82–0596
Latin America, status reports, 82–0590
loose-leaf chewing tobacco market trends, 82–0594
Maxwell international estimates of cigarette market trends, 1978-1980, 82–0589
Micronesia, tobacco expenditures, taxes, and consumption patterns, 82–0538
price support program, proposed modifications, 82–0601
sales trends, 1980-1981, 82–0598
West Germany and East Germany, economic trends, 82–0600
worldwide cigarette and cigar production trends, 82–0592

TOBACCO LEAF COMPONENTS
alkaloid physiology and biosynthesis, 82–0303
Burley constituent modification by curing, 82–0581
chemical and physical criteria for modern products, 82–0306
chemical and physical properties, 82–0324
crop rotation effects on polyphenols, 82–0297
fraction 1 protein, 82–0316
glass capillary gas chromatographic analysis, 82–0322
phenol content determination method, 82–0307
pyrolysis of nonvolatile precursors, cigarette flavor relationship, 82–0313
senescence and curing, chemical transformations, 82–0318

TOBACCO PROCESSING
sheet reinforcement with hardwood pulp, 82–0584

TOBACCO PYROLYSIS PRODUCTS
char oxidation analysis, 82–0319
leaf precursors relationship, 82–0306
nicotine recovery and measurement, 82–0310

TOBACCO, RECONSTITUTED
- denitration with addition of carbohydrate and/or protein, 82–0582
- hardwood pulp for reinforcement, 82–0584

TOBACCO SMOKE
- Burley constituent modification by curing, 82–0581
- determination of phenols, 82–0317
- indoor air pollution, health hazards, 82–0523
- pulmonary immune system response in rats, 82–0339
- pyrolysis of nonvolatile leaf precursors, flavor relationship, 82–0313
- toxicity in lung diseases, 82–0409

TOBACCO SUBSTITUTES
- analysis of parsley and marihuana cigarettes, 82–0310
- nicotine chewing gum, 82–0568

TRACHEOBRONCHIAL EPITHELIUM
- reduced tracheal mucus transport, 82–0431

TRIGLYCERIDES
- carbon monoxide exposure effects, 82–0440
- weight, height, and ponderal indexes relationship, 82–0446

TWINS
- Finland, smoking, alcohol, and physical activity relationship, 82–0532
- respiratory function tests in smokers and nonsmokers, 82–0418

UNION OF SOVIET SOCIALIST REPUBLICS
- atherosclerosis precursors in schoolchildren in Kaunas, 82–0464
- government responses to smoking hazards, 82–0565
- tobacco sales trends, 1980-1981, 82–0598

UNITED KINGDOM
- child growth vs. parental smoking, 82–0502
- cigarette yield decrease, 82–0309
- decreasing government support for tobacco regulation, 82–0604
- legislation to ban cigarette advertising unlikely, 82–0603
- social class and town of residence, effect on alcohol consumption and smoking, 82–0516
- taxation effects on smoking in Britain, 82–0597
- television smoking cessation series, 82–0554
- youth smoking prevalence and expenditures, 82–0544

URANIUM
- urinary concentrations in smokers vs. nonsmokers, 82–0330

URIC ACID
- theophylline metabolites in smokers vs. nonsmokers, 82–0340

URINE
- mutagenic activity in smokers, 82–0353
- mutagenic chemical studies in smokers vs. nonsmokers, 82–0334
- nicotine excretion changes in smokers under stress, 82–0519
- theophylline clearance and metabolism in smokers vs. nonsmokers, 82–0340
- uranium levels in smokers vs. nonsmokers, 82–0330

UTERUS
- blood flow reduction following nicotine injection in rats, 82–0481

VASCULAR DISEASES
- oral contraceptives and smoking risk factor, 82–0329

VITAMIN A
- lung cancer risk reduction, 82–0400

VOLATILE COMPOUNDS
- inhaling device for evaporation from smoking materials, 82–0579

WEST INDIES
- tobacco industry economics by country, 82–0590

WITHDRAWAL CLINICS
- acupuncture treatment, 82–0572

WITHDRAWAL SYMPTOMS
- nicotine chewing gum use, 82–0568
- nicotine tolerance in rats, 82–0336
- soldiers, performance effects in stress situations, 82–0529

WOMEN
- adolescent females smoking habits, 82–0537
- cessation of smoking, reasons, methods, and problems, 82–0513
- increase in smoking-associated diseases, 82–0368
- maternal smoking hazards, 82–0490
- Mexican Americans, lower lung cancer mortality than Anglos, 82–0380
- myocardial infarction among oral contraceptive users, 82–0472
- Ottawa, Canada, smoking initiation and cessation survey, 82–0566
- perinatal risks from maternal smoking, 82–0479
- reduction in smoking following screening and intervention, 82–0465
- smoking habits and related problems, 82–0527
- sudden cardiac death, smoking as factor, 82–0475

YUGOSLAVIA
- antismoking programs, 82–0567
- lung cancer and cardiac infarct, physical risk factors, 82–0377

lung neoplasm incidence in Slovenia, smoking relationship, 82–0376

176

SMOKING AND HEALTH LITERATURE SEARCH REQUEST

Individual requesting search

Title or position

Individual other than requester completing form

Telephone number

Organization

Date of request

Address including zip code

Purpose of search

☐ Research

☐ Paper presentation

☐ Writing article ☐ Lecture

☐ Other ____________________

SEARCH REQUIREMENTS Give below a detailed statement of information requirements together with any necessary background data to meet your needs. Be as specific as possible to help formulate search strategy.

If topic has been reviewed or cited in past issues of the *Smoking and Health Bulletin* or *Bibliography on Smoking and Health* give pertinent accession number(s) or citation of articles.

Give below other articles which may serve as a guide on subject area (list full bibliographic citation if known)

List relevant key words or index terms appropriate to search.

If a search has been performed on subject utilizing other data sources, please list sources, results of search and search strategy if known.

SEARCH RESTRICTIONS Check appropriate search out-put desired.

☐ Full range (no restrictions)

☐ Year(s)__________only

☐ Citation only (no abstract)

☐ English language only

☐ Other language __________

AGENCY USE ONLY. Do Not Write Below This Line.

Search statement or criteria	Date search received Date search released
	Number of documents retrieved

☆U.S. GOVERNMENT PRINTING OFFICE: 1982-361-166:313

NOTICE

If you no longer need this publication, check here____, return this sheet to the address below, and your name will be dropped from the mailing list.

If you have a change in name or address, please write the change on this sheet and return it to the address below.

____

____

____

WHEN REQUESTING A CHANGE PLEASE BE SURE YOUR FORMER LISTING ALSO APPEARS ON THIS SHEET.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health
Rockville, Maryland 20857

U.S. MAIL

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS

BOOK

982/3

NEWARK PUBLIC LIBRARY

JUL 14 1982

U. S. GOVT. PUBS.
DEPOSITORY 376

MAY-JUNE 1982

Abstracts 82-0607-82-0915

Smoking and Health Bulletin

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

HE 20.7211:

INTRODUCTION

All items appearing in the Smoking and Health Bulletin become part of the permanent document collection of the Technical Information Center (TIC) of the Office on Smoking and Health and can be referred to by their accession number.

In the past, at the end of each calender year, it has been customary to publish the Bibliography on Smoking and Health which contained a cumulative author and subject index based on the individual Bulletins issued that year. However, this year, due to fiscal and other constraints, it may not be possible to issue the index in the form of the Bibliography . All readers are therefore advised to keep the individual Bulletin issues for both 1981 and 1982 until further notice.

The TIC offers bibliographic search and retrieval services for information on smoking, tobacco, and tobacco use through its Automated Search and Retrieval System. Readers are encouraged to submit searches on the search request form found at the end of each Bulletin issue. Multiple copies of this form can be ordered from the TIC. Currently the automated file contains approximately 25,000 records, the majority of which were published and processed into the TIC collection since 1970. The TIC collection totals more than 35,000 items.

Bibliographic and reference services continue to be offered. Readers can take advantage of these services by writing or calling the TIC at the number listed below. Staff limitations currently exists for photocopying services, therefore your cooperation is sought when requesting such services. Until further notice no more than three items per Bulletin per request can be provided. Requests for services above this three document limit cannot be honored. In order that we may continue to provide photocopying services to the largest number of readers, we ask before requesting such items from the TIC collection, to first check your local resources regarding their availability. Visitors may use the TIC library during regular officer hours, 8:30 a.m. to 5:00 p.m. weekdays. Advance arrrangements of any planned visits are suggested.

We welcome suggestions or comments from our readership regarding our publications and services.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Telephone: 301-443-1690

ISSN: 0081-0363
MAY-JUNE 1982
Abstracts 82-0607-82-0915

Smoking and Health Bulletin

CONTENTS

AGRICULTURE 149
CHEMISTRY 149
PHARMACOLOGY AND TOXICOLOGY 151
MORTALITY AND MORBIDITY 158
NEOPLASTIC DISEASES 160
NON-NEOPLASTIC RESPIRATORY DISEASES 168
CARDIOVASCULAR DISEASES 174
PREGNANCY AND INFANT HEALTH 183
OTHER DISEASES AND CONDITIONS 185
BEHAVIORAL AND PSYCHOLOGICAL ASPECTS 188
SMOKING PREVENTION AND INTERVENTION 197
SMOKING CESSATION METHODS 205
TOBACCO PRODUCT ADDITIVES 208
TOBACCO MANUFACTURING AND PROCESSING 211
TOBACCO ECONOMICS 212
LEGISLATION 215
GENERAL 216
AUTHOR AND ORGANIZATIONAL INDEX i
SUBJECT INDEX vii

AGRICULTURE

82-0607. Podeur, G.; Cazamajour, F.; Chouteau, J. **Influence d'un Ecimage Precoce sur les Caracteristiques Physiques et Chimiques des Tabacs Noirs. [Influence of Early Topping on Physical and Chemical Characteristics of Dark Tobacco.]** *Annales du Tabac* 16(2): 61-66, 1979-1980, French.

Characteristics of tobacco leaves of PBD6 variety from early topping, i.e., 55 to 60 days after planting, were compared with those of the leaves from normal topping, i.e., 15 days later. Over a period of 3 years (1977 to 1979), early topping produced a 20 percent higher average yield of dry tobacco than normal topping. Physical and chemical characteristics of tobacco were determined after 8 days of treatment at 55°C. Early topping was found to cause an average decrease of 12 percent in filling power (volume of 10 gr of shredded tobacco) and a slight lowering of combustion time. Total alkaloids in the dry tobacco increased an average of 30 percent due to early topping. Smoke condensate was analyzed from cigarettes smoked on a model 302 Filtrona machine according to CORESTA standards. Nicotine in condensate from cigarettes made of low, median, and high leaves of early topping and tar in condensate from cigarettes made of low leaves only increased significantly in comparison with products of normal topping. The increases of the nicotine/tar ratio in the smoke condensate by 1.6, 1.6, and 2.2 percent, respectively, as a consequence of early topping, were due mainly to corresponding increases in nicotine content of the smoke condensate.

82-0608. Steffens, G. L. **Growth Regulator Combinations for Tobacco Sucker Control: Maleic Hydrazide and a *N*-Benzyldinitroaniline (CGA-41065).** *Tobacco International* 182(17): 47-49, August 22, 1980.

To reduce potentially undesirable residues of maleic hydrazide (MH) on cured tobacco leaf, combinations of MH and *N*-ethyl-*N*-(2-chloro-6-fluorobenzyl)-2',6'-dinitro -4'-trifluoromethylaniline (CGA-41065) were evaluated for effectiveness of control of axillary bud (sucker) growth on tobacco. Results from both greenhouse (cv. Connecticut Broadleaf) and field (cv. Hicks, a flue-cured type) show that combining the two inhibitors provides a broader range of sucker control than either one alone. In both types of experiments the addition of CGA-41065 to MH sprays resulted in a decreased slope of the curves for amount of chemical applied vs. percentage sucker control. An effective proportion of CGA-41065 to MH for greenhouse plants was about 1 part CGA-41065 to 2 parts MH, but for field plants, it was nearly 1 part CGA-41065 to 4 parts MH. (Auth. Abs. Mod.)

82-0609. University of Kentucky. **Tobacco Handbook.** University of Kentucky, College of Agriculture, Cooperative Extension Service, Lexington, ID-45, February 1981, 64 pp.

Advice and information on tobacco agriculture, from planting to curing, is provided and the economic situation for Burley and dark tobacco is analyzed. In a discussion of topping and sucker control, the problem related to West German objections to the levels of maleic hydrazide (MH) residues on mainly flue-cured U.S. tobacco is clarified and practices are suggested to help in maintaining MH residues at an acceptable level. Tables are provided with insecticide recommendations for tobacco beds and fields, including precautions, instructions, and suggestions. Information is also presented on selecting tobacco varieties, producing tobacco transplants, fertilization, pesticides, weed control, diseases, selecting an air-cured tobacco housing and curing facility, renovating and remodeling conventional barns, rail wagons, and tobacco barn plans.

CHEMISTRY

82-0610. Arany-Fuzessery, K. **Combustibility of Tobacco as a Function of Its Chemical Composition and Physical Characteristics.** *Acta Alimentaria* 10(3): 167-174, 1981.

A correlation was established between the pyrolysis temperature of cigarette glow and the amount of carcinogenic substances formed. The burning capacity of tobaccos of different varieties and cultivated in different areas of Hungary were studied as a function of their chemical composition. Positive and negative correlations between the chemical components of tobacco and its burning period were determined by linear regression and correlation calculation and are shown in tabular form. Particular attention was paid to the relationship between the L value (distance between the minimum and maximum of the exothermic section of the DTA curve plotted by the Derivatograph) and the burning period. It was found that the correlation coefficient was positive and of a value higher than 0.05 in the case of Ca, K, cellulose, alkalinity of ash, and (K + Ca)-Cl. It was negative for chloride, starch, pectin, water-soluble and total volatile organic acids, and sugars. It appears that the combustibility of tobacco is in close correlation with its chemical composition. The correlation, however, is affected by many other factors as well.

82–0611. Gudzinowicz, B. J.; Gudzinowicz, M. J. **Analysis of Drugs and Metabolites by Gas Chromatography—Mass Spectrometry. Volume 7. Natural, Pyrolytic, and Metabolic Products of Tobacco and Marijuana.** New York, Marcel Dekker, Inc., 1980, 567 pp.

A complete analysis of the natural, pyrolytic, carcinogenic, and metabolic products of tobacco and marihuana via gas chromatography (GC) and integrated gas chromatography-mass spectrometry (GC-MS) is presented. The chemical composition of tobacco leaf is discussed in terms of (1) nicotine and other alkaloids, (2) N'-nitrosonornicotine, (3) hydrocarbons, (4) alcohols of tobacco, (5) sterols, (6) fatty acids and esters, and (7) ammonia and hydrazine. A discussion of the chemical composition of tobacco smoke includes (1) tobacco alkaloids in smoke and biological fluids, (2) low-boiling volatiles (general), (3) hydrocarbons, (4) keytones, (5) phenolic constituents, (6) acidic components, (7) polynuclear aromatic hydrocarbons and related compounds, (8) sulfur-containing compounds, and (9) analysis of specific miscellaneous compounds. The GC and/or GC-MS conditions required to separate and identify carcinogens in tobacco smoke are given, and 153 polynuclear aromatic hydrocarbons that have been identified in tobacco and marihuana smoke condensate are tabulated, and their abundance in μg/100 cigarettes is featured. A total of 452 references, 136 figures, and 143 tables accompany the text, such as: chemical composition of tobacco leaf; relative retention times of nicotine bases and high boiling pyridine bases; pyrolysis products of nicotine; operating conditions of packed and capillary columns; chemical composition of tobacco smoke (515 compounds listed); phenols in condensed smoke from filter-removed cigarettes; chatechol in the mainstream of tobacco smoke; and mass spectra and relative abundance of fluorenes. The natural and pyrolytic products of marihuana are also explored, including the pharmacology, absorption, metabolism, and excretion of delta-9-tetrahydrocannabinol (Δ^9-THC), the major active ingredient of marihuana. The specific topics are: (1) cannabinoid patterns and their use in determining chemical race and origin, (2) chemical disposition of the marihuana plant, (3) chemical constituents of marihuana smoke, and (4) cannabinoids and metabolic products in biological media.

82–0612. Katz, T.; Pitner, T. P.; Kinser, R. D.; Ferguson, R. N.; Einolf, W. N. **Isolation and Identification of Two New Seco-Cembranoids From Cigarette Smoke.** *Tetrahedron Letters* 22(48): 4771-4774, 1981.

The isolation and identification from cigarette smoke of two new C_{20} seco-cembranoids, 5-isopropyl-8,12-dimethyl-3E,8E,12E,14-pentadecatetraen-2-one, and its 12Z isomer, are reported. The compounds were separated from the cigarette smoke of duvane-producing varieties of *Nicotiana tabacum*. The isolation scheme and spectral data are discussed. The double bond positions were determined by ozonolysis and the stereochemistry confirmed by ^{13}C nuclear magnetic resonance (NMR).

82–0613. Kim, J. O.; Park, K. H.; Park, E. S. **Iptambae Chosonggwa Yon'gisongbun'gwaui Kwan'gye. [Correlation Between Tobacco Leaf and Smoke Compositions.]** *Journal of the Korean Society of Tobacco Science* 1(2): 93-102, November 1979, Korean.

Flue-cured and Burley tobacco produced in Korea in 1978 was analyzed for correlations between tobacco leaf and smoke compositions. The tests were done mainly to find out the correlation between the dry condensate and nicotine contents in smoke and its chemical compositions. The dry condensate and nicotine contents vary as to tobacco varieties, production sites, and methods of drying and aging of tobacco leaf. As a result of the analysis of tobacco smoke, the following 10 composition elements were extracted under various test conditions: hexane extract, alcohol-benzene extract, acetone extract, hot water extract residue, ether extract, total alkaloid, total nitrogen, crude ash, crude fiber, and total sugar. The tests showed that those compositions of flue-cured tobacco smoke which related closely with dry condensate were total alkaloid, hexane extract, and crude ash. The total alkaloid and hexane extract were in positive relation at the 0.1-1 percent level, and crude ash in negative relation at the 1 percent level. The main compositions of Burley tobacco were alcohol-benzene extract, hot water extract, hot water extract residue, and crude ash. The alcohol-benzene extract, hot water extract, and hot water extract residue were in positive relation at the 1 percent level, and crude ash in negative relation at the 1-5 percent level. Shiro's test showed that total alkaloid and crude ash were related with dry condensate for both flue-cured and Burley tobaccos. This test showed that those two elements were related only for flue-cured tobacco.

82–0614. Lee, J. K. **Tambaeyon'gi Songbunjung Nait'rosaemin Kyeyol Hwahammulgwa Karsinojenesis. [Nitrosamine Compounds in Cigarette Smoke and Carcinogenesis.]** *Journal of the Korean Society of Tobacco Science* 1(2): 152-158, November 1979, Korean.

Nitrosamine compounds, how they are formed and how they cause cancer, are discussed with special emphasis given to mutagenesis. NNN (N-nitrosonornicotine) represents the nitrosamine compounds in tobacco leaves and smoke. In fresh tobacco leaves, before the curing process, NNN is not present. However, after curing, there are great amounts of NNN. How NNN is formed during the curing process is unknown, but it is now known that nicotine and/or nornicotine in contact with nitrite act as main agent(s) for nitrosation. Nitrosamine compounds are recognized as carcinogenic and mutation-causing elements. NNN causes lung adenomas in mice, and NAB causes esophageal tumors in mice. However, the biochemical basis for carcinogenesis remains to be clarified. Most of the carcinogenic compounds are converted during metabolism to activated particles which can interact and transform cells by chemical reaction with DNA, RNA, or protein. In case of cyclic or acyclic N-compounds, the α-hydroxylation is an important activation stage causing cancers. More research is needed to clarify the interaction between the carcinogenic elements in tobacco and cellular components such as DNA, RNA, and protein.

82–0615. Matsushima, S.; Ishiguro, S.; Sugawara, S. **Hatabako Seiyu no Seibunsosei to Koki Toso Kanren. [Relationship Between Composition of Tobacco Essential Oil and Its Aroma.]** *Journal of the Agricultural Chemical Society of Japan* 54(12): 1027-1035, 1980, Japanese.

The main components of essential oil from flue-cured, Turkish, and Burley tobaccos were identified and quantitatively determined by gas chromatography and gas chromatography-mass spectrometry. The aromas of mixtures of the main compounds were evaluated by sensory tests. The aroma of essential oil from flue-cured tobacco seemed to be formed mainly by a combination of linolenic acid, linoleic acid, solanone, megastigmatrienone, and furfuryl alcohol, while that from Turkish tobacco appeared to be formed by β-methylvaleric acid, linolenic acid, and linoleic acid. Compounds contributing to the aroma of essential oil from Burley tobacco could not be determined. Gas chromatograms and tables of essential oil components of tobacco are also presented. (Auth. Abs. Mod.)

See also, **82–0637**

82–0616. Patrianakos, C.; Hoffmann, D. **Chemical Studies on Tobacco Smoke LXIV. On the Analysis of Aromatic Amines in Cigarette Smoke.** *Journal of Analytical Toxicology* 3: 150-154, July-August 1979.

A method for the analysis of aromatic amines in tobacco smoke is presented, and results from analyses of cigarettes are described. Aromatic amines may be important in tobacco carcinogenesis. The amines from the smoke are trapped in dilute hydrochloric acid and are enriched together with the basic portion, derivatized to pentafluoropropionamides and determined by gas chromatography with a ^{63}Ni-electron capture detector (detection limit 50 pg aniline/cigarette). The mainstream smoke of one U.S. 85-mm cigarette without filter tip contained 102 ng of aniline, 61 ng of 2-, 3-, and 4-toluidine, 55.8 ng of ethyl- and dimethylaniline, 4.3 ng of 1-naphthylamine, 6.9 ng of 2-, 3-, and 4-aminobiphenyl, and 5.8 ng of 2-methyl-1-naphthylamine. Sidestream smoke contained levels of aromatic amines 20 to 68 times higher than those in the mainstream smoke. The results from smoke analyses of experimental cigarettes support the concept that the nitrate content and the protein content of tobacco are determining factors for the smoke yields of aromatic amines. (Auth. Abs. Mod.)

PHARMACOLOGY AND TOXICOLOGY

82–0617. Aliperta, A.; Perna, A.; Bellissimo, U.; Chianca, M. C. **Fumo Di Sigarette Ed Alterazioni Istochimiche Del Muco Bronchiale. [Cigarette Smoking and Histochemical Alterations of the Bronchial Mucus.]** *Rassegna Di Patologia Dell'Apparato Respiratorio* 28(3): 261-269, July 1978, Italian.

The relationship between smoking and alterations in mucogenesis was examined by studying the PAS-positive gradient between the gland acini and corresponding secretion ducts. Serious alterations of the glandular mucus were present in 69.9 percent of the smokers (116 out of 166). Normal mucogenesis or only slight alterations were found in the nonsmokers. In smokers with glandular hypersecretion, fucomucin synthesis was affected to a limited degree, if at all, in only a small number of cases (18.7 percent). However, the production of sialomucin was totally suppressed in 61.4 percent of the cases and reduced in 8.4 percent. Solphomucin production was also inhibited, although not as extensively (54.2 percent). In 44 cases of glandular hypertrophy-hyperplasia, where the only exogenous factor was smoking, fucomucin was reduced in 15.9 percent, sialomucin was reduced in 9.1 percent and totally suppressed in 61.4 percent, and solphomucin was reduced in 65.9 percent. Among smokers where glandular hypertrophy of the bronchial wall occurred, a relative reduction in fucomusina, limited or complete inhibition of sialomucins, and no production of solphomucins were found.

82–0618. Allen, R. J.; Wadden, R. A. **Analysis of Indoor Concentrations of Carbon Monoxide and Ozone in an Urban Hospital.** *Environmental Research* 27(1): 136-149, February 1982.

In a hospital study, indoor levels of carbon monoxide (CO) were found to be higher than outdoor levels, a difference attributed to indoor sources of CO, with smoking considered a probable factor. Ozone (O_3) levels were also measured, and found to be lower indoors than outdoors. Carbon monoxide (CO) and ozone (O_3) were monitored in an office and two intensive care units of an urban hospital for 1 year. Annual indoor CO concentrations averaged 0.2 to 1.3 ppm higher than outdoor concentrations (significant at $p<0.05$), with indoor levels of 2.2 to 3.1 ppm. Annual indoor O_3 concentrations averaged 0.005 ppm less than those outdoors (significant at $p<0.05$), with a range of indoor means from 0.007 to 0.013 ppm. The predicted and observed annual average indoor concentrations were within 0.3 ppm for CO for areas with identified sources and 0.003 ppm for O_3. (Auth. Abs. Mod.)

82–0619. Balfour, D.J.K.; Benwell, M.E.M. **Effects of Chronic Nicotine Administration on Plasma Corticosterone and Brain 5-Hydroxyindoles in Stressed Rats.** *British Journal of Pharmacology* 74(1): 212-213, September 1981.

Long-term nicotine treatment effects on plasma corticosterone and on brain levels of 5-hydroxyindoles in rats exposed to psychological stress were examined. Rats were injected with nicotine or saline (controls) for 39 days, while on the 40th day half the nicotine-treated rats received their usual nicotine and half were given saline (withdrawn group). Controls were given saline as usual. In some experiments rats were subjected to psychological stress after each injection, while in other experiments rats received stress only on the 40th day. After the final stress session the rats were measured for brain levels of 5-hydroxytryptamine and 5-hydroxyindole acetic acid, and for plasma corticosterone levels. In rats stressed only on the 40th day, neither long-term nicotine treatment nor withdrawal appeared to have any significant effect on plasma corticosterone or on 5-hydroxyindole levels. Among the rats who were repeatedly stressed, corticosterone levels of nicotine-treated and withdrawn rats were high and significantly different from those of the saline-treated rats. Neither long-term nicotine exposure nor withdrawal significantly changed their brain levels of 5-hydroxyindole.

82–0620. Clarke, P.B.S.; Kumar, R. **Effects of Nicotine on Intracranial Self-Stimulation in Non-Tolerant Rats.** *British Journal of Pharmacology* 74(1): 212P, September 1981.

In nontolerant rats, nicotine has been found to depress or facilitate intracranial self-stimulation, which may reflect an action of nicotine on central reward mechanisms or an effect on the animals' ability to perform motor tasks. Responses to an electrical learning situation were measured in 11 male hooded rats. Independent measures served to control for drug effects upon motor performance. Subcutaneous injections of nicotine tartrate (0, 25, 50, 100, 200, 400 μg/kg base: BDH; neutralized with NAOH) were followed by 80-minute test sessions. Each rat was tested under all conditions in a randomized sequence. Test days were three or four days apart and each rat served as its own control. Results indicated that at low doses subjects responded faster, with a greater proportion of rewarded responses the higher the stimulation current. At higher doses the response accuracy was not enhanced in conjunction with the increased activity produced by nicotine.

82–0621. Dodsworth, H.; Dean, A.; Broom, G. **Effects of Smoking and the Pill on the Blood Count. (Letter).** *British Journal of Haematology* 49(3): 484-488, November 1981.

In blood tests of 425 men and 312 women, the white cell count, mean cell volume, hemoglobin, and packed cell volume were all higher in smokers than in nonsmokers. The changes in parameters were dose-related, and increased with increasing cigarette consumption. The lymphocyte count was also higher, but only significantly so in men. There was no variation in mean cell volume with age in women; however, cell volume increased significantly with age in those women taking the contraceptive pill, regardless of smoking status. The neutrophil count was significantly elevated in all smokers.

82–0622. Downing, R. W.; Rickels, K. **Coffee Consumption, Cigarette Smoking and Reporting of Drowsiness in Anxious Patients Treated With Benzodiazepines or Placebo.** *Acta Psychiatrica Scandinavica* 64(5): 398-408, November 1981.

Data obtained from anxious outpatients treated with either chlordiazepoxide or diazepam (n=533), or placebo (n=285), were used to explore the impact of coffee/tea and cigarette consumption upon the frequency of reporting of drowsiness after 2 weeks of treatment. Strong evidence was provided that both cigarette smoking and coffee drinking affect the frequency with which drowsiness is reported by patients receiving the two benzodiazepines. However, not only the magnitude but also the direction of the impact of coffee consumption upon drowsiness depends upon the level of cigarette smoking and vice versa. For patients smoking ≤ 1 pack of cigarettes/day, drowsiness is reported less frequently by heavy coffee users, while for patients smoking > 1 pack of cigarettes/day, drowsiness is reported more frequently by heavy coffee users. Similarly, for patients drinking ≤ 2 cups of coffee/day, drowsiness occurs less frequently among heavier smokers while for patients drinking > 2 cups of coffee/day, drowsiness occurs more frequently among heavier smokers. These results are consistent with reports that particular substances contained in coffee and tea and in cigarette smoke stimulate the synthesis of hepatic enzymes which metabolize both the benzodiazepines here studied and the substances which have stimulated enzyme synthesis. (Auth. Abs.)

82–0623. Hannan, M. A.; Recio, L.; Deluca, P. P.; Enoch, H. **Co-Mutagenic Effects of 2-Aminoanthracene and Cigarette Smoke Condensate on Smoker's Urine in the Ames *Salmonella* Assay System.** *Cancer Letters* 13(3): 203-212, August 1981.

Cigarette smoke condensate (CSC) and cigarette smoker's urine were each found to exhibit a synergistic effect with the aromatic amine 2-aminoanthracene (2AA) on mutagenicity. CSC and smoker's urine were tested alone and in combination with direct and indirect mutagens for histidine reversion in the Ames *Salmonella* assay system. The synergistic effect was not found when these agents were combined with the direct mutagens, ethylmethane sulfonate and methylmethane sulfonate, or the indirect mutagens, benzo(*a*)pyrene and 9,10-dimethyl-1,2-benzanthracene, tested in this study, nor was the synergistic effect observed when 2AA was tested with urine from a nonsmoker. The results reflected a specificity of the comutagenic action of factor(s) in cigarette smoke or smoker's urine and 2AA, and further studies were suggested to find the basis of the comutagenic effects observed. (Auth. Abs. Mod.)

82-0624. Hecht, S. S.; Lin, D.; Chen, C. B. **Comprehensive Analysis of Urinary Metabolites of N'-Nitrosonornicotine.** *Carcinogenesis* 2(9): 833-838, 1981.

A detailed study of the urinary metabolites of N'-nitrosonornicotine has been performed, employing a simple high pressure liquid chromatographic method. The percentage excretion of the principal urinary metabolites was determined over a dose range of 3-300 mg/kg in the F-344 rat, as follows: 4-hydroxy-4-(3-pyridyl)butyric acid (37.1-53.3 percent, respectively, of the dose), N'-nitrosonornicotine-1-N-oxide (6.7-10.7 percent), norcotinine (3.2-5.1 percent), 4-oxo-4-(3-pyridyl)butyric acid (31.1-12.8 percent), and N'-nitrosonornicotine (3.3-5.2 percent). In the strain A mouse and Syrian golden hamster, the urinary metabolites were qualitatively similar to those observed in the F-344 rat. The interrelationships of the various metabolites of N'-nitrosonornicotine which have been observed in vitro and in vivo were established. The in vitro metabolites resulting from 2'-hydroxylation by liver microsomes, myosmine, and 4-hydroxy-1-(3-pyridyl)-1-butanone were converted, by the F-344 rat, primarily to 4-oxo-4-(3-pyridyl)butyric acid as a urinary metabolite. The in vitro metabolite resulting from 5'-hydroxylation by liver microsomes, 2-hydroxy-5-(3-pyridyl)tetrahydrofuran, gave 4-hydroxy-4-(3-pyridyl)butyric acid as its major urinary metabolite, apparently via 5-(3-pyridyl)tetrahydrofuran-2-one. N'-nitrosonornicotine-1-N-oxide, the remaining major in vitro metabolite, was excreted to a large extent unchanged in F-344 rat urine. The urinary metabolites from 2'-hydroxylation and 5'-hydroxylation of N'-nitrosonornicotine, 4-oxo-4-(3-pyridyl)butyric acid, and 4-hydroxy-4-(3-pyridyl)butyric acid, respectively, were not formed from the in vivo metabolite norcotinine and were not interconverted significantly by the F-344 rat. Thus, these metabolites appear to be reliable indicators for the two possible in vivo α-hydroxylations of N'-nitrosonornicotine. (Auth. Abs.)

82-0625. Heinemann, G.; Schievelbein, H.; Eberhagen, D.; Rahlfs, V. **Der Einfluss von Nahrungsmitteln und Rauchen auf die Klinisch-Chemische Diagnostik von Phaochromozytom, Neuroblastom und Karzinoid-Syndrom. [The Influence of Diet and Smoking on Clinical Chemical Diagnosis of Pheochromocytoma, Neuroblastoma, and Carcinoid Syndrome.]** *Klinische Wochenschrift* 59(20): 1165-1173, October 15, 1981, German.

The possible effect of cigarette smoking on the diagnosis of secondary hypertension caused by adrenergic tumors, e.g., pheochromocytoma (Ph), was investigated by determining the catecholamines (epinephrine (E) and norepinephrine(NE)) and their metabolites (vanilline mandelic acid (VMA) and metanephrines (MN)) in 24-hour urine of smokers and nonsmokers (controls). During 14-day periods, 29 male and female smokers each consumed an average of 24 cigarettes/day. Each cigarette contained 0.49 mg nicotine and 8.2 mg tar. At the end of each smoking period, E, NE, VMA, and MN were determined in urine of the smokers and the 19 controls. Statistical evaluation of the data obtained did not show a significant difference in any of the four variables between the smokers and nonsmokers. It is concluded that, apparently, smoking of 20 to 30 cigarettes/day does not stimulate an increased excretion of E, NE, VMA, and MN in urine, and hence does not interfere with diagnosis of secondary hypertension due to Ph.

82-0626. Hocko, M. **Toksikologichna Kharakteristika na Glikolmonoatsetata. [Toxicologic Characteristics of Glycol-Monoacetin.]** *Problemi na Khigienata* 5: 92-97, 1980, Bulgarian.

Acute and subacute toxicity of glycol monoacetin, which is used in the production of cigarette filters, was studied in tests with animals. Glycol monoacetin was administered either orally to test the exposure of a cigarette consumer, or by inhalation, or by dermal exposure in view of the possible effect on workers. Symptoms of poisoning and mortality were observed only upon oral administration of acute doses of the liquid extract from cigarette filters. Erythrocytes and leukocytes decreased significantly after a 30-day oral application of the extract. A slight irritation of the upper respiratory tract or skin was observed in mice and rats after forced inhalation of warm air through a cigarette filter or 21-day application of the substance to the skin. It was concluded that glycol monoacetin is only mildly toxic regardless of application method, and its cumulative effect is insignificant. The observed effects on liver function in the form of decreased activity of certain enzymes and parenchymatous distrophy are considered to be transient. In acute poisoning cases (at 9 ml/kg weight approximate lethal dose), glycol monoacetin affects the nervous system.

82-0627. Igarashi, S. **Kitsuen To Kimo I: Baiyokimo Saibo Nitaisuru Nikochin no Eikyo. [Tobacco Smoking and the Liver. I. Effects of Nicotine on Cultured Hepatic Cells.]** *Kanzo* 21(10): 1390, 1980, Japanese.

Nicotine in liver cells was found to prohibit protein synthesis. Nicotine at 0.33, 3.3, 33, or 333 μg/ml with ^{14}C-leucine 1 μc was added to solutions of liver and incubated for 6 hours at 37°C. After cleaning, the Lowry method and the anthrone method were applied. Results were presented in a table and appear to indicate that nicotine in the liver cells reduced the incorporation of ^{14}C-leucine.

82-0628. Ikushima, S.; Muramatsu, I.; Fujiwara, M. **Effects of 4-Aminopyridine on the Adrenergic Nerve Terminals of Rabbit Arteries.** *Journal of Pharmacology and Experimental Therapeutics* 219(3): 792-797, December 1981.

Adrenergic nerves of the rabbit pulmonary artery and aorta were stimulated by elecrical pulses and by nicotine. The effects of 4-aminopyridine (4-AP) were investigated on contractile responses of these arteries or on ^{3}H-efflux from (^{3}H)norepinephrine-treated pulmonary artery in response to these stimuli. Tetrodotoxin (0.1-0.3 μM) abolished the contractions and the ^{3}H-efflux induced by an electrical pulse of 0.3 msec duration, but not the adrenergic responses induced by a pulse of 5 msec duration or by nicotine. The adrenergic responses to an electrical pulse of 5 msec (with tetrodotoxin) or nicotine were inhibited by guanethidine (10 μM) or removal of the extracellular calcium. The adrenergic response induced by nicotine was little affected by 4-AP in concentrations up to 100 μM. At 300 μM of 4-AP the concentration-contraction curve of nicotine shifted to the right, and ^{3}H-efflux was markedly reduced. This inhibitory effect of 4-AP on the contraction was not affected by alterations in the concentrations of extracellular calcium. 4-AP did not affect the responses to exogenously applied norepinephrine. These results indicate that 4-AP has inhibitory effects on nicotinic receptors of adrenergic nerve terminals and that nicotine releases norepinephrine in a manner which differs from the release seen with electrical stimulation. (Auth. Abs. Mod.)

82–0629. Jaeger, R. J. **Carbon Monoxide in Houses and Vehicles.** *Bulletin of the New York Academy of Medicine* 57(10): 860-872, December 1981.

Carbon monoxide, found in indoor as well as outdoor environments, is produced indoors by combustion of natural gas, as well as by tobacco smoking. In a clearly dose—responsive manner, carbon monoxide concentration in the expired air of smokers increases with number and temporal proximity to cigarettes smoked. Concentrations in human blood range from less than 1 percent in nonsmokers to 5-10 percent in smokers. In fetuses of smoking mothers, as much as 7.6 percent of carboxyhemoglobin was reported. Such voluntary intoxication as well as involuntary exposure associated with indoor air pollution are considered with respect to the effects of carbon monoxide on behavior and performance of the offspring. (Auth. Abs. Mod.)

82–0630. Janus, J.; Kaminska-Kosmider, A. **Zawartosc Wazopresyny w Podwzgorzu i Czesci Nerwowej Przysadki u Swinek Morskich Poddanych Dlugotrwalemu Dzialaniu Dymu Tytoniowego. [Vasopressin Content in Hypothalamus and Neurohypophysis of Guinea Pigs After Long-Term Exposure to Tobacco Smoke.]** *Endokrynologia Polska* 32(1): 53-57, 1981, Polish.

Seven male guinea pigs were exposed for 2 hours/day, 6 days per week, to air with tobacco smoke from seven to eight cigarettes during each 2-hour session. After 12 weeks of exposure, the animals were killed, their neurohypophysis and hypothalamus were separated out, and extracts from both were prepared for determination of their respective vasopressor activities. At the same time, the vasopressin activity was determined in extracts from the organs of 10 control animals. A significant (about 58 percent) decrease in vasopressin content of hypothalamus was found in the tobacco smoke exposed animals as compared with the controls. The vasopressin content of neurohypophysis in the exposed animals was not significantly different from that in the controls.

82–0631. Janzon, L.; Lindell, S.-E.; Trell, E.; Larme, P. **Smoking Habits and Carboxyhaemoglobin. A Cross-Sectional Study of an Urban Population of Middle-Aged Men.** *Journal of Epidemiology and Community Health* 35(4): 271-273, December 1981.

A cross-sectional population study on the distribution of carboxyhemoglobin concentrations in the morning, before smoking, in a Swedish urban population of 1,037 men born in 1931, shows that the median carboxyhemoglobin concentration increased as daily tobacco consumption increased. The median concentration was the same in nonsmokers as in ex-smokers: 0.5 percent. It increased with increasing daily tobacco consumption. However, when carboxyhemoglobin concentrations are measured in reasonably well-standardized circumstances there are large variations between individuals, even in those who smoke equal amounts of tobacco a day. This makes it difficult to predict the concentration in the individual smoker when only his daily tobacco consumption is known. Measurements of carboxyhemoglobin concentration should be a valuable complement to smoking history to identify the smoker at high risk of cardiovascular disease, to provide an extra argument to make the patient give up the habit, and to reinforce the efforts of those who try to do so. (Auth. Abs. Mod.)

82–0632. Juan, H. **Influence of Nicotine on Basal and Stimulated Prostaglandin Biosynthesis in Perfused Vascular Tissue of the Rabbit.** *Archives of Pharmacology* 317(4): 345-350, November 1981.

The prelabelling technique (incorporation of (1-^{14}C)-arachidonic acid) was used to investigate the influence of nicotine (in comparison with acetylcholine) on release and metabolism of arachidonic acid in the isolated perfused rabbit ear. Increasing amounts of nicotine (up to 300μg) and acetylcholine (or bethanecol) were tested to measure the release of prostaglandins. The results show that acetylcholine first stimulates the release of prostacyclin from peripheral vessels via muscarinic receptors and then reduces the stimulated release of prostaglandins possibly due to tachyphylaxis. Nicotine strongly reduces the stimulated release of prostacyclin and PGE_2 possibly due to inhibition of the cyclo-oxygenase. A further influence on the following enzymes, e.g., on prostacyclin synthetase, cannot be excluded. A reduced generation of prostacyclin by nicotine may lead to a reduced protection of the endothelium and to an increased tendency of platelet aggregation, especially in predamaged vessels. (Auth. Abs. Mod.)

82–0633. Luepker, R. V.; Pechacek, T. F.; Murray, D. M.; Johnson, C. A.; Hund, F.; Jacobs, D. R. **Saliva Thiocyanate: A Chemical Indicator of Cigarette Smoking in Adolescents.** *American Journal of Public Health* 71(12): 1320-1324, December 1981.

Saliva thiocyanate (SCN) levels, used as a chemical measure of smoking, tended to be higher in smokers than in nonsmokers in a study group of 1,419 eighth grade students. In that group, 54.9 percent of students admitting to regular smoking of > one pack/week had thiocyanates ≥ 100 μg/ml compared with 2.3 percent nonsmokers at those levels. Of students who smoked ≥ 10 cigarettes in the prior 24 hours, 66.7 percent were at or above 100 μg/ml. Elevated SCN in nonsmokers was uncommon. Saliva SCN is a safe, inexpensive, and acceptable prevalence measurement for cigarette smoking. It can be used in place of self-reports to document smoking of greater than one pack/week in populations of youth. (Auth. Abs. Mod.)

82-0634. Mishra, S. K.; Raviprakash, V. **Non-Adrenergic Inhibitory and Non-Cholinergic Excitatory Neural Involvement in DMPP and Nicotine Action on Fowl Rectum.** *Archives Internationales de Pharmacodynamie et de Therapie* 253(2): 210-219, October 1981.

Pharmacological analysis of the mechanical responses of fowl rectum to 1,1-dimethyl-4-phenylpiperazinium iodide (DMPP) and nicotine showed that nicotinic stimulation produced varied responses on different segments. On the whole/distal segment, DMPP and nicotine induced relaxations. On the proximal segment, DMPP induced relaxation whereas nicotine produced a biphasic response. Termination of the adrenergic function converted the relaxatory responses of DMPP and nicotine to biphasic ones, the contractile component of which was potentiated by neostigmine and partially inhibited by atropine. This response was completely abolished by ganglion blockers. On cooled tissue preparations, neither nicotinic nor electrical stimulations elicited any response. These findings implicate the participation of nonadrenergic inhibitory and non-cholinergic excitatory neural elements besides the adrenergic and cholinergic ones to nicotinic stimulation. (Auth. Abs.)

82-0635. Muranaka, H.; Tamada, T. **Taiekichu Nikochin Oyobi Sono Taishasanbutsu no Biryoteiryo 2. Kecchu Nikochin, Kochinin no Chushutsuteiryoho no Kento Oyobi Sono Oyo. [Microdetermination of Nicotine and Its Metabolites in Biological Fluid: 2. A Study on Extraction Method of Nicotine and Cotinine in Plasma and Its Applications.]** *Rinsho Kagaku* 9(3): 290-299, 1980, Japanese.

A study on the extraction method of plasma nicotine and cotinine for FTD gas chromatography is described. Ether anhydrous is a suitable organic solvent for nicotine, as is chloroform for cotinine. An internal standard method using quinoline or 5-aminoquinoline is profitable to obtain a good result. Two kinds of extraction methods were adopted, the single determination of nicotine, and the simultaneous determination of nicotine and cotinine. Reproducibility of the methods was good and the recovery of nicotine and cotinine was approximately 90-100 percent of added ones to plasma. The method was sufficiently sensitive to detect nicotine of 0.1 ng/ml and cotinine of 0.2 ng/ml. The concentration of nicotine in plasma of 10 nonsmokers was 2.5±2.93 ng/ml and cotinine could not be detected. Plasma nicotine and cotinine concentrations of 20 smokers before smoking were 14.5±8.68 ng/ml and 145.3±138.23 ng/ml, respectively. Reagent blank value in these methods was about 2-3 ng/ml as nicotine and none as cotinine, which may be due to contaminations in reagent and during the procedure. Plasma nicotine rose to maximum value immediately or 15 minutes after voluntary smoking of one cigarette and fell to the former value after 1 hour. In the case of plasma cotinine, its rise and fall was more gradual in comparison with nicotine and the retention time in plasma was longer than nicotine. (Auth. Abs.)

82-0636. Muranaka, H.; Tamada, T.; Higashi, E.; Itani, S. **Biryokessho o Mochiiru Nikochin Teiryoho. [A Microextraction Method for the Determination of Plasma Nicotine.]** *Rinsho Kagaku* 9(4): 386-390, 1980, Japanese.

A microextraction method for the determination of plasma nicotine, which required only 100μl of sample, is described. Gas chromatographic technique was in accordance with the condition of previous reports. Recovery of nicotine in plasma was about 100 percent and reproducibility of the method was sufficient for practical use. Plasma nicotine level of 20 nonsmokers was 4.6±2.08 ng/ml and in 8 smokers, before smoking, was 14.8±10.74 ng/ml. After smoking one cigarette (nicotine content in smoke is 0.8 mg/cigarette), nicotine level in plasma increased to maximum value just after smoking (30.4±21.49 ng/ml) and decreased rapidly to former value for 60 minutes after. Reagent blank value in this method was about 2-3 ng/ml as nicotine. This method is a profitable technique for a rapid determination of plasma nicotine. (Auth. Abs.)

82-0637. National Academy of Sciences. **The Health Effects of Nitrate, Nitrite, and *N*-Nitroso Compounds.** Washington, D.C., National Academy Press, 1981, 538 pp.

The sources and health effects of nitrate, nitrite, and *N*-nitroso compounds are reported. These substances are explored with respect to utility in preserving foods, their chemistry, environmental distribution, and their metabolism and pharmacokinetics in humans. While much of the research focuses on foodstuffs, tobacco products are included as an important source of human exposure. The levels of nitrosamines in tobacco products range from an average of 0.87 μg per U.S. filter cigarette to an average of 1.4 μg per French filter cigarette and approximately 11 μg for one small cigar. If these data are typical of an average cigarette, a pack of 20 U.S. filter cigarettes would provide an intake of approximately 17 μg. By comparison, the intake of nitrosamines from all dietary sources, including beer, has been estimated to be only 1.1 μg per day.

82-0638. Nishioka, H.; Nishi, K.; Kyokane, K. **Human Saliva Inactivates Mutagenicity of Carcinogens.** *Mutation Research* 85(5): 323-333, October 1981.

Human saliva was found to inactivate or decrease the mutagenicity of several substances, including benzo(*a*)pyrene (BaP) and cigarette smoke condensate. Mutagenicities of BaP, 2-(2-furyl)-3-(5-nitro-2-furyl)acrylamide, N-methyl-N'-nitro-N-nitrosoquanidine, 4-nitroquinoline 1-oxide, aflatoxin B_1, and tryptophan pyrolysate-1, with or without metabolic activation, were inactivated by treatment with human saliva to a great extent in the Ames test with *Salmonella typhimurium* test strains TA98 and TA100. Mutagenic activities of quercetin, pyrolysates of beef, salmon, and sodium glutamate, and condensate of cigarette smoke were also decreased to some extent by saliva treatment. These effects showed individual variations. Complex mechanisms may be involved in the inactivation of mutagenicity of carcinogens by saliva, including biochemical reactions with enzymes, vitamins, etc. and/or adsorption with high molecular weight substances in saliva such as proteins, bacterial cells, mucous materials, etc. (Auth. Abs. Mod.)

82–0639. Norioka, T.; Momiyama, K.; Oosone, A.; Saito, Y. **Chokikan Nikochin Toyo no Ratto ni Okeru Ketsuatsu, Taijyu, Kesseishishitsu e no Eikyo. [Effect of Nicotine Drinking on Blood Pressure, Body Weight and Serum Lipids of Rats.]** *Japanese Journal of Alcohol Studies and Drug Dependence* 16(2): 133-140, June 1981, Japanese.

Nicotine was found to influence blood pressure, body weight, and serum lipids in rats. Nicotine was administered to male rats weighing 200 g and 300 g over a period of 27 weeks, by adding low doses (1.14 or 2.28 mg/kg/day) or large doses (3.42 or 4.56 mg/kg/day) to drinking water. The amount of nicotine calculated to be equivalent to that for "one-pack a day" is 1.14 mg/kg/day. The chronic administration of nicotine produced a gradual lowering of systolic pressure of the rats, and its withdrawal resulted in a fluctuating pressure response. In the other group, the chronic administration of nicotine at 4.56 mg/kg/day and 37.6 ml of 15 percent alcohol produced a gradual elevation of systolic pressure. Nicotine showed a tendency to increase body weight in all groups, but rats drinking both nicotine and alcohol showed no clear increase. Nicotine drinking rats tended to show an increase of serum lipids in all groups, while the withdrawal of nicotine tended to show a decrease in all groups. The total cholesterol of other groups, except the group drinking both nicotine and alcohol, increased; it decreased at 5 weeks after the withdrawal of nicotine. On the other hand, the rate of decline of HDL-cholesterol was not so remarkable. The effect of nicotine drinking on lipoperoxide was not the same as for other lipids. The administration of the same dose of nicotine to spontaneously hypertensive rats showed a lowering of blood pressure and of the rate of increase in body weight, compared with controls. (Auth. Abs. Mod.)

82–0640. Paigen, B.; Ward, E.; Steenland, K.; Bolanowska, W.; Gessner, T.; Chang, R. L.; Wood, A. W.; Conney, A. H. **Absence of Seasonal Variation in Antipyrine Metabolism.** *Clinical Pharmacology and Therapeutics* 31(2): 144-150, February 1982.

The induced activity of aryl hydrocarbon hydroxylase (AHH), measured by the metabolism of benzo(*a*)pyrene to fluorescent products in cultured human lymphocytes, shows a strong seasonal variation. The in vivo metabolism of antipyrine, which is also catalyzed by microsomal cytochrome P-450-dependent monooxygenases, has been reported to be correlated with AHH inducibility in human lymphocytes. To determine whether antipyrine metabolism also showed seasonal changes, we measured antipyrine half-life in 10 nonsmokers and 8 smokers at the two times of the year that correspond to the high and low peaks of inducible AHH activity as measured in lymphocytes. The mean antipyrine half-life determined in all 18 subjects in summer was almost identical to that found in winter. AHH activity in cultured human lymphocytes from the nonsmoking subjects was determined in control and 3-methylcholanthrene-induced cells to obtain inducibility ratios of 4.2±0.56 (SEM) in the summer and 1.4±0.14 (SEM) in the winter. These results indicate that the seasonal variation in AHH inducibility in human lymphocytes is not reflected by a corresponding seasonal variation in antipyrine metabolism in vivo. (Auth. Abs.)

82–0641. Shamberger, R. J.; Bratush, C. M. **Cadmium, Selenium and Zinc Levels in Kidneys.** In: Hemphill, D. D. (Editor). *Trace Substances in Environmental Health.* 14th Symposium, University of Missouri, Columbia, 1980, pp. 203-210.

Cadmium (Cd), selenium (Se), and zinc (Zn) levels were measured from the subscapular region of the kidney cortex from 123 patients who died from different diseases. Selenium content was lower in patients with cancer or cancer metastases. Zinc levels were significantly higher in kidneys from patients with cancer or patients with cancer and metastases. The Zn/Se ratio had a greater significant difference from controls than did Se or Zn alone. Male and female nonsmokers and cigar smokers had a significantly lower kidney Cd content than cigarette smokers. Nonsmoking cancer patients with metastases had a greater kidney Cd content than nonsmokers with other diseases. Hypertensive and atherosclerotic nonsmokers, renal patients, and nonwhites did not have a significantly greater Cd content than did patients with other diseases. The Cd/Se ratios were greater in cancer patients and cancer patients with metastases than in the controls. There was a positive significant correlation between Zn and Cd in 123 patients. (Auth. Abs.)

82–0642. Shaper, A. G.; Pocock, S. J.; Walker, M.; Wale, C. J.; Clayton, B.; Delves, H. T.; Hinks, L. **Effects of Alcohol and Smoking on Blood Lead in Middle-Aged British Men.** *British Medical Journal* 284(6312): 299-302, January 30, 1982.

A survey of middle-aged men in 24 British towns showed a strong association between blood lead concentrations, alcohol consumption, and cigarette smoking. The association with alcohol persisted after age, social class, body mass index, cigarette smoking, water lead concentrations, and the town of residence had been taken into account. There was an independent but less pronounced association between cigarette smoking and blood lead concentrations after adjustment for the other factors. The possible mechanisms include a decreased excretion of lead due to alcohol-induced hepatic dysfunction and an increased lead intake from cigarette smoking. These findings have implications for widespread measurement of blood lead concentrations in adults in the community and for all studies attempting to relate blood lead concentrations to environmental exposure. (Auth. Abs.)

82–0643. Sprince, H. **Ascorbic Acid, Sulfur Compounds, and Anti-Adrenergic Agents as Protectants Against Acetaldehyde Toxicity: Implications in Alcoholism and Smoking.** *British Journal on Alcohol and Alcoholism* 16.1: 5-9, 1981.

Acetaldehyde appears to be a toxic agent of heavy smoking and of heavy drinking. Several possible protectants against its effects may be available. Acetaldehyde in cigarette smoke is ciliatoxic, and also inhibits the phagocytic activity of lung alveolar macrophages. Its relative importance is not certain. Acetaldehyde is a powerful releasing agent of tissue catecholamines. Several possible protectants against acetaldehyde toxicity are suggested, including ascorbic acid, several sulfur compounds, and certain anti-adrenergic agents.

82–0644. Topping, D. L. **Effects of Tobacco Smoke and Its Constituents on Lipid and Carbohydrate Metabolism.** In: Clemens, M. J.; Ashwell, M. (Editors). *Biochemistry of Cellular Regulation.* Volume II. Clinical and Scientific Aspects of the Regulation of Metabolism. Boca Raton, Florida, CRC Press, Inc., 1980, pp. 165-183.

Tobacco smoke and its constituents, especially nicotine and carbon monoxide, are discussed relative to their possible effects on lipid and carbohydrate metabolism. Considerations which affect the composition of inhaled smoke are mentioned, as well as difficulties encountered in using animals in smoking experiments. One apparent acute effect of nicotine is a rise in plasma free fatty acid levels, an effect which is considered with regard to myocardial metabolism, plasma lipoprotein secretion, and body temperature. Although much is known of nicotine's effects on lipid metabolism, less is known about its effects on carbohydrate metabolism. Overall, little is understood about the metabolic effects of chronic nicotine administration. The possible acute effects of carbon monoxide on lipid metabolism of the liver, and heart and arterial tissue are examined. Carbon monoxide's possible chronic effects on atherosclerosis and plasma lipoprotein metabolism are mentioned; however, little information is available about carbon monoxide's chronic metabolic effects. Further work in the field of smoking and metabolism is suggested.

82–0645. van Cantfort, J.; Gielen, J. E. **Ontogenetic Variation in Rat Liver, Lung, and Kidney Monooxygenase Induction by Low Doses of Benzo(*a*)pyrene and Cigarette-Smoke Condensate.** *British Journal of Cancer* 44(6): 902-910, December 1981.

The specific lung aryl hydrocarbon hydroxylase (AHH) induction, previously observed after the inhalation of cigarette smoke, is not due to the route followed by the inhaled smoke, for the same phenomenon occurs after i.p. injection of either cigarette smoke condensate (CSC) or benzo(*a*)pyrene (BaP) in low doses. In this respect lung AHH behaves completely differently from the liver and kidney enzyme, in which organs, basal AHH activity (which is low in the fetus) increases rapidly after birth to reach the adult level 2 months later, and is only inducible by CSC and low doses of BaP in unweaned rats. In the lung, the basal AHH activity (low in the fetus) increases abruptly at birth, peaks in 5-day-old rats, and then decreases slightly. Contrary to enzyme activity in other tissues, lung AHH cannot be induced in unweaned young animals. The enzyme subsequently becomes sensitive to inducing agents and is highly inducible in 90-day-old rats. Similar behavior occurs in two other enzymes linked to cytochrome P_1450: ethoxycoumarin deethylase and ethoxyresorufin deethylase. The results could be related to the particular susceptibility of the lung to develop cancer after the inhalation of cigarette smoke. (Auth. Abs.)

82–0646. Vincek, W. C.; Martin, B. R.; Aceto, M. D.; Tripathi, H. L.; May, E. L.; Harris, L. S. **Synthesis of 4,4-Ditritio-(+)-nicotine: Comparative Binding and Distribution Studies With Natural Enantiomer.** *Journal of Pharmaceutical Sciences* 70(11): 1292-1293, November 1981.

The preparation of 4,4-ditritio-(+)-nicotine (V*b*) (specific activity 10.3 Ci/mmole) from (+)-nicotine (I*b*) via (—)-4,4-dibromocotinine (III*b*)is described. The resulting V*b* has facilitated binding and disposition experiments with I*b* and with I*a* or (—)-nicotine. Although I*b* is 10-30 times less potent than (—)-nicotine (I*a*) in the central nervous system, its binding affinity for the crude mitochondrial or nuclear fraction of whole rat brain is only three times less than that of I*a*. However, distribution studies showed that the maximum brain levels of (—)-(^{3}H)nicotine are nearly twice those of (+)-(^{3}H)-nicotine following administration of a 2-µg/kg dose. Binding affinity and disposition of the stereoisomers account for a portion of the pharmacological stereospecificity of nicotine. (Auth. Abs. Mod.)

82–0647. Wo, Y.I.S.; Park, K. H. **Butilsetedhaidrosianisol Kwa Flavone Uihan Yon'gisongbunui Saengch'ense Hwalsongokchee Kwanhan Yon'gu. [The Inhibition Effects of Butylatedhydroxyanisole and Flavone on the Microsomal Activation of Cigarette Smoke Components in Rats.]** *Journal of the Korean Society of Tobacco Sciences* 2(2): 8-13, 1980, Korean.

Flavone and butylatedhydroxyanisole (BHA) each appeared to inhibit the microsomal activation of cigarette smoke condensate (CSC) in rats, although flavone may have been the stronger inhibitor. The activities of lactic acid dehydrogenase (LDH) in serum were measured in time intervals of 3, 6, 12, 18, 24, and 30 hours, respectively, after the injection of CSC at 5 mg/kg or of its Neutral Portion (NP) at 10 mg/kg in Wistar male rats. Flavone (1 mg/kg) and BHA (1 mg/kg) were injected along with CSC or NP. The significant enhancement of LDH activity in serum was observed in both cases of rats treated with CSC and NP. A drastic decrease of LDH activity was observed after 12 hours of injection of CSC along with flavone. In contrast with flavone, BHA reduced the enzyme activity to a lesser extent. Flavone can be considered a better inhibitor of the action of CSC in vivo than is BHA. (Auth. Abs. Mod.)

See also, **82-0813**

MORTALITY AND MORBIDITY

82-0648. Ashley, M. J.; Frecker, R. C. (Editors). **Proceedings of the Conference, Smoking or Health in the 80's.** *Canadian Journal of Public Health* 72(6): 369-420, November-December 1981.

Proceedings of the conference, Smoking or Health in the 80's, sponsored by the Ontario Council of Health, are presented. Canada has one of the world's highest per capita consumption rates, though smoking is on the decline. Therefore, the focus of the conference was on the overall detrimental effects of cigarettes on health, and possible formal control of the smoking habit. Subjects covered include smoking-related cancers, chemical carcinogenesis, atherosclerosis, the pathophysiological effects on the respiratory system, maternal smoking in human reproduction, involuntary smoking, the cultural determinants of tobacco use, the self-treatment of smoking and obesity, legislation and restrictive measures, the role of public information and education in control of smoking, and the government/media influence in cessation of smoking.

82-0649. Doll, R. **The Smoking-Induced Epidemic.** *Canadian Journal of Public Health* 72(6): 372-381, November-December 1981.

A global view of the harmful effects of smoking is presented. Worldwide studies of morbidity and mortality trends demonstrate various facets of the dilemma, such as the principal effects on health in general, effect on women, passive smoking findings, and interactive effects with other factors (diet, alcohol, asbestos). It is concluded that the epidemic of smoking-induced disease is beginning to disappear from the countries in which it first took hold, in particular Britain, Finland, and Austria, but it would be unfortunate if, while this is happening, developing countries will repeat the experience.

82-0650. Klebba, A. J. **Mortality From Diseases Associated With Smoking: United States, 1960-77. Data From the National Vital Statistics System, Series 20, No. 17.** U.S. Department of Health and Human Services, Public Health Service, Office of Health Research, Statistics, and Technology, National Center for Health Statistics, DHHS Publication No. (PHS) 82-1854, January 1982, 82 pp.

In successive reports on smoking and health issued by the Surgeon General, 14 diseases were identified for which mortality was considered to be related to smoking. This report examines trends for these 14 groups for 1950-77 and updates an earlier National Center for Health Statistics (NCHS) report that showed trends for 12 of these cause groups for 1950-64. The eight diseases considered to be causally related to smoking (particularly cigarette smoking) are (1) malignant neoplasm of the trachea, bronchus, and lung; (2) malignant neoplasm of the larynx; (3) malignant neoplasm of the lip; (4) chronic and unqualified bronchitis; (5) ischemic heart disease; (6) emphysema and chronic obstructive lung disease without mention of asthma, bronchitis, or emphysema; (7) malignant neoplasm of the esophagus; and (8) malignant neoplasm of the buccal cavity and pharynx, excluding lip. Between 1950 and 1977 the trend of the age-adjusted death rate was upward for the first of these causes, relatively stable for the second and third, downward for the fourth and fifth groups of causes, and upward for the remaining three groups. The six causes or groups of causes considered to be statistically related to smoking are: (1) cirrhosis of the liver; (2) peptic ulcer; (3) malignant neoplasm of the bladder; (4) five specified noncoronary cardiovascular diseases; (5) malignant neoplasm of the pancreas; and (6) malignant neoplasm of the kidney. (Auth. Abs.)

82-0651. McEvoy, L.; Land, G. **Life-Style and Death Patterns of the Missouri RLDS Church Members.** *American Journal of Public Health* 71(12): 1350-1357, December 1981.

If the disparate mortality patterns found for members of the Reorganized Church of Jesus Christ of Latter-Day Saints (RLDS) in Missouri compared to those of Mormons in Utah (who advocate similar lifestyles) persist under more direct comparisons, they could provide an opportunity to assess the impact of similar lifestyles in separate settings. Comparisons of mortality data from 1972 to 1978 for the Missouri RLDS showed lower rates than non-Mormon whites in Missouri, non-Mormons of Independence, Missouri, and Utah Mormons. Utah Mormons had nonsignificantly lower rates for lung cancer and ischemic heart disease, but the Missouri RLDS had significantly lower rates for pneumonia/influenza and violent deaths. Lifestyle effects (Mormons are dissuaded from the use of tobacco, alcohol, and hot drinks) can be seen in mortality linked with smoking in the Missouri RLDS, compared with the other Missouri groups: lung cancer, ischemic heart disease, pneumonia, and influenza all showed the expected relationships.

82-0652. Silverberg, E. **Cancer Statistics, 1982.** *Ca-A Cancer Journal for Clinicians* 32(1): 15-31, January-February 1982.

Estimates of cancer incidence for 1982 are presented in graphic and tabular form, based on data from the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) Program (1973-1978). Information is broken down by age, sex, cancer site, mortality, new cases, age-adjusted death rates for 42 selected countries, 5-year survival rates by site and race, and cancer incidence in children. Nonmelanoma skin cancer and carcinoma in situ are not included. Lung cancer mortality in 1978 for males was 34 percent (n = 71,006) and for females, 16 percent (n = 24,080). Estimated lung cancer mortality for 1982 is 80,000 males and 31,000 females.

82-0653. Treffert, D. A. **"Rustproofing" People. Wellness in Perspective.** *Postgraduate Medicine* 71(1): 179-184, January 1982.

Wellness, prevention, prospective medicine, and risk appraisal must be aligned to a reliable data base gathered by careful study and inquiry. Prospective medicine and risk appraisal are attempts to substitute data for slogans and facts for beliefs in an effort to make wellness a credible science. The following risk factors are identified and discussed: smoking, motor vehicle accidents, rich diet, sedentary lifestyle, alcohol, and obesity. Unequivocally, cigarette smoking is the most serious risk factor, implicated in most studies of coronary heart disease (CHD), lung cancer, and chronic obstructive lung disease. Smoking more than 10 cigarettes/day raises the risk of arteriosclerotic heart disease 150 percent and lung cancer 50 percent. Total mortality in various age groups is about twice as high among cigarette smokers as among nonsmokers. Of this excess mortality, 19 percent is due to lung cancer and 37 percent to CHD. A cautionary note is included: to maintain a sensible balance in "rust proofing," preservation of health must not be allowed to overshadow enjoyment of life.

82-0654. U.S. Department of Health and Human Services. **The Health Consequences of Smoking for Women. A Report of the Surgeon General.** U.S. Department of Health and Human Services, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, 1980, 385 pp.

The first signs of an epidemic of smoking-related disease among women are now appearing. Because women's cigarette use did not become widespread until the onset of World War II, those women with the greatest intensity of smoking are now only in their thirties, forties, and fifties. As these women grow older, and continue to smoke, their burden of smoking-related disease will grow larger. Four main themes emerge from this report to guide future public health efforts. First, women are not immune to the damaging effects of smoking already documented for men. The apparently lower susceptibility to smoking-related diseases among women smokers is an illusion reflecting the fact that women lagged one-quarter century behind men in their widespread use of cigarettes. Second, cigarette smoking is a major threat to the outcome of pregnancy and well-being of the newborn baby. Third, women may not start smoking, continue to smoke, quit smoking, or fail to quit smoking for precisely the same reasons as men. Unless future research clarifies these differences, we will find it difficult to prevent initiation or to promote cessation of cigarette smoking among women. Fourth, the reduction of cigarette smoking is the keystone in our nation's long-term strategy to promote a healthy lifestyle for women and men of all races and ethnic groups. (Auth. Abs. Mod.)

82-0655. U.S. Department of Health and Human Services **The Health Consequences of Smoking. The Changing Cigarette: A Report of the Surgeon General.** U.S. Department of Health and Human Services, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHHS Publication No. (PHS)81-50156, 1981, 269 pp.

Indications are that there is no safe cigarette and no safe level of consumption. Smoking cigarettes with lower yields of tar and nicotine reduces the risk of lung cancer and, to some extent, improves the smoker's chance for longer life, provided there is no compensatory increase in the amount smoked. However, the benefits are minimal in comparison with giving up cigarettes entirely. It is not clear what reductions in risk may occur in the case of diseases other than lung cancer. The evidence in the case of cardiovascular disease is too limited to warrant a conclusion, nor is there enough information on which to base a judgment in the case of chronic obstructive lung disease. In the case of smoking's effects on the fetus and newborn, there is no evidence that changing to a lower tar and nicotine cigarette reduces risk. Carbon monoxide has been impugned as a harmful constituent of cigarette smoke. There is no evidence, however, of changes in the risk of diseases due to variations in carbon monoxide levels. Smokers may increase the number of cigarettes they smoke and inhale more deeply when they switch to lower yield cigarettes. Compensatory behavior may negate any advantage of the lower yield product or even increase the health risk. The tar and nicotine yields obtained by present testing methods do not correspond to the dosages that the individual smokers receive; in some cases they may be seriously underestimated. The cigarettes being produced today may introduce new risks through their design, filtering mechanisms, tobacco ingredients, or additives. The chief concern is additives. The Public Health Service has been unable to assess the relative risks of cigarette additives because information was not available from manufacturers as to what these additives are. (Auth. Abs. Mod.)

82-0656. Wingard, D. L. **The Sex Differential in Mortality Rates: Demographic and Behavioral Factors.** *American Journal of Epidemiology* 115(2): 205-216, February 1982.

MORTALITY AND MORBIDITY

Increased smoking among men relative to women was examined as a factor in the difference in male and female mortality rates. The unadjusted relative mortality risk for men compared to women is 1.5 ($p < 0.001$). Adjustment for smoking and alcohol consumption factors decreased this difference, while others, such as physical activity and marital status, increased the difference in mortality rates. The other factors considered were age, race, socioeconomic status, occupation, physical health status, use of health services, weight, sleeping patterns, social contacts, church and group membership, and life satisfaction. Adjustment for all of these behavioral risk factors does not account for the difference in male and female mortality rates; an interaction of biologic and behavioral elements may more fully explain this difference.

82-0657. Zamorano, T. **Tabaco o Salud: Elija. [Smoking or Health: The Choice Is Yours.]** *Revista Medica de Chile* 108(8): 761-767, 1980, Spanish.

The health hazards of smoking to smokers and nonsmokers are discussed, and an antismoking campaign is proposed. The harmful constituents of tobacco include: nicotine, cancerogenous substances (found mainly in the smoke), irritants (responsible mainly for effects on the bronchi), and carbon monoxide. The major risks associated with smoking include cancer, cardiovascular disease, chronic bronchitis, pulmonary emphysema, and effects on the unborn child and the young. Many more women have begun the habit and lung cancer is now as widespread among the female population as it is among males. One-third of the deaths of working age males in developed nations is due to coronary disease, while three out of four cases of coronary disease are due to smoking. Cigarette smoke may affect nonsmokers and very young children of smoking parents. An outline is provided for an antismoking campaign, the major points of which are: educating the public by advertising, and protecting the nonsmoker by legal provisions such as prohibiting smoking in public places, prohibiting the sale of cigarettes to persons under 16, and prohibiting the sale of cigarettes in machines.

See also, **82-0737, 82-0837**

NEOPLASTIC DISEASES

82-0658. Adelstein, A. M. **Life-Style in Occupational Cancer.** *Journal of Toxicology and Environmental Health* 6(5/6): 953-962, 1980.

An analysis of population data in Britain found that standardized mortality ratios (SMRs) for lung cancer were correlated with the presence of the smoking habit. Among 25 occupations measured for whether or not individuals smoked, lung cancer SMR showed a high correlation coefficient with the proportional current smoking ratio, the latter described as the ratio of the observed number currently smoking cigarettes to the number expected based on age-standardized proportions. The relationship was weaker when smoking habits were measured for quantity smoked, since the measure varied little among occupations. The correlation was also smaller when lung cancer mortality rates were standardized for social class. General cancer mortality data were also examined for the effects of factors such as occupation, social class, and area.

82-0659. Alderson, M. R. **Epidemiology.** In: Duncan, W. (Editor). *Recent Results in Cancer Research. Prostate Cancer,* Volume 78, Berlin, Springer-Verlag, 1981, pp. 1-19.

No clear relationship between smoking or alcohol consumption and the risk of prostate cancer was found in a review of the present knowledge about the etiology of prostate cancer. Routine mortality and morbidity statistics for England and Wales and other selected countries were examined as well as the results of special studies. A number of major studies, which have categorized individuals by their smoking habits and followed them for many years, have established morbidity and mortality patterns. No excess prostate cancer in smokers was found. No clear pointers could be found for factors that could be avoided to achieve any marked effect on the incidence of this condition. Further research areas and the value of screening for this disease are also discussed.

82-0660. Anderson, M. W.; Boroujerdi, M.; Wilson, A.G.E. **Inhibition *In Vivo* of the Formation of Adducts Between Metabolites of Benzo(*a*)pyrene and DNA by Butylated Hydroxyanisole.** *Cancer Research* 41(11): 4309-4315, November 1981.

Butylated hydroxyanisole (BHA) appears to inhibit benzo(*a*)pyrene (BaP)-induced pulmonary adenoma formation by inhibiting the amount of the benzo(*a*)pyrene diol-epoxides (BPDE): DNA adducts formed in the lung. Radioactivity was detected in the DNA of both lung and liver following oral administration of a carcinogenic dose of (^3H)BaP to A/HeJ mice. Analysis by high pressure chromatography showed that the major adduct in both tissues cochromatographed with the (±)-7β,8α-dihydroxy-9α,10α-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene (BPDEI): deoxyguanosine adduct. The 7β,8α-dihydroxy-9β,10β-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene (BPDEII): deoxyguanosine adduct was 10 to 15 percent of the BPDEI adduct in both lung and liver. Another adduct, possibly derived from BaP phenol(s), was also detected in lung tissue and was 10 to 20 percent of the BPDEI adduct. Treatment of animals with BHA decreased the amount of the BPDEI adduct in the lung and the liver approximately 55 and 75 percent, respectively. The decrease in the amount of this adduct in the lung appears to correlate with the inhibition of pulmonary adenoma formation. (Auth. Abs. Mod.)

82-0661. Beaumont, J. J.; Weiss, N. S. **Lung Cancer Among Welders.** *Journal of Occupational Medicine* 23(12): 839-844, December 1981.

Deaths from lung cancer among 3,247 welders in western Washington during the period 1950 through 1976 were identified. Relative to those among men of comparable age and race in the population as a whole, lung cancer mortality rates among the welders were elevated by 32 percent ($p=0.06$). When the analysis was restricted to the period beginning 20 years after first employment, the excess was 74 percent ($p<0.001$). An excess was also found when welders were compared with nonwelders in the same union: the attributable risk was 23.1 per 100,000 per year. A review of 11 published studies showed that most demonstrate an excess risk, with 6 of the 11 showing an excess in the range of 30 to 50 percent. In addition to the work-related hazards, there is evidence from several studies that welders may smoke cigarettes more than other occupational groups and the nation as a whole. (Auth. Abs. Mod.)

82-0662. Benarde, M. A.; Weiss, W. **Coffee Consumption and Pancreatic Cancer: Temporal and Spatial Correlation.** *British Medical Journal* 284(6313): 400-402, February 6, 1982.

In examining data on the relationship between coffee consumption and pancreatic cancer, it was found that cigarette smoking is much more uniform and significant as an etiological factor than coffee. The temporal trends involving cigarette consumption and pancreatic cancer mortality are consistent by race and sex. The etiology of pancreatic cancer is multifactorial, however, and occupational factors, as well as coffee, may confound the smoking—pancreatic cancer association.

82-0663. Brittebo, E. B.; Tjalve, H. **Formation of Tissue-Bound N'-Nitrosonornicotine Metabolites by the Target Tissues of Sprague-Dawley and Fisher Rats.** *Carcinogenesis* 2(10): 959-963, 1981.

The organ specificity exhibited by the N-nitrosamines may be due to an ability of the sensitive tissues to metabolize the compounds to their ultimate carcinogenic forms. N'-nitrosonornicotine (NNN) induces tumors preferentially in the nasal cavity and the esophagus in Sprague-Dawley and Fisher rats. The distribution and metabolism of N'-(^{14}C)nitrosonornicotine ((^{14}C)NNN) were studied in these rats using whole-body autoradiography. After intravenous injections of (^{14}C)NNN, a localization of tissue-bound metabolites was found in the mucosa of the nasal cavity and esophagus. These tissues were also found to have a capacity to form tissue-bound metabolites from NNN in vitro. These data suggest that in situ metabolic activation of NNN plays an important role in the pathogenesis of the NNN-induced tumors of the nasal cavity and the esophagus in the rat. (Auth. Abs. Mod.)

82-0664. Chameaud, J.; Perrrud, R.; Chretien, J.; Masse, R.; LaFuma, J. **Combined Effects of Inhalation of Radon Daughter Products and Tobacco Smoke.** In: Sanders, C. L. (Editor). *Pulmonary Toxicology of Respirable Particles* , 19th Annual Hanford Life Sciences Symposium, Richland, Washington, October 22, 1979, DOE Symposium Series No. 53, 1980, pp. 551-557.

The cocarcinogenic effect of tobacco smoke on rats exposed to radon products was investigated. In one experiment, 100 rats were exposed to 4,000-WLM (working level month) cumulated dose of radon daughter products. Fifty animals were then administered tobacco smoke by inhalation in a fume box during 5 months (total of 352 hours). In the group inhaling radon only, 17 cancers appeared; in the radon-tobacco group, 32 cancers were observed, many of them larger and more invasive than those seen in animals exposed to radon only. Under the same conditions tobacco smoke was inhaled by rats previously exposed to lower doses of radon daughter products (two groups of 30 rats each, at 500 and 100 WLM, respectively). Again the number of cancers observed was greater than the number of cancers expected if the rats had inhaled radon only. The carcinogenic and potentiating action of tobacco smoke was clearly demonstrated. (Auth. Abs. Mod.)

82-0665. Clarke, E. A.; Morgan, R. W.; Newman, A. M. **Smoking as a Risk Factor in Cancer of the Cervix: Additional Evidence From a Case-Control Study.** *American Journal of Epidemiology* 115(1): 59-66, January 1982.

Results of a case-control study in Toronto, Canada, demonstrated a twofold risk of invasive squamous cell carcinoma of the cervix among current smokers relative to women who had never smoked. Eligible cases were defined as women aged 20-69 years who were residents of York County and were admitted to Princess Margaret Hospital between October 1, 1973 and September 30, 1976 with newly diagnosed, pathologically confirmed, invasive squamous cell carcinoma of the cervix. Information on social and economic factors was collected by personal interview of each case and control. The analysis was limited to women who had complete smoking histories and for whom "age at first intercourse" was available. The information obtained appears to provide evidence that smoking is a risk factor in carcinoma of the cervix, an effect which was not diminished by simultaneously adjusting for age, education, and indices of sexual behavior. This association was further supported by the observations that ex-smokers were at a lower risk than current smokers (1.7 vs. 2.3), and that the risk increased with the amount of cigarettes smoked (2.2 for less than half a pack per day to 2.9 for more than one pack per day). (Auth. Abs. Mod.)

82-0666. Dalbey, W. E.; Nettesheim, P.; Griesemer, R.; Caton, J. E.; Guerin, M. R. **Lifetime Exposures of Rats to Cigarette Tobacco Smoke.** In: Sanders, C. L. (Editor). *Pulmonary Toxicology of Respirable Particles*, 19th Annual Hanford Life Sciences Symposium, Richland, Washington, October 22, 1979, DOE Symposium Series No. 53, 1980, pp. 522-535.

Specific pathogen-free Fischer-344 rats were exposed by inhalation to cigarette tobacco smoke in a regimen considered to result in a maximal tolerated dose. Total pulmonary deposition of smoke particulates from a single cigarette was 0.25 mg in young rats. Rats were exposed to smoke from seven cigarettes per day for up to 2.5 years at which time 30 percent of the exposed rats remained alive. Mortality of smoke-exposed animals during exposure was not different from that of untreated and sham-exposed controls. Of the exposed animals, 9 percent had tumors in the respiratory tract (one nasal adenocarcinoma, one nasal squamous-cell carcinoma, and in the lung, five adenomas, two alveologenic carcinomas, and one squamous carcinoma in 7 out of 80 animals) as opposed to only 1 percent tumor incidence among the control animals (i.e., a single alveologenic carcinoma in 93 animals). Smoke exposure did not change the total percent of animals with at least one tumor at some site in the body. However, relative to controls, exposed rats had significantly fewer tumors in the hypophysis, hematopoietic-lymphoid system, uterus, and ovary, and an increased number of tumors in the dermis in addition to the respiratory tract. Several nonneoplastic changes were observed throughout the respiratory tract. Hyperplastic and metaplastic areas were seen in the epithelium of the nasal turbinates, larynx, and trachea of exposed animals at death. Lungs of exposed rats contained areas of focal alveolitis; these areas consisted of accumulations of pigmented macrophages, epithelial hyperplasia, fibrosis, and disruption of alveolar structure. (Auth. Abs.)

82-0667. Doll, R. **Avoidable Cancer: Attribution of Risk.** *South African Cancer Bulletin* 25(3): 125-146, July-September 1981.

The causes of human cancer discussed are divided into 11 categories: smoking, alcohol, diet, food additives, sexual activity, infection, occupation, pollution, industrial products, medical practice, and geophysical factors. Tobacco accounts for a substantial proportion of avoidable cancer cases. The principal impact of eliminating smoking would be on lung cancer, which occurs 10 times more often in smokers than nonsmokers. An appreciable effect would also be produced on incidence of cancer of the mouth, pharynx, larynx, esophagus, bladder, kidney, and pancreas. Cancer risks from the synergistic effects of tobacco, such as with ionizing radiation, asbestos, air pollution, and alcohol consumption, would also be reduced. Results of studies indicate that in the absence of smoking, total mortality from all cancers would be reduced by about 30 percent.

82-0668. Doll, R.; Peto, R. **The Causes of Cancer.** New York, Oxford University Press, 1981, 121 pp.

Evidence that the various common types of cancer are largely avoidable diseases is reviewed. Lifestyle and other environmental carcinogenic factors are divided into a dozen categories, and for each category the evidence relating those particular factors to cancer onset rates is summarized. Where possible, an estimate is made of the percentage of current U.S. cancer mortality that might have been caused or avoided by that category of factors. By far the largest reliably known portion is the 30 percent of current U.S. cancer deaths due to tobacco, although it is possible that some nutritional factor(s) may eventually be found to be of comparable importance. The percentage of deaths due to cigarettes is still increasing, and must be expected to continue to increase because of the delayed effects smoking. In (and before) middle age, where biases are smaller, there appear to be a few real increases and a few real decreases in mortality from some particular types of cancer, but there is no evidence of any generalized increase other than that attributable to smoking. Moderate increases or decreases caused by some new agent(s) or habit(s) might of course be overlooked in such large-scale analyses. But, such analyses do suggest that, apart from cancer of the respiratory tract, the types of cancer that are currently common are not peculiarly modern diseases and are likely to depend chiefly on some long-established factor(s). Trends in mortality and in onset rates for many separate types of cancer are studied in detail in appendices. (Auth. Abs. Mod.) See also 81-1414.

82-0669. Farber, E. **Chemical Carcinogenesis and Smoking.** *Canadian Journal of Public Health* 72(6): 381-384, November-December 1981.

The "state of the art" in chemical carcinogenesis and its relationship to cancers associated with smoking is reviewed. Carcinogenesis is a multistep process with both irreversible and reversible steps starting with initiation which generates altered cells. Initiation is a two-part process: the induction of chemical lesions in DNA and in other cellular macromolecules, and their fixation by a round of cell proliferation. Metabolic activation of most carcinogens is a prerequisite for interaction with DNA and other cellular components. The epithelium and other cells of the bronchus and trachea are capable of activating polycyclic aromatic hydrocarbons to active metabolites. It is believed that the interaction with DNA may be among the most important steps. Repair processes exist in many cells but genetic defects may lead to an increase in sensitivity to some carcinogenic influences. Cell proliferation in initiation is crucial; evidence indicates that initiated cells have to be stimulated to undergo focal hyperplasia. This process is called promotion, which is the selective stimulation of initiated cells to grow, relative to the surrounding cells. The possible mechanisms of promotion are largely unknown for the respiratory tract but at least four have been suggested: (a) differential inhibition, (b) differential stimulation, (c) differential necrosis with regeneration, and (4) differential recovery. The focal proliferations show at least two options: (a) regression or maturation to normal appearing tissue, or (b) persistence. It is believed that an important component of cancer development with smoking is the promoting effect of components in smoke.

82-0670. Garfinkel, L. **"Nicht vom Eigentlichen Problem Ablenken." ["Let Us Not Be Diverted From the Real Problem."]** *Munchener Medizinische Wochenschrift* 123(40): 1483-1484, October 2, 1981, German.

The problem of the effect of passive smoking on lung cancer development is discussed. It is stated that the effect of passive smoking cannot be evaluated simply according to the smoking habits of the spouse of a nonsmoker. Lung cancer risk, alleged to be twice as high in passive smokers as in nonsmokers not exposed to the smoke of their partner, is hardly conceivable because of the absence of cell modifications in the tracheobronchial tract of passive smokers. Thus, the problem of passive smoking is believed to be insignificant and the presently available evidence does not support the conclusion that passive smoking presents even a small risk. Results of a survey sponsored by the American Cancer Society, covering 176,000 nonsmoking women and their husbands, showed that differences in mortality rate are not statistically significant between the groups with light- or heavy-smoking husbands, and the group with nonsmoking husbands. Matched groups analysis showed no increased risk of lung cancer and no dose—effect relationship in women with smoking husbands. However, a definite judgment about passive smoking effect is withheld pending further research. It is emphasized that passive smoking should not divert attention from the real problem of smoking.

82-0671. Gibel, W. **Probleme des Tabakschadens aus Onkologischer Sicht. [Oncologic Viewpoint of Damage From Smoking.]** *Das Deutsche Gesundheitswesen Zeitschrift fuer Klinische Medizin* 36(30): 1241-1243, July 1981, German.

International and East German statistics show a correlation between smoking and the incidence of cancers, particularly cancers of the bronchi and lungs. In 1977, the East German National Cancer Register recorded 6,500 new cases of malignant tumors in the bronchi and lungs of men and 900 such cases in women. The morbidity rate of these tumors is shown to have increased continuously since 1956. This fact is correlated with a parallel increase in cigarette consumption of 53 percent in the 1960 to 1978 period. This consumption increase is attributed to intensification of smoking, starting smoking at an earlier age, and smoking by an increasing number of women. Presently, in East Germany, 53 percent of men and 17 percent of women are smokers, but the number of female smokers is increasing faster than that of male smokers. Smoking prevention measures in East Germany are directed toward education of children and juveniles, prevention of smoking in industrial workshops and offices, and warning patients against smoking or helping smokers to change their habit.

82-0672. Glashan, R. W.; Cartwright, R. A. **Occupational Bladder Cancer and Cigarette Smoking in West Yorkshire.** *British Journal of Urology* 53(6): 602-604, December 1981.

Cigarette smoking was found to be associated with an increased risk of bladder cancer in an occupational study of chemical industry workers, leather workers, hairdressers, printers, and dye users (744 males, 247 females). Bladder cancer patients were interviewed for information on cigarette smoking habits, as well as coffee and alcohol drinking habits, saccharin use, past medical history, past drug treatment, occupational history, hobbies, and family history. An increased bladder cancer risk was associated with cigarette smoking for both sexes, with a slightly greater risk in males. The results were statistically significant. The study data also suggested that dye manufacturing process workers who smoked had an increased bladder cancer risk (3.5) while the nonsmokers had almost no risk. Significant increased risk was also shown for printers who smoked, although the numbers were too small to indicate the exact influence of smoking. Results for other occupations were not significant.

82-0673. Grufferman, S.; Wang, H. H.; DeLong, E. R.; Kimm, S.Y.S.; Delzell, E. S.; Falletta, J. M. **Environmental Factors in the Etiology of Rhabdomyosarcoma in Childhood.** *Journal of the National Cancer Institute* 68(1): 107-113, January 1982.

In a case—control study of childhood rhabdomyosarcoma (RMS), families of 33 cases and 99 controls were interviewed. A number of miscellaneous potential risk factors were evaluated and are summarized in tabular form. Fathers who were ever-smokers (relative to those who were never-smokers) conferred a relative risk (RR) of 3.9 ($p=0.003$). This finding was in contrast to the findings of an RR of 0.8 for children of mothers who were ever-smokers and of 1.0 for children of mothers who smoked cigarettes during their pregnancy. Cases had had fewer immunizations than controls, particularly smallpox vaccination ($RR=0.2$; $p=0.001$), and conversely had more preventable infections. An RR of 3.2 ($p=0.03$) was found associated with exposure to chemicals and an RR of 3.7 ($p=0.004$) was found associated with diets that included organ meats. Mothers of cases were more likely to be over age 30 at subject's birth, to have used antibiotics preceding or during pregnancy, and to have had an overdue and/or assisted delivery. Other findings suggest that low socioeconomic status is associated with an increased risk of RMS. In particular, it is believed that the association with father's smoking deserves further evaluation. The aggregate findings imply that environmental factors may play an important role in the etiology of childhood RMS. (Auth. Abs. Mod.)

82-0674. Hirayama, T. **Was spricht fuer, was gegen die Schaedlichkeit des Passivrauchens? [Arguments Pro and Con on Passive Smoking Hazard.]** *Munchener Medizinische Wochenschrift* 123(40): 1480-1483, October 2, 1981, German.

Correlation between lung carcinoma incidence in nonsmoking wives and smoking of their husbands is discussed in an interview with the head of the Epidemiological Division of the National Institute for Cancer Research in Tokyo, Japan. A positive correlation was found in the author's recently published study (*British Medical Journal* 282: 183-185, January 17, 1981), in agreement with the American and Greek studies on the same subject. However, the author found this correlation to be highly significant statistically, in contrast with the nonsignificant correlation in the U.S. study by L. Garfinkel (*Journal of the National Cancer Institute* 66(6): 1061-1066, June 1981). The different conclusions of the two studies are explained in terms of different methodology and other variables. It is calculated that a nonsmoking wife of a smoking husband inhales daily smoke equal to an average of 5.8 cigarettes. Over a period of years, the constant carcinogenic stimulation by passively inhaled smoke could explain the prevalence of adenocarcinoma in passively smoking wives. Also, chronic bronchitis and asthma are positively, although not significantly, associated with passive smoking. Breast carcinoma is of marginal significance. See also 81-1088 and 81-1418.

82–0675. Hoffmann, D.; Adams, J. D. **Carcinogenic Tobacco-Specific *N*-Nitrosamines in Snuff and in the Saliva of Snuff Dippers.** *Cancer Research* 41(11): 4305-4308, November 1981.

Varying levels of tobacco-specific *N*-nitrosamines (TSNA) were found in a selection of snuff products as well as in the saliva of snuff dippers. Human data indicate an increased risk for cancer of the oral cavity for snuff dippers. Popular snuff products from the United States, Germany, Sweden, and Denmark were analyzed for tobacco-specific *N*-nitrosamines (TSNA). These compounds are formed during tobacco processing from nicotine, nornicotine, and anatabine and represent the only known carcinogens in snuff. *N'*-Nitrosonornicotine, a moderately active carcinogen, ranged in dry snuff from 3.5 to 77 ppm; 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, a relatively strong carcinogen, ranged from 0.6 to 7.0 ppm; and *N'*-nitrosoanatabine, thus far not bioassayed, ranged from 0.8 to 44 ppm. The concentrations of TSNA in a given snuff product can vary widely, and aging in the open air can lead to an increase in TSNA. Analysis of the saliva of snuff dippers revealed that these nitrosamines are extracted from the tobacco plug during snuff dipping and that their concentrations in saliva can vary widely between users. Efforts should be made to reduce the TSNA in snuff by modifications of the production process and by wrapping individual snuff portions in airtight packets. (Auth. Abs. Mod.)

82–0676. Keast, D.; Ayre, D. J.; Papadimitriou, J. M. **A Survey of Pathological Changes Associated With Long-Term High Tar Tobacco Smoke Exposure in a Murine Model.** *Journal of Pathology* 135(4): 249-257, December 1981.

A variety of pathological changes were found among Balb/c mice exposed to a daily equivalent of 30 high-tar filtered cigarettes for periods of up to 95 weeks. Detailed surveys of the gross and histopathological data indicated that induction or production of significant numbers of malignant tumors of several types occurred in the animals exposed to tobacco smoke. There were also significant histopathological changes which consisted mainly of interstitial pneumonia and focal low-grade emphysema. These features contributed significantly to the reduction in life span of the test animals in which the main causes of death were malignancies and inflammatory diseases of the lungs. (Auth. Abs. Mod.)

82–0677. Kobara, Y.; Sato, A.; Matsui, Y.; Imai, H.; Honda, K.; Oshima, S. **Genhatsusei Haigan no Nyuinji Rinshozo to Soshikigata. [Relationship Between Clinical Findings and Histology of Primary Lung Cancer.]** *Bulletin of the Chest Disease Research Institute, Kyoto University,* 12(1/2): 17-27, March 1979, Japanese.

Clinical, laboratory, and personal data were collected from 578 male and 150 female primary lung cancer patients admitted to the Chest Disease Research Institute of Kyoto University Hospital between 1966 and 1977. All patients were classified into four categories: those suffering from epidermoid carcinoma (290 males and 39 females), those suffering from adenocarcinoma (146 males and 76 females), those suffering from undifferentiated cell carcinoma (137 males and 27 females), and those suffering from alveolar cell carcinoma (5 males and 8 females). The data show that 342 (59.2 percent) of the men and 64 (45.1 percent) of the women had a past record of smoking. Using the Brinkman Index (BI) of the amount of smoking, it was found that the higher the BI, the higher the frequency of epidermoid carcinoma. Of those suffering from epidermoid carcinoma, 76.3 percent had a BI rating higher than 600. Also, among those suffering from adenocarcinoma or undifferentiated cell carcinoma, an overwhelming proportion of them had a BI rating higher than 600. When the patients were divided into two groups, the heavy smoker group with BIs higher than 400, and the light smoker group with BIs lower than 400, there was no statistically significant difference observed between the epidermoid carcinoma patients and the undifferentiated carcinoma patients. However, there was a statistically significant difference between the two above-mentioned types of lung cancer patients and the adenocarcinoma patients.

82–0678. Lehnert, G. **Krank durch Passivrauchen? [Sick From Passive Smoking?]** *Munchener Medizinische Wochenschrift* 123(40): 1485-1488, October 2, 1981, German.

The epidemiological studies by T. Hirayama (*British Medical Journal* 282: 183-185, January 17, 1981) and L. Garfinkel (*Journal of the National Cancer Institute* 66(6): 1061-1066, June 1981) are critically evaluated, together with these authors' interviews published in this issue. A serious doubt is expressed about the representativity of the random sample of population in the Japanese study, in which the estimates of the length and intensity of exposure to passive smoking are believed to be unrealistic and not credible. The finding of a high incidence of adenocarcinoma among passive smoking wives does not reflect a cause-and-effect relationship between passive smoking and lung carcinoma. Statistical analysis of the Japanese data confirms their unreliability. At most, the existence is admitted of a "low risk association" based on statistical significance of the Japanese data. However, the increased risk of passive smoking to wives of smoking men must be verified by objective tests of population subgroups. To date, no scientifically convincing proof of the increased risk of lung carcinoma could be found. See also 81-1088 and 81-1418.

82-0679. Liddell, F.D.K. **Latent Periods in Lung Cancer Mortality in Relation to Asbestos Dose and Smoking.** In: *IARC Scientific Publication No. 30, Part 2.* International Agency for Research on Cancer, 1980, pp. 661-665.

No relationship was found between smoking history, dosage of asbestos exposure, and the interval between first exposure and death from lung cancer. Among the 4,462 deaths in a cohort of 10,939 men who worked in the Quebec chrysotile mines and mills, 244 were due to lung cancer. All 244 cases were classified according to degree of exposure, length of time exposed, and smoking habit (nonsmokers, ex-smokers, light smokers, 15-25 cigarettes per day, and 26-50 cigarettes per day). The interval from first asbestos exposure to death averaged 39.6 years, and was related to age at first employment, and age at death. Though smoking was closely related to lung cancer mortality, there was no relationship between asbestos dosage or smoking and the exposure-death interval.

82-0680. Lin, R. S.; Kessler, I. I. **A Multifactorial Model for Pancreatic Cancer in Man. Epidemiologic Evidence.** *Journal of the American Medical Association* 245(2): 147-152, January 9, 1981.

A study conducted in more than 115 hospitals in 5 metropolitan areas found that smoking is one of the risk factors for pancreatic cancer. Smoking exposure histories were compared in cases and controls using matched-pair analysis. Among both sexes combined, cigarette smoking histories in terms of dose, duration, and exposure intensity were about the same in case and control subjects. The female cases, however, were somewhat more likely to have smoked 10 or more cigarettes per day than the controls (43 percent to 31 percent). Of all the risk factors surveyed, it was suggested that occupational factors, decaffeinated coffee, and habitual wine drinking may interact to enhance the pancreatic cancer risk among males; and that a combination of gynecologic factors, decaffeinated coffee, and cigarette smoking may have an additive or multiplicative effect among females. Suggested mechanisms for a possible association between pancreatic cancer and smoking included the presence of a specific pancreatic carcinogen in the smoke or cholecystokinin-pancreozymin mediated effects on the pancreas triggered by increases in blood lipids caused by smoking.

82-0681. Matthay, R. A.; Balmes, J. R. **Lung Cancer: A Persistent Challenge.** *Geriatrics* 37(2): 109-131, February 1982.

Lung carcinoma is the leading cause of death from cancer in American men and the second most frequent cancer killer of American women. The average patient is a heavy smoker over 60 years of age; fewer than 5 percent of patients are under 40. In this study, the etiology, pathology, clinical manifestations, treatment, and prognosis of the disease are reviewed. Lung neoplasm development was found to be proportional to total exposure to cigarette smoke: number of cigarettes smoked, duration of smoking years, age of initiation, depth of inhalation, and tar and nicotine levels in cigarettes smoked. Compared with nonsmokers, average smokers have approximately a nine- to tenfold increased risk and heavy smokers a 20-fold risk. Smoking is chiefly associated with the squamous cell variety and to a lesser degree with oat cell. Although adenocarcinoma is chiefly associated with environmental and occupational factors, smoking may have a synergistic role. Data indicate that within 13 years after smoking cessation carcinoma risk is comparable to that of nonsmokers.

82-0682. Miller, G. H.; Klatsky, A. L. **Alcohol and Mortality. (Letters).** *Annals of Internal Medicine* 96(1): 120-121, January 1982.

A study on alcohol and mortality (*Annals of Internal Medicine* 95(2): 139-145, August 1981) which found an apparent beneficial effect of moderate alcohol ingestion is questioned due to the introduction of perceived biases. The increase in mortality from the abstaining group would indicate that there should be twice the number of smokers in this group compared with the moderate group, especially since the abstainers had twice the number of lung cancer cases. This increase in percentage of smokers would also account for the increased mortality of the abstaining group due to cardiovascular diseases which are usually smoking-related. Klatsky replies that all alcohol consumption groups contained equal proportions of current established smokers, but abstainers included more persons who had never smoked, though these were few. That heavy drinkers had a history of greater smoking intensity might have been a factor in their excess mortality; however, the excess mortality of nondrinkers over moderate drinkers cannot be explained by associated smoking. All lung cancer deaths did occur in smokers or ex-smokers. This finding may be a weak piece of evidence supporting the smoking-drinking synergism in cancer production. See also 81-1758.

82-0683. Miller, W. J., Jr.; Cooper, R. **Rising Lung Cancer Death Rates Among Black Men: The Importance of Occupation and Social Class.** *Journal of the National Medical Association* 74(3): 253-258, 1982.

A number of possible factors, including smoking, which may account for the greater rise in cancer mortality, particularly lung cancer, among black men than among white men are discussed. Environmental factors such as socioeconomic status and occupational exposure may be important. Black males make up a disproportionate share of blue collar workers, job levels where smoking is more common than among professional, technical, and managerial personnel. At present, there is a greater prevalence of smokers and a lower quitting rate among black men than among white men. However, per capita cigarette consumption is lower among black men than white men. Cigarette smoking may be related to occupation, sex, race, and socioeconomic grouping. The cancer excess among black men may be due to an association between occupation, socioeconomic grouping, and the harmful effects of migration and racism, together with cigarette smoking. The higher prevalence of lung cancer among black men may be due to the larger percentage of black men in jobs with possibly hazardous environmental exposure rather than to different smoking habits. Efforts to address the cancer trend among blacks should consider occupational work conditions as well as smoking.

82–0684. Morrison, A. S.; Buring, J. E.; Verhoek, W. G.; Aoki, K.; Leck, I.; Ohno, Y.; Obata, K. **Coffee Drinking and Cancer of the Lower Urinary Tract.** *Journal of the National Cancer Institute* 68(1): 91-94, January 1982.

Coffee drinking showed little or no association with the incidence rate of cancer of the lower urinary tract ("bladder cancer"), in a study which included factors such as smoking. Broadly based series of cases and series of controls drawn from the general population of each area were assembled and interviewed in Boston, Massachusetts (587 cases, 528 controls), Manchester, England (541 cases, 725 controls), and Nagoya, Japan (289 cases, 586 controls). With adjustment for cigarette smoking habits, only small and irregular changes in risk were seen with increasing frequency of coffee consumption. Duration of coffee drinking showed little relation to risk of bladder cancer. Apparent associations of coffee drinking and bladder cancer may be due to incomplete control of the effect of cigarette smoking. (Auth. Abs. Mod.)

82–0685. Nettesheim, P.; Topping, D. C.; Jamasbi, R. **Host and Environmental Factors Enhancing Carcinogenesis in the Respiratory Tract.** *Annual Review of Pharmacology and Toxicology* 21: 133-163, 1981.

A variety of possible factors which may influence respiratory carcinogenesis are mentioned, including smoking, age-specific susceptibility, immune suppression, dietary vitamin A, respiratory tract infections, two-stage carcinogenesis, chrysotile asbestos, inert particles, nickel subsulfide, benzo(*e*)pyrene, and irritant gases and vapors. Lung cancer appears to have a multifactor etiology. The major interaction of agents or factors may lie within a given environmental or occupational setting. In other instances it appears to be the interaction of an occupational with a nonoccupational exposure that results in a high lung cancer risk. The most convincing example of this kind appears to be the high incidence of bronchogenic carcinoma in smoking asbestos workers. Finally, certain host factors (e.g., age, immune status), nutritional factors, or preexisting diseases may predispose individuals and render them highly susceptible to carcinogenic agents in the environment. (Auth. Abs. Mod.)

82–0686. Selikoff, I. J. **Occupational Respiratory Diseases: Asbestos-Associated Disease.** In: Last, J. M. (Editor). *Public Health and Preventive Medicine.* Eleventh Edition, New York, Appleton-Century-Crofts, 1980, pp. 568-598.

Cigarette smokers were shown to be at high risk of asbestos-related lung cancer in this study of the health hazards of asbestos exposure. In a group of 370 asbestos insulation workers followed for 5 years, no deaths from lung cancer occurred among the 87 nonsmokers in the group, although there was ample evidence of asbestos exposure. However, among the 283 workers with a history of smoking, 24 deaths from lung cancer occurred, where only 3 might be expected. Thus, in this group, a smoking asbestos worker had approximately 90 times the risk of lung cancer than a nonsmoker who did not work with asbestos. Even in the absence of cigarette smoking, asbestos exposure increases the risk of lung cancer, as well as fatal progressive pulmonary fibrosis. Nevertheless, asbestos mortality for smokers of a pack or more per day is 2.8 times as high as that of nonsmokers. One reason for the delay in confirming the significance of asbestos as a factor in lung cancer is the distribution of risks according to smoking habits. A history and overview of asbestos-caused health problems and worker compensation is also given.

82–0687. U.S. Department of Health and Human Services; U.S. Department of Labor. **Workplace Exposure to Asbestos.** U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 81-103, November 1980, 41 pp.

A booklet prepared by the National Institute for Occupational Safety and Health and the Occupational Safety and Health Administration on asbestos exposure is presented. A wide variety of topics are covered, including smoking and asbestos, asbestos nomenclature and definitions, asbestos sampling and analysis, biological effects in animals and in humans, exposure to asbestiform minerals other than commercially mined asbestos, nonoccupational exposure to commercial sources of asbestos, and dose—response relationships. Evidence on the respiratory effects of smoking and asbestos exposure concludes that smoking and asbestos can each independently increase lung cancer mortality risk. If there is exposure to both factors, the combined effect for lung cancer seems to be multiplicative instead of additive.

82–0688. Varela, A. S.; Troitino, R. P.; Saceda, P.G.-B.; Nunez-Cortes, J. M. **Tabaco y Cancer. [Tobacco and Cancer.]** *Revista Clinica Espanola* 161(3): 173-176, 1981, Spanish.

A survey was conducted of the smoking habits of 487 males over 45 years of age; 227 of whom had cancer with tumors localized in the lungs, larynx, oral cavity, lips, stomach, and bladder; 260 were used as controls. There were 224 smokers in the control group (86.5 percent) and 222 in the group with cancer (97.7 percent). It was found that both groups smoked a median of 20 cigarettes/day with the exception of the 60- to 64-year-old group, where the controls smoked an average of 19 cigarettes/day and the others 28. Both groups began smoking at about age 16 except for the 45- to 49-year-olds; the median age was 15 for those with cancer and 17 for the controls. Both groups had smoked for an average of 43 years with the exception of the 45- to 49-year-olds who averaged 31 years for those with cancer and 27 for the controls. No significant differences were found between the two groups with respect to inhalation, but the cancer group kept the cigarette between the lips longer than did the controls, indicating some relation to the greater carcinogenic effect of sidestream smoke than mainstream smoke. It was found that 62 percent of the group with cancer smoked early in the morning before breakfast, while this was true in only 38 percent of the controls. It is concluded that the age at which one begins smoking is more related to carcinogenesis than the number of cigarettes smoked or the length of time one has been smoking.

82–0689. Vutuc, Ch.; Kunze, M. **Zigaretten-Rauchkondensat-Exposition und Berufsanamnese bei weiblichen Lungenkrebspatienten. [Cigarette Smoke Condensate Exposure and Occupational History in Female Lung Cancer Patients.]** *Onkologie* 4(4): 184-187, August 1981, German.

In a retrospective case-control study on female lung cancer 130 (65 percent) of 200 cases suffered from a tumor group Kreyberg I, and 70 (35 percent) from a Kreyberg II tumor. Significantly more Kreyberg I cases (75 percent) than controls (22 percent) were smokers as well as significantly more Kreyberg I than Kreyberg II cases (29 percent). There was no difference in the proportion of ex-smokers, but the duration of nonsmoking was significantly shorter in Kreyberg I cases (4 years) than in controls (12 years) and in Kreyberg II cases (9 years). Patients with Kreyberg I (88 percent) and Kreyberg II (90 percent) tumors preferred significantly more often high-tar group III (> 24 mg/cigarette) cigarettes than controls (63 percent). Patients with a Kreyberg I tumor differ significantly from controls by longer smoking careers (40 years) and higher daily consumption of group III (18/day) and group I (18/day) cigarettes. Patients with a Kreyberg II tumor had a significant higher consumption (20/day) of group I cigarettes than controls. Female lung cancer patients (74 percent) more often than controls (64 percent) reported occupations other than housewives. There were no other significant differences in the occupational patterns between patients and controls. (Auth. Abs.)

82–0690. Ward-Booth, R. P.; Gabriel, A.; Tanner, N.S.B. **Unexpectedly Low Frequency of Lymph Node Spread in Oral Cancer in Sri Lanka. (Letter).** *Lancet* 1(8264): 165, January 16, 1982.

In Sri Lanka, mouth neoplasms account for as high as 22 percent of all cancers compared with only 2 percent in the United Kingdom. It is believed that persistent chewing of betel quid (betel leaf wrapped around areca nut, quick lime, and tobacco) accounts for the high incidence rate, since tumors tend to arise from the site where the quid is kept. During a 3-month period, 102 consecutive patients were reviewed and all had histologically proven oral squamous cell carcinoma. Eighty-three had lesions over 2 cm in diameter occurring on the tongue, lip, soft palate, or buccal mucosa; 71 lesions arose from the buccal mucosa and 20 from the tongue. Lymph node spread in Sri Lanka is compared to Western studies, and it is concluded that although Sri Lanka patients presented late with large fulminating lesions, cervical lymph node involvement seemed less common.

82–0691. Winters, T. H.; Di Franza, J. R. **Radioactivity in Cigarette Smoke. (Letter).** *New England Journal of Medicine* 306(6): 364-365, February 11, 1982.

The detrimental effects of the radioactive components of tobacco smoke have been underestimated, making it less likely that chemical carcinogens alone are responsible for tobacco-related carcinoma. Alpha emitters (polonium-210 and lead-210) in cigarette smoke result in appreciable radiation exposure to the bronchial epithelium of smokers and probably involuntary (passive) smokers. In a person smoking 1.5 packs per day, the radiation dose is the equivalent of the dose to the skin from 300 x-ray films of the chest per year. Also, it has been determined that 75 percent of the alpha activity of cigarette smoke enters the ambient air, making it available for deposit in the lungs of others. Alpha radiation, as a possible etiologic factor in tobacco-related carcinoma, merits further research.

82–0692. Young, J. L., Jr.; Percy, C. L.; Asire, A. J. (Editors). **Surveillance, Epidemiology, and End Results: Incidence and Mortality Data, 1973-77.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 57, NIH Publication No. 81-2330, June 1981, 2,004 pp.

The Surveillance, Epidemiology, and End Results (SEER) Program is a continuing project of the Biometry Branch of the National Cancer Institute begun in 1972. The goals of the SEER program are to: (1) determine periodically the incidence of cancer in selected geographic areas of the United States with respect to demographic and social characteristics of the population; (2) estimate cancer incidence for the United States on an annual basis; (3) monitor trends in incidence of specific forms of cancer with respect to geographic area and demographic and social characteristics of the population; (4) determine periodically survival experience for cancer patients diagnosed among residents of selected geographic areas of the United States; (5) monitor trends in cancer patient survival with respect to form of cancer, extent of disease, therapy, and demographic, socioeconomic, and other parameters of prognostic importance; (6) identify cancer etiologic factors by conducting special studies which disclose groups of the population at high or low cancer risks; (7) identify factors related to patient survival through special studies of referral patterns, diagnostic procedures, treatment methods, and other aspects of medical care; and (8) promote specialty training in epidemiology, biostatistics, and tumor registry methodology, operation, and management. Detailed information on the incidence of and mortality from cancer in the United States during the initial 5 years of the program (1973-1977) is statistically presented, classified according to sex, race, age, and residence of the patient; anatomic site; and histologic type of cancer.

See also, **82-0614, 82-0645, 82-0696, 82-0805**

NON—NEOPLASTIC RESPIRATORY DISEASES

82-0693. Agaj, A.; Mermolja, M.; Haxhiu, M. A.; Popovac, D. **Citoloske Promene Respiracijskog Trakta Kao Posledica Pusenja. [Cytologic Changes in Respiratory Tract in Relation to Smoking.]** *Plucne Bolesti i Tuberkuloza* 33(1): 5-7, January-March 1981, Serbo-Croatian.

A cytologic examination of sputum specimens was carried out to determine the effect of smoking on cell population. The sputum specimens were taken from 209 male and 71 female subjects, aged 40 to 60 years. There were 149 nonsmokers, 77 moderate smokers of less than 24 cigarettes/day, and 54 heavy smokers of over 25 cigarettes/day. The percentages of macrophages, columnar cells, metaplastic cells, and eosinophilic granulocytes were found to be the lowest in nonsmokers, significantly higher in moderate smokers, and highest (except for granulocytes) in heavy smokers. The higher percentage of abnormal columnar cells in smokers indicates that inflammatory changes in bronchial mucosa are much more frequent in smokers than in nonsmokers. The advent of metaplastic epithelium is the long-term effect of noxious substances. A gradual increase of atypical cells in the epithelium impairs normal function of bronchial mucosa, and hence, increases the risk of lung cancer. The increased number of metaplastic cells in sputum of heavy smokers indicates that the degree of impairment of bronchial mucosa increases with the increased number of smoked cigarettes. Hence, the risks of severe chronic obstructive lung diseases and carcinoma of the lung are increased.

82-0694. Beck, G. J.; Schachter, E. N.; Maunder, L. R.; Heyden, S.; Pratt, P. C. **Exposure to Cotton Dust and Respiratory Disease. (Letters).** *Journal of the American Medical Association* 247(2): 175-176, January 8, 1982.

The conclusion that after long-term exposure to cotton dust, chronic obstructive airway disease, particularly byssinosis, does not occur in nonsmokers and is reversible with smoking cessation (*Journal of the American Medical Association* 244(16): 1797-1798, October 17, 1980) is refuted. It is argued that data from a prospective study of 383 cotton textile workers aged 45 years and older showed a prevalence of byssinosis of 12 percent among nonsmoking cotton textile workers and 1 percent in a group of control subjects at followup. In addition, chronic bronchitis was nearly six times more prevalent in the cotton textile workers. Even among retired cotton textile workers, loss of lung function continued to be larger than expected, suggesting that the lung dysfunction is irreversible. Other areas of question in the original article include the interpretation of one study cited, the applicability of another study, and the relevancy of other articles referred to. In defense of their article, Heyden and Pratt quote extensively from an earlier study (*Journal of Occupational Medicine* 15(3): 212-221, March 1973) which states that "Within primary work areas, a 50 to 100 percent increase in byssinosis prevalence was generally observed among smokers over nonsmokers...." See also 81-1459 and 73-0485.

82-0695. Coggins, C.R.E. **Changes in the Respiratory Physiology of Rats Exposed to Different Phases of Cigarette Smoke.** In: Sanders, C. L. (Editor). *Pulmonary Toxicology of Respirable Particles*, 19th Annual Hanford Life Sciences Symposium, Richland, Washington, October 22, 1979, DOE Symposium Series No. 53, 1980, pp. 420-430.

Cigarette smoke consists of particulate matter, defined as that fraction retained by a glass-fiber filter disk, and vapor phase, the fraction which passes through such a filter. Knowledge of the comparative effects of the two phases on respiratory physiology would be helpful in identifying the specific components of smoke responsible for inducing changes. The work described here was carried out to obtain such information, by using in vivo and in vitro techniques. Rats were exposed in Battelle Mk III inhalation machines to smoke from commercially available flue-cured cigarettes. One group received a 4 percent dilution of whole smoke in air, and a second group was exposed to the same concentration of smoke which had been passed through a glass-fiber filter. A third group of animals was sham-exposed. Animals were exposed for 1 hour/day, 5 days/week for a total of 4, 8, or 12 weeks. In vivo respiratory physiology measurements were made just before sacrifice; quasi-static pressure—volume hysteresis loops were made on excised lungs. Results obtained from smoke-exposed animals showed pronounced increases over controls in terms of lung compliance and residual volume. Similar effects were observed in animals exposed to whole smoke and in those exposed to vapor phase alone, which implies independence of the changes from inhaled particulates. Work is continuing on attempts to identify which smoke component(s) are responsible for the changes. (Auth. Abs.)

82–0696. Goerisch, I.; Weilepp, M.; Goerisch, V. **Rauchgewohnheiten bei Patienten mit Kehlkopferkrankungen. [Smoking Habits of the Patients With Diseases of the Larynx.]** *Zeitschrift fuer Erkrankungen der Atmungsorgane* 157(2): 212-218, 1981, German.

A total of 616 patients of a Leipzig, East Germany, clinic were interviewed and the results were compared with the data from literature. Among the interviewed, 510 were men and 106 were women, with either a benign disease of the larynx, mostly chronic laryngitis (347 patients), or a malignant tumor of the larynx (269 patients). There were 76.4 percent smokers among the former patients versus 90.7 percent among the latter. Most of the 82.6 percent of all 616 patients had been smoking over 100,000 g. tobacco, preferably as filter cigarettes, over a period of 10 years or longer, and most of the smokers inhaled. The percentages of nonsmoking female patients with both benign and malignant diseases of the larynx were significantly higher than the respective percentages of nonsmoking male patients. Only among male patients, the percentage of nonsmoking patients with malignant tumors was lower than the corresponding percentage of those with benign disease. The finding that patients of both sexes with a malignant tumor had smoked more tobacco over a longer period than those with a benign disease of the larynx was shown to be highly significant.

82–0697. Guyatt, A. R.; Holmes, M. A.; Cumming, G. **Can Carbon Monoxide Be Absorbed From the Upper Respiratory Tract in Man?** *European Journal of Respiratory Diseases* 62(6): 383-390, December 1981.

To investigate the possible absorption of carbon monoxide (CO) into the blood from tobacco smoke confined to the mouth and upper airways, an indirect study was made on 12 normal subjects of the recovery of CO from a bolus administered at various levels during a preceding inspiration. No measurable CO uptake was observed unless the bolus was inhaled early enough in inspiration to reach the alveolar region of the lung. However, some movement of a tracer gas, argon, was seen from the upper airways during breath-holding so that some CO might escape into the lungs this way. No detectable CO uptake occurred in one subject who inspired the bolus into his nasal cavities. It is concluded that the elevated carboxyhemoglobin levels seen in smokers can only occur when smoke is inhaled into the lung, since no appreciable uptake can occur in the upper airways. (Auth. Abs.)

82–0698. Higgins, M. W.; Keller, J. B.; Becker, M.; Howatt, W.; Landis, J. R.; Rotman, H.; Weg, J. G.; Higgins, I. **An Index of Risk for Obstructive Airways Disease.** *American Review of Respiratory Disease* 125(2): 144-151, February 1982.

Smoking was found to be one of the risk factors for obstructive airways disease (OAD) among a study population. Predictors of OAD were identified, and models for estimating risk of developing OAD were derived for the adult population of Tecumseh, Michigan. Men and women 16 to 64 years of age when first studied were reexamined after an average interval of 15 years. Incidence rates were significantly higher in cigarette smokers, especially those who continued to smoke. Other risk factors included: sex; age; a low degree of lung function initially; a physician's diagnosis of chronic bronchitis or asthma; a history of cough, wheeze, shortness of breath, frequent upper or lower respiratory tract infections, bronchitis or pneumonia; leanness; and familial chronic bronchitis. The excess risk of OAD associated with cigarette smoking and reduced lung function and the benefits of stopping smoking were clearly apparent. For example, the risk of developing OAD in the next 15 years is about 1 in 200 for a 45-year-old male nonsmoker whose forced expiratory flow at 50 percent of vital capacity ($Vmax_{50}$) equals 100 percent of predicted, if he doesn't take up smoking. The risk for a man of the same age who smoked 40 cigarettes a day and whose $Vmax_{50}$ equals 80 percent of predicted is 1 in 5 or 6 if he doesn't cut down on his smoking and about 1 in 15 if he stops smoking. (Auth. Abs. Mod.)

82–0699. Irwin, R. S.; Erickson, A. D.; Pratter, M. R.; Corrao, W. M.; Garrity, F. L.; Myers, J. R.; Kaemmerlen, J. T. **Prediction of Tracheobronchial Colonization in Current Cigarette Smokers With Chronic Obstructive Bronchitis.** *Journal of Infectious Diseases* 145(2): 234-241, February 1982.

Results of a study of subjects with chronic obstructive bronchitis suggests that cigarette smoking interferes with the clearance and/or detoxification of bacteria from the tracheobronchial tree. To help in clarifying the conflicting data on the role of tracheobronchial microflora in chronic bronchitis, the tracheobronchial microflora of a homogeneous group of clinically stable patients with chronic obstructive bronchitis was characterized by transtracheal aspiration. Their mean percentage ratio of forced expiratory volume in 1 second to the forced vital capacity was 45 percent. The results were: (1) a bacterial tracheobronchial microflora was present in only 50 percent of the patients, (2) viridans streptococci were the bacteria most frequently isolated, and (3) the presence or absence of a tracheobronchial microflora was significantly associated with the amount of present cigarette smoking. The fact that patients who smoked less than one pack per day were most likely to have a sterile tracheobronchial tree ($p = 0.015$) implied that there was a critical amount of cigarette smoke that impaired the clearance and/or detoxification of bacteria from the tracheobronchial tree and that it must have been persistently present. (Auth. Abs. Mod.)

82–0700. Jenkins, R. A.; Gayle, T. M. **An Instrumental Inhaled-Smoke Dosimeter for the Quantitative Characterization of Aerosol Exposures.** In: Sanders, C. L. (Editor). *Pulmonary Toxicology of Respirable Particles* , 19th Annual Hanford Life Sciences Symposium, Richland, Washington, October 22, 1979, DOE Symposium Series No. 53, 1980, pp. 68-86.

A tobacco smoke inhalation bioassay model which involves passive, intermittent exposure of male beagle dogs to whole cigarette smoke is described. Included in the design is an instrumentation package which uses a smoke-concentration sensor and a laminar-flow meter. The light-emitting-diode—phototransistor aerosol sensor is mounted at the entrance to a tracheal cannula and relates backscattered infrared light to smoke concentration in the cannula. Response of the sensor is linear over a wide range of aerosol concentrations (10 μg liter to 100 mg/liter). Inhaled flow is measured at the inspiration valve of the smoke-exposure system. This configuration permits visualization and quantitation of animal breathing patterns during smoke exposure. Amplified signals are multiplied electronically so that smoke registers as being inhaled only when it is drawn into the cannula. The integral of the product of smoke concentration and instantaneous flow rate is directly related to the inhaled smoke dose. Evaluation of the system under a wide range of inspiratory flows and simulated breathing patterns indicates that the relative standard deviation of the integrated response per unit of particulate matter is about 4 percent for the smoke of a standard experimental-blend cigarette. Observations made during field tests with beagle dogs indicate that animals seldom overrange the linear range of the flow system and that fogging of the sensor by exhaled breath is not a significant problem. Animals were observed to inhale 23 to 100 percent of the tobacco smoke made available for inhalation, with most animals averaging 90 percent. (Auth. Abs. Mod.)

82–0701. Jones, N. L. **The Pathophysiological Consequences of Smoking on the Respiratory System.** *Canadian Journal of Public Health* 72(6): 388-390, November-December 1981.

The ways cigarette smoke harms the lungs depends on how many particles are inhaled, where they land, how long they stay, their chemical properties, concentration, deposition, clearance, and their pathogenic effects. Where particles are deposited depends on their size, shape, and density: smaller particles are deposited deeper and may have different effects. Solubility and acidity influence the extent of the damage. The particles also may provoke an antigen reaction in the lungs. Clearance of the particles from the lungs is slower in smokers, possibly due to poor ciliary function. Surface mucus-secreting cells are more abundant in smokers, and the larger, deeper mucosa-producing glands occupy two to three times the normal space in the walls of the airways. Some constituents of cigarette smoke are capable of killing the macrophage in very small airways lacking cilia, thus releasing the enzymes into the alveoli. There is a risk the alveolar wall will be digested, producing emphysema. The downhill course of patients with chronic obstructive diseases (emphysema, asthma, chronic bronchitis) is more rapid in those who smoke more, but the decline can be lessened with smoking cessation. Studies in identical twins suggest that the effects of smoking are enhanced or inhibited by genetic predisposition.

82–0702. Kado, M. **Kokyuki no Kansen Bogyo Kiko ni Kansuru Kenkyu. Dai-2 Hen: Kokyuki Kansen Bogyo Inshi Toshite no Lysozyme ni Kansuru Kenkyu. [Studies on the Defense Mechanisms in the Respiratory System. Report II. Studies on Lysozyme as a Defense Factor Against Infection.]** *Bulletin of the Chest Disease Research Institute, Kyoto University* 13(1/2): 32-43, March 1980, Japanese.

An investigation of the distribution of lysozyme injected in the internal organs of rats suggested that the lysozyme level of the respiratory system rose temporarily following cigarette smoke inhalation. The lysozyme distribution was studied in the lung, kidney, spleen, and plasma. An experimental apparatus was used to allow rats to inhale cigarette smoke for 5 minutes, 15 minutes, 1 hour, or 2 hours. It was found that the amount of lysozyme increased significantly after the inhalation of cigarette smoke.

82–0703. Kalliomaki, P.-L.; Rahkonen, E.; Vaaranen, V.; Kalliomaki, K.; Aittoniemi, K. **Lung-Retained Contaminants, Urinary Chromium and Nickel Among Stainless Steel Welders.** *International Archives of Occupational and Environmental Health* 49(1): 67-75, 1981.

The magnetic method of measuring the amount of lung-retained contaminants as well as urinary chromium and nickel determinations was performed among 83 stainless steel (SS) welders who have used matural metal arc (MMA) and tungsten inert gas (TIG) welding techniques. The welders were divided into four groups according to the time percentage used for MMA welding. Only exposure to MMA/SS welding fumes resulted in clearly elevated chromium concentrations in the urine, the correlation coefficient between the values of urinary chromium and MMA/SS percentage being very significant ($p < 0.001$). Among the smokers the urinary chromium values were increased ($p < 0.05$) perhaps owing to contaminated cigarettes. In many workplaces the urinary chromium values of several welders exceeded the value of 30 μg/l which is the recommended reference value in Finland. Owing to the solubility properties of nickel compounds in SS welding fumes urinary nickel concentrations were only slightly elevated among MMA/SS welders, and therefore, the urinary nickel determinations do not reflect the level of exposure to nickel compounds. For the smokers, urinary nickel values were also increased ($p < 0.01$). The measured average remanent magnetic field of the chest area correlated well ($p < 0.01$) with the use of the MMA technique. A very significant correlation ($p < 0.001$) existed between the average remanent magnetic fields of the chest and the urinary chromium values of MMA/SS welders. (Auth. Abs. Mod.)

82–0704. Knapp, R. J. **The Isolation of an Endogeneous Nicotine-Like Substance and the Characterization of Its Binding to Brain.** Doctoral Dissertation, University of Rochester, New York, University Microfilms International 81-22379, 1980, 132 pp.

The site at which 1-methylhistamine competes with nicotine for binding does not appear to have nicotinic cholinergic properties. The research presented supports the hypothesis that brain tissue possesses a noncholinergic nicotine receptor and suggests that 1-methylhistamine may serve as its endogeneous ligand. The isolation of nicotine-like substances from the brain, using the inhibition of ^{3}H-nicotine binding to glass fiber filters as an assay for the presence of nicotine-like activity in chromatographic fractions, is described. While isolation was not entirely successful it did indicate the importance of nicotine-like properties of 1-methylhistamine. Membrane-binding studies using brain tissue obtained from rats and ^{3}H-nicotine showed the presence of two high affinity stereoselective sites for this drug. One of these sites, the higher affinity site ($Kd=3.18 \times 10^{-10}M$), was unaffected by sodium concentrations between 1 and 100mM. 1-Methylhistamine and mecamylamine could compete with nicotine at this higher affinity site. Neither 1-methylhistamine or mecamylamine competed for the lower affinity site ($Kd=1 \times 10^{-8}M$) in a sodium chloride concentration of 100mM. The lower affinity site was found to be dependent on the sodium concentration, but potassium concentrations between 10 and 100mM could alter binding of nicotine to both sites. d-Tubocurarine, a nicotinic cholinergic antagonist, did not compete with nicotine for binding on either site. This finding along with the observation that mecamylamine was a competitive ligand for this site suggests that the higher affinity site being sodium independent was probably noncholinergic.

82–0705. Kozak, J. **Kourent a Patologie Dychani. [Smoking and Respiratory Pathology.]** *Prakticky Lekar* 60(7/8): 254-258, December 22, 1980, Czechoslovakian.

The contents and the physical and chemical properties of cigarette smoke, and its influence on the morphology of bronchial mucous membranes, are examined. The factors on which the physical and chemical properties of cigarette smoke depend include the quality of the tobacco, the filter, additives, cigarette length, and paper quality. Research dealing with the morphological changes in animals such as mice, rats, and dogs is reviewed. Changes, particularly effects on the mucociliary transport, which result from smoking over a period of at least 1 year, are evaluated. It appears that once chronic bronchitis occurs, the mucus transport is irrevocably disrupted. Also discussed is the influence of smoking on the ventilatory functions of the lungs, the mechanics of breathing, and other lung functions.

82–0706. Liard, R.; Perdrizet, S.; Reinert, P. **Wheezy Bronchitis in Infants and Parents' Smoking Habits. (Letter).** *Lancet* 1(8267): 334-335, February 6, 1982.

The suggestion that respiratory symptoms in children are related to an irritant factor in parental cigarette smoke is made, citing a study of 424 babies, aged 6 to 12 months. The mothers were interviewed about their babies' respiratory diseases and on the number of cigarettes smoked per day at home. No relation was found between the number of cigarettes and either upper respiratory tract diseases or cough and simple bronchitis. But there was a strong relation between "wheezy bronchitis" and cigarette consumption. The fact that wheezy bronchitis was related to smoking whereas simple bronchitis was not supports the hypothesis of irritation rather than cross-infection. (Auth. Abs. Mod.)

82–0707. Martin, A. J.; Landau, L. I.; Phelan, P. D. **Asthma From Childhood at Age 21: The Patient and His Disease.** *British Medical Journal* 284(6313): 380-382, February 6, 1982.

Information was obtained from 336 21-year-olds, who had begun wheezing before the age of 7, about their knowledge of asthma and its effect on their current lifestyle. Two-thirds of the subjects were still symptomatic. A control group of 62 subjects was available for comparison. The incidence of smoking was disturbingly high in all asthma groups. The higher the number of cigarettes ever smoked and the higher the current tobacco consumption the less satisfactory was the progress of asthma. Both cigarette smoking and severity of asthma contributed to chronic production of sputum. Knowledge about asthma was poor, particularly among those with less troublesome symptoms. Half of those with frequent episodic asthma and one-third with persistent asthma did not regard excess use of bronchodilator aerosols as potentially dangerous. Over three-quarters of those with frequent episodic asthma and two-fifths of those with persistent asthma were not receiving adequate treatment. One-third of those with persistent asthma were missing substantial time from work because of respiratory illness, and a similar proportion were restricting sporting activities. (Auth. Abs. Mod.)

82–0708. Merchant, J. A.; Pratt, P. C.; Vollmer, R. T. **Letters to the Editor. (Letters).** *Archives of Environmental Health* 36(1): 43-45, January-February 1981.

Criticisms are presented of an earlier paper (*Archives of Environmental Health* 35(3): 133-138, May-June 1980) which had proposed the thesis that airways obstruction in cotton textile workers was reversible if no centralobular emphysema (CLE) was present. A commentary which followed the paper (*Journal of the American Medical Association* 244(16): 1797-1798, October 17, 1980) is also criticized. It is argued that probable biases were introduced in the paper's selection and definition of exposed and control groups, and that the occupational history data was inadequate. Subjects without a charted smoking history were not included in the analysis. The sample size for cotton exposure was small as opposed to cigarette smoke exposure. There was evidence for bronchitis being proportionately larger for nonsmoking cotton textile workers than for all the smokers. Finally, it is argued that the contention that the airway lesions are "reversible" was not supported by the paper's data or by the epidemiological literature. Some of the paper's authors respond, defending their use of selection factors and occupational information. It is also pointed out that the suggestion that bronchial lesions associated with exposure to cotton dust might be reversible was no more than a suggestion. See also 80-1692 and 81-1459.

82–0709. Mitchell, C.; Emmerson, B.; Aroney, C. **Prevalence of Respiratory Morbidity in Brisbane Waterside Workers. A Study of Possible Asbestos-Related Disease.** *Medical Journal of Australia* 2(3): 139-141, August 8, 1981.

Results of a study of 93 Brisbane waterside workers who had been exposed to several dusts, particularly that of asbestos fiber, suggests that development of parenchymal lung disease is probably related to asbestos-dust exposure. A modified British Medical Research Council questionnaire was used to record respiratory symptoms, smoking habit, and detailed occupational history. The mean age of the workers was 61.2 (range, 42-80); 18 nonsmokers, 40 ex-smokers, and 40 current smokers. Forty-nine subjects complained of dyspnea while walking uphill, 46 had basal endinspiratory crepitations, and chest X-ray films showed small irregular opacities in 41 subjects, and pleural abnormalities in 27 subjects. The prevalence of cough, sputum production, and dyspnea were related to cigarette consumption, whereas the prevalence of wheeze, dyspnea, and crepitation were not. Internal comparisons showed that ventilatory function was correlated significantly with duration of employment on the wharves, independently of the effect of age and total cigarette consumption. Forced vital capacity decreased by 20 mL/year on wharves, and one-second forced expiratory volume by 34 mL/year, in excess of the age-related decrease. (Auth. Abs. Mod.)

82–0710. Musk, A. W.; Cotes, J. E.; Bevan, C.; Campbell, M. J. **Relationship Between Type of Simple Coalworkers' Pneumoconiosis and Lung Function. A Nine-Year Follow-Up Study of Subjects With Small Rounded Opacities.** *British Journal of Industrial Medicine* 38(4): 313-320, November 1981.

Smoking adversely affected several indicators of pulmonary function in a study group of coalworkers with simple pneumoconiosis. The subjects who had the simple pneumoconiosis in 1968 were re-examined 9 years later. The participants completed a questionnaire on respiratory symptoms and smoking habits and a full occupational history. Pulmonary function was measured, and indices of coal dust exposure were calculated. Chest radiography and statistical procedures were also used. It was found that subjects who were cigarette smokers in 1978 had a significantly lower forced expiratory volume in 1 second (FEV$_1$), forced vital capacity (FVC), and FEV/FVC, as well as a higher residual volume/total lung capacity (RV/TLC). Ex-smokers had a lower transfer factor for the lung for carbon monoxide, a lower transfer coefficient, and a higher RV/TLC compared to nonsmokers. Those subjects with irregular opacities did not appear to be unusual in their smoking habits.

82–0711. Poe, R. H.; Dass, T.; Celebic, A. **Small Airway Testing and Smoking in Predicting Risk in Surgical Patients.** *American Journal of the Medical Sciences* 283(2): 57-63, March-April 1982.

The value of the maximum expiratory flow volume curve (MEFV), the closing volume (CV), and the single breath nitrogen test (SBN$_2$) was assessed in comparison to smoking history as a means of predicting pulmonary complication in patients undergoing surgery. The 185 patients were tested, and their smoking habits evaluated by questionnaire. Of these, 55 patients had upper abdominal surgery, and 130 had other surgery under general or spinal anesthesia. There were 96 smokers (52 percent), 79 (43 percent) of which had one or more abnormal test parameters. The CV was abnormal in 59, alveolar plateau in 18, and MEFV in 12. Fifty-four percent of the smokers and 30 percent of the nonsmokers had abnormal small airway tests. Sixteen of 185 patients (8.6 percent) developed a postoperative respiratory complication determined by review of the hospital record. Eleven were in the 96 smokers, but 13 were in the 79 patients with abnormal small airway tests. The incidence was highest (38 percent) in smokers with an abnormal alveolar plateau who underwent upper abdominal surgery. Identification of the patient at risk was better served by tests of small airway abnormality than by smoking history alone. (Auth. Abs. Mod.)

82–0712. Rossman, M. D.; Dauber, J. H.; Cardillo, M. E.; Daniele, R. P. **Pulmonary Sarcoidosis: Correlation of Serum Angiotensin-Converting Enzyme With Blood and Bronchoalveolar Lymphocytes.** *American Review of Respiratory Disease* 125(3): 366-369, March 1982.

Blood and bronchoalveolar (BA) cell populations were compared with serum angiotensin-converting enzyme activity (SACE) in smoking ($n=8$) and nonsmoking ($n=14$) patients with recently diagnosed or active sarcoidosis. There was no correlation between SACE and blood lymphocyte populations, but a significant correlation was found between the number of BA lymphocytes and SACE and the number of BA T cells and SACE. The proportion of BA lymphocytes correlated well with SACE for nonsmokers ($r=0.659$, $p<0.002$) but not for smokers ($r=0.539$, $p>0.15$). Although the increase in the number of total BA lymphocytes was of borderline significance ($0.05<p<0.1$), in smoking patients with sarcoid, the increase in T cells and activated T cells in BA fluid was significant compared with that in control subjects. For the total patient group (smokers and nonsmokers), only the results for BA lymphocytes and T cells were compared with those in normal subjects because substantial differences existed between smokers and nonsmokers in cell yield, the number of macrophages, and the number of "activated" T cells. The increased number of BA lymphocytes in the total patient group was due almost entirely to the increased recovery of BA T cells. In no case was SACE elevated when the number of BA lymphocytes was normal, but in 10 patients with increased numbers of BA lymphocytes, SACE was normal. In fact, SACE was increased in only 7 of the 22 patients. These results suggest that both

SACE and the number of lymphocytes in lavage fluid reflect the intensity of the lung's inflammatory response in sarcoidosis but that quantification of BA lymphocytes is a more sensitive measure than SACE of active pulmonary sarcoidosis. (Auth. Abs. Mod.)

82–0713. Samet, J. M.; Schrag, S. D.; Howard, C. A.; Key, C. R.; Pathak, D. R. **Respiratory Disease in a New Mexico Population Sample of Hispanic and Non-Hispanic Whites.** *American Review of Respiratory Disease* 125(2): 152-157, February 1982.

A survey of major respiratory disease prevalence among study groups of Hispanics and Anglos in Bernalillo County, New Mexico, found that the Hispanics had a lower cigarette consumption and a different disease prevalence. Physician-confirmed chronic bronchitis or emphysema, and asthma were reported less often by Hispanics. Although patterns of cigarette usage (current, previous, never) were similar, current and cumulative cigarette consumption was significantly lower in Hispanics. Most differences in symptom frequency and the lower Hispanic prevalence of chronic bronchitis or emphysema were attributable to lower cigarette consumption by Hispanics. However, the prevalence of asthma remained significantly lower among Hispanics after controlling for cigarette smoking. (Auth. Abs. Mod.)

82–0714. Simecek, C. **Reflection of Passive Exposure to Smoking in the Home on the Prevalence of Chronic Bronchitis in Non-Smokers.** *Czechoslovak Medicine* 3(4): 308-310, 1980.

Analysis of data assembled in an epidemiological investigation of 123,235 adults from three districts of the West Bohemian region, including 57,217 female nonsmokers from a total of 66,806 females, and 23,473 male nonsmokers from a total number of 56,429 males, revealed a highly significant ($p<0.005$) rise in the prevalence of symptoms of chronic bronchitis in subjects exposed passively to smoking in the home. Exposure to other adverse factors increased the prevalence approximately three times in both sexes. The effect of passive exposure to smoking was, however, manifested far more in men. No reason was found in the investigated data for the interpretation of these differences. It appears that the cause could be the different behavior of smokers to nonsmokers in the home, where the smoker-husband usually is considerate to his nonsmoker wife during her pregnancy and child care, while in women smokers this motivation is lacking. (Auth. Abs.)

82–0715. Sparrow, D.; Bosse, R.; Rosner, B.; Weiss, S. T. **The Effect of Occupational Exposure on Pulmonary Function. A Longitudinal Evaluation of Fire Fighters and Nonfire Fighters.** *American Review of Respiratory Disease* 125(3): 319-322, March 1982.

Firefighters showed reduced pulmonary function compared to nonfirefighters during this examination of the chronic effect of firefighting on pulmonary function, a difference which could not be accounted for by difference in smoking status. A longitudinal analysis was conducted of 168 firefighters and 1,474 nonfirefighters enrolled in a study of normal aging. Spirometric measurements of forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV$_1$) were obtained on all subjects over a 5-year intake period (1963 to 1968), and subjects were reexamined 5 years later (1968 to 1973). Questionnaire information about smoking habits, respiratory symptoms, and illness was also obtained. Firefighters had a greater loss of pulmonary function (FVC and FEV$_1$) than nonfirefighters ($p=0.007$ and $p=0.054$). This occupational effect could not be explained by differences in age, height, smoking status, or initial level of pulmonary function between the two occupational groups. Although respiratory symptoms were greatest among current cigarette smokers, they were not significantly different between either occupational group. However, firefighters generally reported more symptoms when smoking was controlled for. These results confirm earlier reports of a chronic effect of firefighting on pulmonary function and suggest an association of this occupation with increased respiratory symptoms and disease independent of cigarette smoke. (Auth. Abs. Mod.)

82–0716. Yarnell, J.W.G.; St. Leger, A. S. **Respiratory Morbidity and Lung Function in Schoolchildren Aged 7 to 11 Years in South Wales and the West of England.** *Thorax* 36(11): 842-846, November 1981.

The suggestion that the respiratory morbidity of children residing in South Wales is substantially higher than that among children residing in some other areas of the United Kingdom was tested. A case—control survey was carried out among 2,228 children aged 7 to 11 years in schools matched for size and socioeconomic characteristics in urban and rural areas in South Wales and the West of England (Avon). The survey confirmed that respiratory morbidity was higher among children in South Wales. Smoking habit and other environmental factors such as higher annual rainfall may provide an explanation. Household cigarette smoking was more common in Welsh families (62 percent) than those from Avon (54 percent). Similarly, 43 percent of women from South Wales reported smoking during pregnancy compared with 36 percent from Avon. However, objective measurements of the children's respiratory health did not detect any consistent difference in the lung function of children in South Wales compared with that of children in Avon. (Auth. Abs. Mod.)

82–0717. Yarnell, J.W.G.; St. Leger, A. S. **Respiratory Infections and Their Influence on Lung Function in Children: A Multiple Regression Analysis.** *Thorax* 36(11): 847-851, November 1981.

The relationship between a history of respiratory infections (and associated variables) in children and lung function in later life was examined in a study among 2,228 children aged 7 to 11 years. In a multiple regression analysis only a few variables showed marked and consistent effects on lung function. Respiratory tract infections showed increasing impairment of lung function with repeated infections, but the impairment was smaller than that caused by current asthma. Similar observations may be made for winter cough and smoking in pregnancy but only the change in FEV$_{0.75}$I (forced expiratory volume in 0.75 second adjusted for height) for winter cough achieved statistical significance. Marked area effects were apparent for FEV$_{0.75}$I but not for the ratio. Household smoking, maternal and paternal phlegm, and age at infection failed to show any concistent relationship with lung function. The present data are consistent with the hypothesis that smoking in pregnancy has an independent effect on lung function. The slope of the regression line did not attain statistical significance but demonstrated a consistent dose—response relationship. Compared to the effect of asthma or recurrent bronchitis on lung function, however, the effect was small. Current maternal and total household smoking habit failed to show any independent relationship to lung function. (Auth. Abs. Mod.)

See also, **82–0674, 82–0676, 82–0686, 82–0749**

CARDIOVASCULAR DISEASES

82–0718. Arber, F.; Darioli, R.; Monti, M.; Essinger, A. **Interet du Dosage du HDL-Cholesterol dans le Bilan des Arteriopathies des Membres Inferieurs. [Importance of Determination of High-Density Lipoprotein Cholesterol in Peripheral Arterial Disease.]** *Schweizerische Medizinische Wochenschrift* 111(50): 1969-1971, December 12, 1981, French.

The incidence of known atherogenic risk factors was compared with that of high-density lipoprotein(HDL)-cholesterol in 13 female and 39 male patients with peripheral arterial disease (PAD) evaluated by arteriography. The incidence of smoking, determined to be at least 20 cigarettes/day for 10 years in 90.2 percent of all patients, was lower in female than in male patients. Thus, smoking appears to be the most important atherogenic risk factor, followed by dyslipidosis, sedentary lifestyle, and HDL-cholesterol level decrease relative to its level in healthy controls (16 men, all smokers, and 13 women, of whom 3 were smokers). The decrease in HDL-cholesterol level in 76.9 percent of the patients compared to the controls was very significant for both men and women. Of all the risk factors studied, only HDL-cholesterol level was significantly correlated with the severity of the PAD. It is concluded that the decrease in HDL-cholesterol level may be considered as a risk factor independent of smoking, and closely associated with the incidence and gravity of PAD.

82–0719. Auerbach, O.; Carter, H. W. **Smoking and the Heart.** In: Bristow, M. R. (Editor). *Drug-Induced Heart Disease.* Meyler and Peck's Drug-Induced Diseases, Volume 5. Amsterdam, Elsevier/North-Holland Biomedical Press, 1980, pp. 359-376.

Autopsies of 1,056 hearts of men who had died of noncardiac causes were performed to determine the effects of smoking on the vessels. Diabetics and hypertension cases were excluded. In a macroscopic study, the arteries were classified by degree of atherosclerosis, then segmented and rated for degree of fibrous thickening, atheromas, and calcification. Myocardial sections from 10 locations in each heart were microscopically examined for degree of fibrous thickening, atheroma of the intima of myocardial and subepicardial arteries, and hyaline thickening of the myocardial arterioles. Both fibrous thickening and atherosclerosis were higher in smokers than in nonsmokers, and increased with number of cigarettes consumed per day, and with age. Advanced atherosclerosis was more than four times higher in men smoking two or more packs per day than in those who never smoked. Atheroma and calcification were less common, but showed a greater gradient by age group. Fibrous thickening of the subepicardial and myocardial arteries was directly associated with number of cigarettes consumed, with a greater increase observed in the myocardial arteries. Advanced fibrous thickening of the intima was found in nearly 28 times as many of the men who smoked two packs per day as in men who never smoked regularly. Moderate to advanced hyaline thickening of the arterioles in the myocardium was even more significantly related to smoking, and was found in 98.6 percent of the cases with a two-pack-per-day smoking habit, but was never found in the nonsmoking group.

82–0720. Bazin, M.; Turcotte, H.; Lagace, R.; Boutet, M. **Effets Cardiovasculaires de la Fumee de Cigarette Chez le Rat. Etude de la Permeabilite Endotheliale Aortique et Capillaire Myocardique Chez le Rat. [Cardiovascular Effects of Cigarette Smoke in the Rat. Aortic Endothelium and Myocardial Capillaries Permeability Study in the Rat.]** *Revue Canadienne de Biologie* 40(3): 263-276, September 1981, French.

The permeability of aortic endothelium and myocardial capillaries was examined in 72 Sprague-Dawley rats using the diffusion tracer peroxidase. Animals were subjected to cigarette smoke for periods of 1 day (acute effects), 2 weeks (subacute effects), and 15 weeks (chronic effects), and were sacrificed 5 minutes or 8 hours after smoking. Peroxydase was not generally present at the subendothelial level in the aortic endothelium of control animals and sham-smokers. However, in smokers, the increase in peroxidase permeability is proportional to the smoking period. Moreover, in these smokers junctional and subendothelial vacuolar dilatations were observed which correspond to degenerative morphologic changes. Cigarette smoke has a different effect on the right ventricle. Endothelial permeability of the myocardial capillaries increases in animals subjected to cigarette smoke for 1 day and those who smoked for 2 weeks sacrificed 5 minutes after smoking. This increase in permeability is not related to the significant morphologic changes observed in the myocardium and endothelial capillaries. However, the myocardial endothelial capillaries, the subendothelial space, and the adjacent interstitial space were generally free of peroxidase in animals subjected to cigarette smoke for 15 weeks and in those smoking for 2 weeks sacrificed 8 hours later. An adaptive phenomenon to the effects of cigarette smoke appears to exist in the myocardial endothelium capillaries contrary to that which is observed in the thoracic aorta. (Auth. Abs.)

82-0721. Booyse, F. M.; Osikowicz, G.; Radek, J. **Effect of Nicotine on Cultured Bovine Aortic Endothelial Cells.** *Thrombosis Research* 23(1/2): 169-185, July 1 and 15, 1981.

The short- (1-4 days) and long-term (10 passages, 12-14 weeks) effect of nicotine tartrate (NT) has been examined on the following cultured bovine aortic endothelial cell properties and functions: growth properties, morphology, total protein and collagen synthesis and turnover, fibrinolysis, lactate production, and platelet-subendothelial interaction/adhesion. Low concentrations of NT ($< 10^{-4}$M) had no apparent effect on endothelial cell proliferation, morphology, DNA synthesis (thymidine incorporation), overall protein synthesis (leucine and proline incorporation), fibrinolysis, lactate production, and platelet-subendothelial interaction/adhesion. However, NT (10^{-5} and 10^{-4}M) did increase the net accumulation of collagen in these cultures. In addition, high concentrations of NT ($> 10^{-4}$M) caused rapid and extensive vacuolation of these cultured bovine endothelial cells and also increased lactate production twofold. (Auth. Abs.)

82-0722. Criqui, M. H.; Wallace, R. B.; Mishkel, M.; Barrett-Connor, E.; Heiss, G. **Alcohol Consumption and Blood Pressure. The Lipid Research Clinics Prevalence Study.** *Hypertension* 3(5): 557-565, September-October 1981.

Measurement of alcohol consumption and systolic and diastolic blood pressure (BP), in a cohort of 2,482 men and 2,301 women 20 years of age or older in nine North American populations, revealed a negative relationship with smoking. Men at the highest level of alcohol consumption ($\geq$ 30 ml alcohol per day) had the highest BP, while women either at the highest level of alcohol consumption or consuming no alcohol had the highest BP. Men aged $\geq$ 35 years of age consuming $\geq$ 30 ml alcohol per day were 1.5 to 2 times more likely to be hypertensive than nondrinkers. Multivariate analysis showed systolic and diastolic BP in both men and women to be positively and significantly ($p<0.05$) related to alcohol consumption. This relationship was independent of the potential confounding effects of age, obesity, cigarette smoking, regular exercise, education, and gonadal hormone use in women. Analyses suggested the univariate U-shaped alcohol-BP association in women was confounded by differences in obesity and cigarette smoking in nondrinking women, and by very low alcohol consumption in hypertensive women using medication. Multivariate data are presented demonstrating a strong relationship between age and BP and obesity and BP. However, cigarette smoking shows a negative relationship to BP; smoking a pack a day is associated with a systolic BP reduction of 2 to 3 mm Hg. It is concluded that alcohol appears to have a modest but consistent and independent effect on systolic and diastolic BP. (Auth. Abs. Mod.)

82-0723. Daniel, M. L. **Smoking, Exercise, and Selected Coronary Risk Factors.** Doctoral Dissertation, University of Arkansas, Fayetteville, University Microfilms International 81-27228, May 1981, 118 pp.

Males between the ages of 18 and 45 were recruited from Fayetteville and the surrounding communities, on the basis of their being sedentary (having engaged in no regular exercise for the preceding two years). Those who smoked at least one pack of cigarettes per day were placed in a smoker's group ($n = 13$); those who had abstained from smoking for at least the preceding two years were placed in a nonsmoking group ($n = 18$). Fasting blood samples were taken from each participant and analyzed for levels of sugar, triglycerides, cholesterol, and for the lipoprotein makeup of the cholesterol. Additionally, each participant was measured for percentage of body fat, and for resting levels of systolic and diastolic blood pressure. Using age as a guide, an individualized exercise program was prescribed, consisting of an aerobic circuit training routine, conducted a minimum of three and maximum of five times per week for 8 weeks. Records were kept of exercise sessions attended, working heart rates, and recovery heart rates. At the end of the 8-week period, measures of blood chemistry, blood pressure, and body fat were again taken. Seven smokers and ten nonsmokers completed the exercise portion of the program, and posttests were conducted with only these participants. After adjusting for variables, there were no significant differences observed between smokers and nonsmokers on pretest for percent body fat, systolic blood pressure, serum sugar, serum triglycerides, serum cholesterol, lipoproteins, or lipoprotein ratios. Smokers displayed a significantly higher diastolic blood pressure than did the nonsmokers. Following the exercise program, there were no significant changes other than in blood pressure; smokers showed a significantly greater change (loss) in diastolic pressure than did nonsmokers (slight gain). (Auth. Abs. Mod.)

82-0724. Deanfield, J.; Jonathan, A.; Selwyn, A.; Fox, K. M. **Treatment of Angina Pectoris With Propranolol: The Harmful Effects of Cigarette Smoking.** *Cardiology* 68(Supplement 2): 186-189, 1981.

The effects of smoking on the treatment of angina in 10 patients are reported. Each patient underwent 4 randomized 1-week treatments consisting of on and off smoking, and on and off propranolol (360 mg/day). At the end of each treatment a maximal exercise test was performed using 16 precordial leads. Smoking was associated with a significantly higher heart rate, blood pressure, area and severity of ST-segment depression. This was not abolished by propranolol. Thus, smoking aggravates the simple hemodynamic variables used to assess myocardial oxygen requirements and the electrocardiographic signs of ischemia; this is true even after treatment with propranolol. (Auth. Abs.)

82–0725. Dumas, J. P.; Guilland, J. C.; Braud, M. L.; Boggio, V.; Klepping, J. **Reactions Cardio-Vasculaires et Sympathico-Medullosurrenaliennes a l'Intoxication Tabagique Aigue. [Cardiovascular Reaction and Sympathetic Medulla Glandulae Suprarenalis Reaction to Acute Tobacco Intoxication.]** *Comptes Rendus des Seances de la Societe de Biologie et de ses Filiales* 175(6): 866-869, 1981, French.

The effects of cigarette quick-smoking on plasma catecholamines levels, heart rate, and arterial pressure were studied in 15 subjects. Smoking-associated increments in mean plasma norepinephrine and epinephrine as well as in heart rate and arterial pressure were demonstrated. The lack of correlation between the increase of the adrenergic system activity and the hemodynamic parameters can be explained by individual characteristics. (Auth. Abs.)

82–0726. Folts, J. D.; Bonebrake, F. C. **The Effects of Cigarette Smoke and Nicotine on Platelet Thrombus Formation in Stenosed Dog Coronary Arteries: Inhibition With Phentolamine.** *Circulation* 65(3): 465-469, March 1982.

The effects of cigarette smoke on cyclic reductions in coronary flow due to platelet thrombus formation were examined. The circumflex coronary artery of 21 mongrel dogs was stenosed 60 to 80 percent, and the blood flow measured with an electromagnetic flow probe. After the administration of cigarette smoke, plasma epinephrine was elevated nine times the control level ($p<0.001$) and peak mean blood pressure was elevated one and one-half times control ($p<0.01$). The hematocrit increased several percent ($p<0.01$) with cigarette smoke, although blood gases and pH remained unchanged. In all 21 dogs, spontaneous reductions in coronary blood flow were greatly exacerbated in the stenosed circumflex artery, as evidenced by the number of flow reductions, the increased size of the reductions, and the rate of flow reduction. Nicotine administered intravenously in doses comparable to those achieved through absorption of cigarette smoke by the lungs provoked similar responses of α-adrenergic stimulation and potentiation of the platelet thrombus formation. An α-adrenergic antagonist, phentolamine, was given (3 mg/kg) intravenously to inhibit the exacerbated platelet thrombus formation due to cigarette smoke or infused nicotine. In 18 of 21 dogs, an acute occlusive platelet thrombus was prevented 15 minutes after phentolamine and after a cigarette smoke or nicotine challenge. This study confirms a link between cigarette smoking, platelet thrombus formation, and the potential for humans to develop an acute occlusive platelet thrombus in a diseased and stenotic coronary artery. (Auth. Abs. Mod.)

82–0727. Heiss, G.; Haskell, W.; Mowery, R.; Criqui, M. H.; Brockway, M.; Tyroler, H. A. **Plasma High-Density Lipoprotein Cholesterol and Socioeconomic Status. The Lipid Research Clinics Program Prevalence Study.** *Circulation* 62(4): 108-115, November 1980.

Socioeconomic status and lifestyle, as measured by educational achievement, were found to be associated with high-density lipoprotein (HDL) cholesterol in adult populations in a study of 2,182 white females and 2,368 white males, ages 20-59 years, in nine North American populations surveyed. Mean HDL cholesterol values were positively associated with reported educational achievement. Among females of all ages and males ages 20-39 years, a gradient of HDL cholesterol levels from lowest to highest educational stratum was found. A similar trend of lower magnitude was noted among older males. The positive association between educational and HDL cholesterol levels was statistically significant in both sexes ($p<0.0002$) and was consistent for each of the study populations. Differences in mean HDL cholesterol between educational strata were in part explained statistically by education-related differences in body mass, alcohol consumption, cigarette smoking (inversely related to educational achievement in females, the association being more pronounced in younger females), age, and, gonadal hormone use in women. (Auth. Abs. Mod.)

82–0728. Hojnacki, J. L.; Mulligan, J. J.; Cluette, J. E.; Kew, R. R.; Stack, D. J.; Huber, G. L. **Effect of Cigarette Smoke and Dietary Cholesterol on Plasma Lipoprotein Composition.** *Artery* 9(4): 285-304, 1981.

The effect of acute inhalation of cigarette smoke and consumption of dietary cholesterol on plasma lipoprotein composition in atherosclerosis-susceptible White Carneau pigeons was examined. Pigeons were assigned to four treatment groups: (1) controls; (2) sham pigeons fed a cholesterol-saturated fat diet and exposed to fresh air; (3) animals fed the cholesterol diet and exposed to low concentrations of low nicotine-low carbon monoxide (LoLo) cigarette smoke; and (4) birds fed the cholesterol diet and subjected to high concentrations of high nicotine-high carbon monoxide (HiHi) cigarette smoke. Differences appeared in HiHi high density lipoproteins (HDL) which contained more free and esterified cholesterol and total lipid, but less total protein than HDL from sham-smoked pigeons. Similarly, very low density lipoproteins (VLDL) from birds exposed to cigarette smoke (LoLo and HiHi) contained more total lipid, but less total protein, than VLDL from sham pigeons. Inhalation of tobacco smoke also produced a marked depression in the HDL$_2$/HDL$_3$ ratio resulting from an increased proportion of the HDL$_3$ subfraction relative to HDL$_2$. Pigeons (sham, LoLo, HiHi) fed the cholesterol-saturated fat diet circulated HDL with greater free and esterified cholesterol mass than controls. VLDL particles from these three treatment groups were enriched with cholesterol and cholesteryl ester at the expense of triglyceride. Diet also altered the type of cholesteryl ester present in HDL with cholesteryl linoleate representing the predominant form in control pigeons and cholesteryl oleate in cholesterol-fed birds. These results demonstrate that cigarette smoking can mediate alterations in lipoprotein composition independent of changes induced by dietary cholesterol and saturated fat. (Auth. Abs. Mod.)

82–0729. Hubert, H. B.; Holford, T. R.; Kannel, W. B. **Clinical Characteristics and Cigarette Smoking in Relation to Prognosis of Angina Pectoris in Framingham.** *American Journal of Epidemiology* 115(2): 231-242, February 1982.

Among a study group of angina pectoris patients, nonsmokers and ex-smokers appeared to have a better prognosis than smokers. A long-term followup study of 130 Framingham, Massachusettts, men with newly diagnosed uncomplicated angina pectoris was undertaken in an attempt to identify medical and personal characteristics such as smoking which could predict the likelihood of a future coronary event. The lack of association between smoking at diagnosis and a new event appeared to be related to changes in habits after angina onset. Further scrutiny of the data indicated that, irrespective of blood pressure and electrocardiographic findings, those less than 60 years of age at angina onset who were nonsmokers or quitters during followup had a definite prognostic advantage over similarly aged continuing smokers. These results could not be explained by differences in coronary risk factors prior to symptom onset or by changes in factors other than smoking during followup. The findings suggest that stopping the cigarette smoking habit can improve both short-term and long-term prognosis in the younger patient with angina pectoris. (Auth. Abs. Mod.)

82–0730. Jones, B. R.; Barrett-Connor, E.; Criqui, M. H.; Holdbrook, M. J. **A Community Study of Calorie and Nutrient Intake in Drinkers and Nondrinkers of Alcohol.** *American Journal of Clinical Nutrition* 35(1): 135-139, January 1982.

Dietary differences suggest one mechanism whereby moderate alcohol consumption might reduce the risk of coronary heart disease. Smoking habits, exercise, nutrient intake, and alcohol consumption were studied in a 15 percent random sample of a predominately white upper-middle class surburban community in Southern California. In general, alcohol-derived calories were added to the diet, and did not replace calories derived from other nutrients. Consequently, alcohol consumers had a significantly higher total caloric intake than did nondrinkers. Dietary differences were greatest in moderate-drinkers, who tended to consume fewer nonalcohol-derived calories, and less of most specific nutrients. No consistent pattern of cigarette smoking according to alcohol usage was observed in men, but significantly more moderate drinking women smoked cigarettes. Although alcohol intake resulted in an increase in total calories consumed, alcohol drinkers were not more obese than nondrinkers. Since similar proportions of drinkers and nondrinkers exercised regularly, these data may suggest that alcohol calories are not fully utilized. (Auth. Abs. Mod.)

82–0731. Kannel, W. B. **Meaning of the Downward Trend in Cardiovascular Mortality.** *Journal of the American Medical Association* 247(6): 877-880, February 12, 1982.

Several possible causes are suggested for the decline in cardiovascular mortality that has taken place in recent years, including changing trends in smoking. Other possible factors cited include improved medical care, decreased incidence of disease, dietary changes, physical activity, and control of hypertension. Between 1965 and 1978, the proportion of cigarette smokers among men decreased by 35 percent and among women by 10 percent. However, the proportion of heavy smokers did not decrease.

82–0732. Kuch, J.; Lubiszewska, B.; Baraniewski, K.; Serwecinska, T.; Biro, E. **Czestosc Wystepowania Choroby Niedokrwiennej Serca w Populacji Generalnej oraz Jej Związek z Czynnikami Ryzyka. [Incidence of Ischemic Heart Disease in the General Population and Connection With Risk Factors.]** *Kardiologia Polska* 24(4): 269-277, 1981, Polish.

Ischemic heart disease (IHD) was diagnosed in 13.7 percent of 3,174 male subjects, aged 40 to 59 years, among the general population of the South Praga District of Warsaw, Poland. The diagnosis was based on the symptoms of angina pectoris, determined from a questionnaire, or initial ECG recordings suggesting myocardial ischemia. The incidence of IHD increased with age from 7.6 percent in the youngest group to 18.8 percent in the oldest. The incidence of IHD was higher in subjects with increased arterial hypertension, in overweight subjects, and in those with a high individual risk index. The IHD was detected more often in smokers and ex-smokers than in nonsmokers. In the two groups of older subjects (50 to 59 years old), IHD occurred twice as often in ex-smokers as in nonsmokers. Most of the ex-smokers gave up smoking because of bad health. Correlation between IHD and the cited risk factors was independent of age.

82–0733. Kuch, J.; Serwecinska, T.; Lubiszewska, B.; Biro, E.; Baraniewski, K.; Krol, J.; Wojtulewicz, L. **Wystepowanie Czynnikow Ryzyka Chorobi Niedokrwiennej Serca w Populacji Mezczyzn w Wieku 40-59 Lat z Dzielnicy Warszawy Praga Poludnie. [Prevalence of Risk Factors of Ischemic Heart Disease in a Male Population, Aged 40 to 59 Years, in the South Praga District of Warsaw.]** *Kardiologia Polska* 24(4): 279-285, 1981, Polish.

Five basic risk factors of ischemic heart disease (IHD) were evaluated in 3,174 male residents of a subdivision of the South

Praga District of Warsaw. The prevalence of each factor is defined as the percent of the studied population that was determined to have the factor above a given normal value. Thus, cigarette smoking was found in 50.7 percent of the studied subjects, arterial hypertension in 26.9 percent, hypercholesterolemia in 23.4 percent, obesity in 18.4 percent, and low physical activity in 77.3 percent. The subjects who smoked one or more cigarettes/day were defined as smokers. In addition, 27.8 percent of the total sample were ex-smokers. The smoking habit decreased with age from 56.3 percent of the subjects aged 40 to 44 years, to 45.5 percent for those aged 55 to 59 years. The percentage of ex-smokers increased with age. The individual risk index evaluated as the sum of all the risk factors plus age was considered high (11.2 percent of the total population) and increased significantly with age. Hypertension, smoking, and obesity were found to be as prevalent as in the male industrial workers in Warsaw plants. The prevalence of hypercholesterolemia and low physical activity was significantly higher than that previously found in the male industrial population.

82–0734. Kunzler, A.; Kappenberger, L.; Niederhauser, H. U.; Steinbrunn, W. **Rhythmusstoerungen in der Rehabilitationsphase nach Herzinfarkt. [Arrhythmias During Rehabilitation Period After Myocardial Infarction.]** *Schweizerische Medizinische Wochenschrift* 111(50): 1962-1964, 1981, German.

Laboratory tests (exercise test on a bicycle ergometer) and 24-hour continuous ECG monitoring were performed in 45 male patients, aged 37 to 67 years, at the beginning and the end (21 days later) of a 22- to 80-day inpatient rehabilitation period following an acute uncomplicated myocardial infarction. The tests were repeated in 3.5 to 9 months after discharge. During rehabilitation, the patients were not treated with drugs. Severe ventricular arrhythmias were recorded by at least one 24-hour ECG in 14 (31 percent) of the patients. The exercise test revealed only one case of severe arrhythmia. All patients with severe arrhythmia had either reduced work capacity or myocardial ischemia or both. Seven nonsmoking patients (16 percent) had only mild arrhythmias or none at all. The rehabilitation program had no apparent effect on arrhythmias.

82–0735. Leaman, D. M.; Brower, R. W.; Meester, G. T. **Coronary Artery Bypass Surgery. A Stimulus To Modify Existing Risk Factors?** *Chest* 81(1): 16-19, January 1982.

Coronary artery bypass surgery patients were urged before surgery to modify several risk factors such as smoking, but an evaluation 1 year after the operation found little modification of smoking patterns. Other risk factors mentioned were elevated blood pressure, cholesterol, and obesity. The risk factors of a positive family history and diabetes mellitus were considered more difficult to manipulate. Of the 226 patients in the study, 15 were nonsmokers, 102 were ex-smokers, and 109 were smokers before the operation. At the 1 year postoperative evaluation, 15 were nonsmokers, 112 were ex-smokers, and 99 were smokers. Smoking appeared to be a difficult habit to give up, which could be due to many smokers beginning smoking at an early age and forming smoking habits, as well as to the body's dependence on tobacco products. It is suggested that people should be encouraged not to adopt risk behaviors such as smoking. Further research into behavior modification methods may be needed.

82–0736. Lu, C.-T.; Zarins, C. K.; Yang, C.-F.; Sottiurai, V. **Long-Segment Arterial Occlusion: Percutaneous Transluminal Angioplasty.** *American Journal of Roentgenology* 138(1): 119-122, January 1982.

Twenty-one patients aged 54 to 88 years (10 men, 11 women) underwent percutaneous transluminal angioplasty (PTA). Seven of the eight heavy smokers in the group developed reocclusion within 1 week to 7 months after PTA. In contrast, only 2 of the 10 nonsmokers developed reocclusion at 5-24 months followup. In a 5-year followup study of PTA patients, data showed that the arteries of about 45 percent of those patients who had discontinued smoking were still patent, as opposed to no more than 18.8 percent and 14.3 percent for cigarette and pipe/cigar smokers, respectively. It is concluded that long-term benefit for PTA patients can be improved through smoking cessation.

82–0737. Marmot, M. G.; Rose, G.; Shipley, M. J.; Thomas, B. J. **Alcohol and Mortality: A U-Shaped Curve.** *Lancet* 1(8220): 580-583, March 14, 1981.

In a longitudinal study of civil servants, 1,422 men were classified according to their average daily alcohol intake. Over 10 years of followup, the mortality rate was lower in men reporting moderate alcohol intake than in either nondrinkers or heavier drinkers (> 34g alcohol per day). Both the percent of men who were smokers and the mean number of cigarettes consumed per day per smoker show a U-shaped relationship to alcohol consumption, higher in both the nondrinkers and the heavy drinkers. Statistical analysis reveals that moderate drinkers (0.1-34 g/day) have a lower mortality rate than nondrinkers, regardless of smoking status; however, among the ex-smokers, the moderate drinkers have a higher mortality than the heavy drinkers. When analysis was done separately for cardiovascular and noncardiovascular mortality, nondrinking smokers had higher cardiovascular mortality, and heavy drinking smokers had higher noncardiovascular mortality. A multivariate analysis showed the U-shaped relationship between reported alcohol consumption and subsequent mortality to be largely independent of differences in smoking, blood pressure, plasma cholesterol, and grade of employment. (Auth. Abs. Mod.)

82–0738. McNicol, M. W.; Turner, J.A.McM.; Wald, N. J.; Idle, M.; Boreham, J.; Bailey, A.; Van Vunakis, H. **Nicotine, Carbon Monoxide and Heart Disease. (Letters).** *Lancet* 1(8262): 40-41, January 2, 1982.

Results of a study (*Lancet* 2(8250): 775-777, October 10, 1981) which claimed that nicotine is not the major cause of the excess risk of coronary heart disease (CHD) mortality in cigarette smokers are disputed. This conclusion was based on the observation that pipe smokers have higher levels of cotinine than cigarette smokers but have much lower excess of heart disease. To refute this, it is pointed out that: (1) the high average cotinine level found in primary pipe smokers arose because some secondary pipe smokers (who are believed to inhale) were inadvertently included with the primary smokers (who are presumed not to inhale), (2) data on primary cigar smokers indicate that these smokers had low cotinine and low carboxyhemoglobin (COHb) levels and failed to show any absorption of nicotine through the buccal route, and (3) data on pipe smokers in the prospective mortality studies related mainly to primary smokers. It is concluded that nicotine can not be ruled out as a cause of CHD. In response to this and other criticisms (*Lancet* 2 (8254): 1044-1045, November 7, 1981), the authors of the original article stress that the conclusions of the study could not have been otherwise unless nicotine were not metabolized to cotinine similarly in all smokers, or unless nicotine absorbed through the pulmonary alveoli were more toxic than when absorbed through the buccal mucosa. The critical question is whether pipe smokers with serum cotinine levels as high as those found in the study can also have high serum nicotine levels. The general conclusions do not exclude the possibility of an interaction between carbon monoxide and nicotine. A check of the study data on pipe smokers found no error, while additional data indicated that it is wrong to assume that primary pipe smokers do not inhale. They have higher COHb levels than nonsmokers and their COHb levels increase in relation to the amount smoked. Since the cigar smokers smoked only one-fifth of a cigar before the blood test, it would seem likely that their serum cotinine levels would have been higher than observed. The final point, that data on pipe smokers in the prospective mortality studies related mainly to primary smokers, was made in the paper and was the reason why data for primary and secondary pipe smokers were presented separately. See also 82-0477 and 82-0470.

82–0739. Murata, K. **Tokkushu: Shinkekkan Shikkan Yobo no Gendankai II Shinkin Kosoku to shinzo Totsuzenshi. Shikkohin to Shinkin Kosoku. [The Current Stage in the Prevention of Myocardial Disease, II. Myocardial Infarction and Sudden Cardiac Death. Luxury Articles and Myocardial Infarction.]** *Japanese Journal of Clinical Medicine* 38(10): 135-139, 1980, Japanese.

A literature review of the studies on the relationship between myocardial infarction (MI) and tobacco, alcohol, coffee, and tea from the past 10 years is presented. According to the Framingham study, the main factors contributing to MI are blood pressure cholesterol, smoking, and blood sugar, in that order. The occurrence of MI among heavy cigarette smokers (more than 20 cigarettes/day) is at least twice as high as among nonsmokers; there is no difference between pipe or cigar smokers and nonsmokers. Myocardial infarction incidence per 1,000 people is 0.6 among nonsmokers, 1.9 among those who smoke less than 20 cigarettes/day, and 2.1 among those who smoke more than 20 cigarettes/day. The occurrence of myocardial infarction and atherosclerosis among those who used to smoke but quit is lower than among smokers. The risk factor for MI decreased by 50 percent after stopping smoking. The mechanism by which cigarette smoking contributes to myocardial infarction and atherosclerosis is not yet known. Studies conducted on the relationship between smoking and plasma HDL cholesterol indicate that smoking decreases the level of plasma HDL cholesterol.

82–0740. Mustard, J. F. **Cigarette Smoking, Atherosclerosis and Its Clinical Complications.** *Canadian Journal of Public Health* 72(6): 385-388, November-December 1981.

Cigarette smoking is a risk factor in the development of atherosclerosis and its clinical complications such as heart attacks and strokes. Injury to the vessel wall can contribute to the development of all aspects of atherosclerosis, providing a link between the known epidemiological association of cigarette smoking and ischemic heart disease, and the mechanisms through which cigarettes may contribute to atherosclerosis and its clinical complications. Cigarettes may contain substances which can directly or indirectly damage the vessel wall; this effect is important in the development of atherosclerosis as well as the thromboembolic events. Cessation of cigarette smoking is expected to reduce this effect and thereby reduce the risk of further development of the atherosclerotic lesions and clinical complications. The evidence suggests that if the stimuli are sufficiently diminished there may even be a regression of the atherosclerotic lesions. Epidemiological data concerning individuals who stop smoking is compatible with the theory that cessation of cigarette smoking results in a reduction in the risk of death from ischemic heart disease. (Auth. Abs. Mod.)

82–0741. Oldridge, N. B. **Compliance and Exercise in Primary and Secondary Prevention of Coronary Heart Disease: A Review.** *Preventive Medicine* 11(1): 56-70, January 1982.

Compliance rates in primary and secondary prevention of coronary heart disease are reviewed. Participation in health behavior change clinics such as exercise programs is low. The dropout from these programs is likely to be a smoker, blue collar worker, inactive in lifestyle and leisure time, has had more than one myocardial infarction, has a greater percentage of body fat, and is more extroverted than compliers. Reasons for noncompliance include inaccessibility and cost of coming to facility, lack of spouse support, interference with job, inconvenience of sessions, inability to pay program costs, lack of interest, and psychosocial and medical reasons. In designing cardiac rehabilitation programs, these factors should be considered in encouraging exercise and smoking cessation.

82–0742. Oleynikov, S. P.; Shishova, A. M.; Balavadze, M. B.; Zyryaeva, L. A. **Profilaktika Ishemicheskoy Bolezni Serdtsa**

Sredi Naseleniya. [Prevention of Ischemic Heart Disease Among the General Population.] *Vestnik Akademii Meditsinskikh Nauk SSSR* 5: 81-83, 1981, Russian.

The relationship between smoking and other risk factors of ischemic heart disease (IHD) has been studied in representative samples of the general male population of Moscow, aged 40 to 59 years. Forty-six percent of 210 clinically examined men were smokers. There were 38.4 percent of smokers among 65 men with arterial hypertension (160/95 mm Hg) vs. 49.6 percent among 145 men without hypertension. A trend is noted toward increase of diastolic pressure with the increased incidence of smoking. Simultaneously, the average cholesterol and triglyceride blood levels increased significantly. However, these two risk factors were higher in nonsmokers than in smokers or ex-smokers. The IHD incidence was about equal in all three groups. Not a single case of myocardial infarct or intermittent claudication (IC) was detected among nonsmokers, and IC incidence was lower in ex-smokers than in smokers. Long-term disability was more frequent among smokers than among nonsmokers or ex-smokers. As the result of appropriate smoking preventive measures, 20.9 percent of 316 subjects quit smoking and 25.9 percent reduced by 25 percent the number of cigarettes smoked/day.

82–0743. Phillips, N. R.; Havel, R. J.; Kane, J. P. **Levels and Interrelationships of Serum and Lipoprotein Cholesterol and Triglycerides. Association With Adiposity and the Consumption of Ethanol, Tobacco, and Beverages Containing Caffeine.** *Arteriosclerosis* 1(1): 13-24, January-February 1981.

A study of white men and women, aged 20 to 64 years, from an industrial population found possible associations of serum and lipoprotein lipid levels with smoking. The population's consumption of cigarettes and other tobacco products was measured. Other factors examined included adiposity as well as the use of ethanol and beverages with caffeine. For both men and women, the serum level of triglyceride rose with the daily consumption of cigarettes. With serum cholesterol, there were positive partial coefficients for cigarette consumption, though statistically significant for older women and younger men only. Very low density lipoprotein triglyceride level increased with cigarette smoking. Very low density lipoprotein cholesterol level also rose with cigarette smoking, but was independent of all variables considered when controlled for very low density lipoprotein triglyceride. Small increases in low density lipoprotein cholesterol level with cigarette smoking appeared to reflect associated increases in very low density lipoprotein triglyceride. High density lipoprotein cholesterol level decreased with cigarette smoking even after adjustment for its inverse relationship with very low density lipoprotein triglyceride. (Auth. Abs. Mod.)

82–0744. Pryszmont, M. **Ocena Reoencefalograficzna Zmian w Naczyniach Mozgowych pod Wplywem Palenia Tytoniu. [Rheoencephalographic Evaluation of Smoking Effect on Cerebral Blood Vessels.]** *Polski Tygodnik Lekarski* 36(16): 549-551, April 20, 1981, Polish.

The effects of cigarette smoking on blood circulation in the brain were determined from rheoencephalographic (REG) and electrocardiographic (ECG) recordings of 30 healthy subjects of both sexes, aged 19 to 50 years. One half (15) of the subjects were smokers. The recordings were made before smoking with subjects at rest, during smoking of one cigarette, and were continued for 20 to 25 minutes after the end of smoking. At rest, the smokers had a faster average pulse (74.5 beats/min) than the nonsmokers (68.8) and a higher vascular tonus (index of tension in brain vessels). During smoking, the pulse became faster than at rest in both smokers (86.6/min) and nonsmokers (80.7/min), and the vascular tonus also increased in both groups. After smoking, the average pulse in both groups remained somewhat faster than the initial, even 10 minutes after the end of smoking. The same was true for the vascular tonus. The index of blood circulation in the brain decreased equally in both groups after smoking to a level lower than the one prior to smoking.

82–0745. Ramsdale, D. R.; Faragher, E. B.; Bennett, D. H.; Bray, C. L.; Ward, C.; Beton, D. C. **Preoperative Prediction of Significant Coronary Artery Disease in Patients With Valvular Heart Disease.** *British Medical Journal* 284(6311): 223-226, January 23, 1982.

A prognostic index for predicting significant coronary artery disease was established using multiple logistic regression analysis of clinical data from 643 patients with valvular heart disease who had undergone routine coronary arteriography before valve replacement. The index or equation obtained incorporated the presence of angina, a family history of ischemic heart disease, age, cigarette smoking habits, mitral valve disease, sex, and electrocardiographic evidence of myocardial infarction. The equation was validated using prospective data from 387 patients with valvular disease and shown to enable almost a third of routine coronary arteriograms to be omitted while maintaining 95 percent sensitivity for patients with coronary artery disease. Similar analysis of the more detailed prospective data produced a second discriminant function incorporating diastolic blood pressure, total cigarettes smoked in life, the severity of angina, family history of ischemic heart disease, age, current cigarette smoking habits, and the ratio of total to high density lipoprotein cholesterol. This method improved the discrimination between patients with and without coronary artery disease, allowing omission of 30 percent of routine coronary arteriograms with 100 percent sensitivity for patients with coronary disease and omission of 41 percent with a 96 percent sensitivity level. (Auth. Abs.)

82–0746. Raymond, T. L.; Delucia, A. J.; Bryant, L. R. **Failure of Chronic Cigarette Smoke Exposure To Alter Plasma Lipoproteins of Stumptailed Macaques (*Macaca arctoides*).** *Atherosclerosis* 41(1): 27-33, January 1982.

Long-term exposure to cigarette smoke did not appear to change plasma lipoprotein levels in stumptailed macaques, or *Macaca arctoides*. Twenty-one 8-14 kg adult male stumptailed macaques were fed a standard laboratory diet and divided into three groups. The high-dose group and low-dose group were exposed to cigarette smoke at the human equivalent of 3 packs and 1 pack per day, respectively, 7 days per week, for 3-5 years. Eight animals served as cage and sham controls. Peak blood carboxyhemoglobin (COHb) levels measured immediately after smoking showed levels of 0.5 ± 0.1 percent, 3.6 ± 1.0 percent, and 5.7 ± 2.8 percent for sham controls, low-, and high-dose smokers, respectively. Hemoglobin and hematocrit values were 2-7 percent higher for smoking groups, presumably as a consequence of chronically elevated COHb levels. No significant differences were seen in total plasma cholesterol and lipoprotein cholesterol concentration measured at four intervals over a period of 1 year. It is concluded from these data that, in animals fed a low-fat diet, chronic cigarette smoke inhalation fails to alter plasma lipoprotein levels. (Auth. Abs. Mod.)

82-0747. Samkoff, J. S.; Comstock, G. W. **Epidemiology of Pulmonary Embolism: Mortality in a General Population.** *American Journal of Epidemiology* 114(4): 488-496, October 1981.

The basic epidemiologic characteristics of fatal pulmonary embolism as it was recognized in the adult white population of Washington County, Maryland, from 1963 to 1975 are described. Demographic and personal characteristics considered are age, sex, marital status, educational level, adequacy of housing, cigarette smoking, and religious service attendance. There were 316 deaths with pulmonary embolism mentioned on the death certificate during the 12 years of this study. Death was attributed to pulmonary embolism in 55 instances (17 percent) and to other thromboembolic diseases in 41 cases (13 percent). Age and educational level were the only sociodemographic variables significantly associated with risk of fatal pulmonary embolism. Mortality rose logarithmically with age up to age 75. Persons with less than 8 years of schooling had the highest rates, but the association with educational level was not linear. Deaths with pulmonary embolism mentioned showed a significant trend with number of cigarettes smoked daily when ex-smokers were excluded, but this association did not hold for deaths due to pulmonary embolism. Heart disease and cancer were mentioned on the death certificates of persons dying with pulmonary embolism less often than on death certificates in general, casting doubt on an etiologic association. (Auth. Abs.)

82-0748. Snowden, C. B.; McNamara, P. M.; Garrison, R. J.; Feinleib, M.; Kannel, W. B.; Epstein, F. H. **Predicting Coronary Heart Disease in Siblings—A Multivariate Assessment. The Framingham Heart Study.** *American Journal of Epidemiology* 115(2): 217-222, February 1982.

The incidence of clinically recognized coronary heart disease and its relation to risk factors were studied for 596 sibships containing two or more siblings in the Framingham Heart Study cohort. Only the 186 pairs of brothers were considered in the multivariate analysis, since the rate of coronary heart disease in women was low. Multiple logistic regression was performed by using endpoints of coronary heart disease as the dependent variable for the younger brother of the pair. Age, Metropolitan relative weight, systolic blood pressure, total cholesterol, cigarette smoking, and endpoints of coronary heart disease for the older brother represented the independent variables. The incidence of myocardial infarction in the older brother was significantly related to myocardial infarction experience of the younger brother, even after the strong effects of total cholesterol, systolic blood pressure, and cigarette smoking were controlled. These findings suggested that family history of myocardial infarction may be an important independent predictor of myocardial infarction, and that familial aggregation for coronary heart disease may result from predisposition to disease, possibly genetic, that is not reflected in the measured levels of total cholesterol, systolic blood pressure, or cigarette smoking. (Auth. Abs.)

82-0749. Tirlapur, V. G.; Mir, M. A. **Nocturnal Hypoxemia and Associated Electrocardiographic Changes in Patients With Chronic Obstructive Airways Disease.** *New England Journal of Medicine* 306(3): 125-130, January 21, 1982.

Smoking appeared to be a health hazard for patients with hypoxemia in chronic obstructive airways disease (COAD). Nocturnal hypoxemia's effects on the heart were examined by recording electrocardiographic tracings and arterial oxygen saturation (SaO_2) at night in smoking and nonsmoking patients. The actions of the auricles and ventricles were measured by QT, ST-T, and PR electrocardiographic intervals. Marked hypoxemia (mean basal $SaO_2 < 80$ percent) in COAD was found to produce cardiac arrhythmias and ST-T depression, a prolongation of the QT interval, and a shortening of the PR interval. Oxygen therapy reversed the changes, but failed to correct the prolonged QT interval in smokers. Although patients were discouraged from smoking, some may have smoked surreptitiously before monitoring began, and it is not known how long smoking's effects on a tracing persist after smoking stops. It is therefore suggested that for those who continue to smoke, oxygen therapy does not provide its full benefits and does not prevent sudden death from dysrhythmia.

82-0750. Tweed, W. A. **Does Cigarette Smoking Cause Coronary Heart Disease?** *Canadian Medical Association Journal* 126(2): 102, 104, 107, January 15, 1982.

Research studies investigating the causal relationship of cigarette smoking to coronary heart disease are reviewed. The results of all the data show that: (1) there has been a consistent and dose-related increase in the risk of coronary heart disease in cigarette smokers in most populations; (2) the rates of death from coronary heart disease in those who quit smoking are half those in persons who continue to smoke; (3) smoking is most deleterious when combined with other risk factors, especially in genetically high-risk populations; and (4) inconsistencies in the evidence cast doubt on a simple causal relationship between cigarette smoking and coronary heart disease. It is recommended that investigations into the biologic and pathologic effects of smoking be continued to determine the interactive mechanisms between the toxic components of cigarette smoke and the individual.

82–0751. Vandenbroucke, J. P.; Hofman, A.; Ball, K.; Kringlen, E. **Cholesterol, Smoking, and the Oslo Study. (Letters).** *Lancet* 1(8270): 503-504, February 27, 1982.

The Oslo trial (*Lancet* 2(8259): 1303-1310, December 12, 1981) which found that dietary change and cigarette reduction can significantly reduce the incidence of coronary heart disease in high-risk middle-aged men is criticized for faulty design. Three points are raised: (1) since intervention was on both factors for smokers, it is hard to see how one can take advantage of the key feature of randomization and analyze nonrandomized subgroups at the same time; (2) the researchers tried to disentangle the smoking—diet factors by stratification according to the amount of change in the factors upon which they intervened, which is a hazardous method; (3) the conclusion that 60 percent of the preventive effect is due to serum cholesterol reduction and only 25 percent to reduction of cigarette smoking seems farfetched when the total number of prevented cases is 17. Another letter commenting on the Oslo study and a study in Trappist monks (*Lancet* 2(8259): 1310-1313, December 12, 1981) is in complete agreement with their findings. It is suggested that the evidence from such studies merits a nationwide campaign to control diet, hypertension, and cigarette smoking. However, a third letter states that it is too easy to ascribe the decreased mortality to changes in diet and smoking. Clinical and primary prevention trials must control for "unspecific" psychological factors if they are to yield safe conclusions. The "Hawthorne effect" is cited. So long as you do something to patients they will improve. It is argued that the nutrition and smoking hypothesis is not confirmed since the medical literature is in disagreement, and there is no established proof that cigarette smoking is causally related to coronary heart disease. See also 82-0454.

82–0752. Voors, A. W.; Srinivasan, S. R.; Hunter, S. M.; Webber, L. S.; Sklov, M. C.; Berenson, G. S. **Smoking, Oral Contraceptives, and Serum Lipid and Lipoprotein Levels in Youths.** *Preventive Medicine* 11(1): 1-12, January 1982.

Data from a study of cardiovascular risk factors in youth found that smoking may have suppressed the α-lipoprotein cholesterol levels in girls but not in boys. Serum lipids and lipoproteins, anthropometry, and other risk factors were examined in 3,145 children aged 8 to 17 (65 percent white, 35 percent black), and a smoking questionnaire (including questions on oral contraceptive use for older girls) was administered. Smoking history was validated with plasma thiocyanate levels. Smoking began early and increased at age 14 and above. The coefficient of partial correlation between smoking and fasting serum α-lipoprotein (controlling for age) was significantly negative only in white girls ($p<0.01$). Use of oral contraceptives was more frequent among smokers, but is unlikely to account for the smoking effects. Fourteen white and seventeen black girls reported use of oral contraceptives which was associated with an increase in β-lipoprotein cholesterol ($p<0.0001$, $p<0.05$) and triglycerides ($p<0.0001$, $p<0.0005$), and with a decrease in α-lipoprotein cholesterol ($p<0.0001$, $p<0.01$) for whites and blacks, respectively. Serum lipid or lipoprotein response to these stimuli may be influenced by race. (Auth. Abs. Mod.)

82–0753. Weinblatt, E.; Goldberg, J. D.; Ruberman, W.; Frank, C. W.; Monk, M. A.; Chaudhary, B. S. **Mortality After First Myocardial Infarction. Search for a Secular Trend.** *Journal of the American Medical Association* 247(11): 1576-1581, March 19, 1982.

Two earlier studies of prognosis of coronary heart disease among men enrolled in the Health Insurance Plan (HIP) of Greater New York in the 1960s and 1970s made it possible to examine whether prognosis had improved over this 10-year period and to examine possible influences or characteristics such as smoking. The new comparison involved 1,133 men aged 35 to 64 years who had survived a first acute myocardial infarction (MI) and were followed up for mortality after a baseline examination. There was a decline in the proportion of cigarette smokers from the 1960s group to the 1970s group, a decline consistent with the national trend in that time period. There appeared, however, to be little effect on mortality for the 1960s or 1970s in relation to cigarette-smoking habits given at baseline. In both periods, a high proportion of men who had smoked cigarettes at the time of acute MI stated they had quit smoking before the baseline date. The analyses showed no difference in long-term prognosis between patients in the two decades. The observations in this population suggest that any contribution of improved medical care to the nationally observed secular decline in mortality from coronary heart disease in the time period studied was probably restricted to the acute stage of myocardial infarction. (Auth. Abs. Mod.)

82–0754. Wennmalm, A. **Interaction of Nicotine and Prostaglandins in the Cardiovascular System.** *Prostaglandins* 23(1): 139-144, January 1982.

Studies have indicated an inhibitory effect of cigarette smoke or of nicotine on cardiac and vascular PGI_2-formation. As these concordant results were obtained with a variety of techniques, it seems that smoking does interfere with vascular formation of PGI_2. The mechanism behind this inhibition most likely involves inhibition of cyclo-oxygenase. For some unknown reason, thromboxane formation in platelets is not inhibited by nicotine. If smoking decreases vascular PGI_2 but not platelet thromboxane formation, as indicated by experimental results, it is obvious that the aggregatory state of platelets may be displaced in a pro-aggregatory direction. The question whether such a mechanism constitutes the biochemical link between smoking and cardiovascular disease deserves further attention. (Auth. Abs.)

See also, **82-0682, 82-0795, 82-0841**

PREGNANCY AND INFANT HEALTH

82-0755. Beeley, L. **Adverse Effects of Drugs in the First Trimester of Pregnancy.** *Clinics in Obstetrics and Gynaecology* 8(2): 261-274, August 1981.

Smoking as well as a wide variety of drugs are discussed for their possible effects on pregnancy. Types of drugs mentioned include cytotoxic drugs, alcohol, anticonvulsants, anticoagulants, lithium, sex hormones, antimicrobial drugs, anesthetics, anti-emetics, psychotropic drugs, sedatives and hypnotics, and others. Maternal smoking has been associated with reduced birth weight, increased perinatal mortality, and perhaps a greater risk of spontaneous abortion. Although the smoke constituent responsible for smoking's possible effects has not been determined, one possibility is fetal hypoxia caused by carbon monoxide. Some evidence suggests that nicotine might be of more importance. Women should be advised to quit smoking while pregnant, but nicotine chewing gum should not be used to help women to quit.

82-0756. Beeley, L. **Drugs and Breast Feeding.** *Clinics in Obstetrics and Gynaecology* 8(2): 291-295, August 1981.

A wide variety of drugs, including nicotine, which might harm a baby if present in the mother's milk during breastfeeding, are listed, and possible effects noted. A daily consumption of over 20 cigarettes may reduce lactation and lead to jitteriness in the baby. The quantity of a drug a baby is exposed to by breastfeeding is affected by the drug concentration in the milk during feeding, the amount of milk the baby consumes, the degree of drug absorption from the gastrointestinal tract, and the baby's ability to eliminate the drug.

82-0757. Cunningham, D. G.; Garn, S. M.; Johnston, M.; Ridella, S.; Petzold, A. S. **Smoking and Pregnancy. (Letters).** *American Journal of Diseases of Children* 136(1): 82, January 1982.

The assumption of a cause-and-effect relationship between maternal cigarette smoking and lower infant Apgar scores (*American Journal of Diseases of Children* 135(6): 503-506, June 1981) is questioned. It is felt that if this hypothesis is adopted, then any variables which might have a bearing on the hypothesis must be examined. Factors such as alcohol ingestion, illegitimacy, nutritional status, and coexisting illness should be considered as well as social factors that contribute to and are the result of tobacco addiction. In reply it is stated that several hundred articles could be cited which attest to the effects of maternal smoking, and personal observations showed that neonates of smoking mothers are smaller and more often premature, even at constant socioeconomic status. Admittedly, it is not yet known what products of tobacco combustion result in fetal distress, placental defects, increased hemoglobin and hematocrit levels in the newborn, lower Apgar scores, and excessive number of low mental and motor scores on the Baylor test. However, results of investigations do show a dose—response relationship between packs per day and undesirable pregnancy outcomes. See also 81-1885.

82-0758. Deibel, P. **Effects of Cigarette Smoking on Maternal Nutrition and the Fetus.** *Journal of Obstetric, Gynecologic and Neonatal Nursing* 9(6): 333-336, November-December 1980.

Available evidence supports the importance of including facts about smoking and pregnancy in all teaching to women in the childbearing years. The incidence of low birth weight infants is possibly almost twice as high among smoking mothers, and these low birth weight babies are known to be at risk on several counts. Compromised utilization of vitamins and minerals is presented as a causative factor: calcium, vitamin B_{12}, and vitamin C levels are lower in smokers. Other vitamin levels may be affected. Poor nutrition and other factors connected with smoking may result also in stillbirths, spontaneous abortions, congenital anomalies, and cancer in the offspring. Nurses have a unique opportunity to contribute to maternal—child health by teaching and counseling about smoking in pregnancy. (Auth. Abs.)

82-0759. Hammer, R. E.; Mitchell, J. A.; Goldman, H. **Effects of Nicotine on Conceptus Cell Proliferation and Oviductal/Uterine Blood Flow in the Rat.** In: Glasser, S. R.; Bullock, D. W. (Editors). *Cellular and Molecular Aspects of Implantation.* New York, Plenum Press, 1981, pp. 439-442.

Administration of nicotine in adult rats was found to reduce intraoviductal conceptus cell proliferation and to reduce oviductal and uterine blood flow. Adult rats were given two injections a day of saline or nicotine during days 0-5 of pregnancy. Embryos were taken at chosen times from the oviduct or uterus. Their cells were dispersed, and the nuclei were counted. Oviductal and uterine blood flow were examined at day 4 in pseudopregnant rats at certain times after one injection of saline or nicotine. Nicotine treatment during days 0-5 reduced intraoviductal conceptus cell proliferation as shown by a decrease in the mean number of nuclei per conceptus at all times of retrieval. Conceptuses of nicotine-treated rats displayed a decreased cell number at entry into the uterus (on day 3). Cell number was still reduced into day 5. Oviductal and uterine blood flow were temporarily reduced at 10 minutes following administration of nicotine.

82–0760. Kaelin, A. C.; Cockle, S. M.; Cummings, A. J. **The Assay of Human Placental Aryl Hydrocarbon Hydroxylase Activity: A Comparison of Two Methods.** *British Journal of Clinical Pharmacology* 12(2): 260-264, August 1981.

Two methods used for assaying the aryl hydrocarbon hydroxylase (AHH) activity in human placentas are compared. One method used 2,5-diphenyloxazole (PPO) as a substrate, measuring the fluorescence of the PPO metabolite. The other method was a radiometric assay using tritiated zoxazolamine. Using the PPO method, it was found that AHH activity could vary throughout an individual placenta. The variation was smaller between aliquots of minced tissue than between aliquots of whole placenta. Using the PPO method, it was found that placental activities were greater in smoking mothers than in the only nonsmoking mother in which activity was found. A comparison of AHH activities in nine placental samples from smokers as measured by both the fluorimetric and radiometric assays showed a good correlation between the two methods' results. The blank values, however, expressed as a percentage of the experimental values, were much higher for the radiometric assay than for the fluorimetric assay. For homogenates where AHH activity is very low, the radiometric assay is apparently not sensitive enough, with the blank as the limiting factor.

82–0761. Kirkinen, P.; Roos, M. **Fetal Growth Retardation in Hypertensive Pregnancies.** In: Kurjak, A.; Rippmann, E. T.; Sulovic, V. (Editors). *Current Status of EPH Gestosis.* International Congress Series 534, 1981, pp. 353-359.

It is speculated that smoking may have a protective effect against hypertensive disease during pregnancy. In a study which examined hypertension as a factor in intrauterine fetal growth retardation (IUGR), maternal smoking was associated with 35 percent of cases of IUGR where no other causes were involved. However, smoking was associated with only 6 percent of the hypertension-related cases. Of cases of IUGR with a pathological cause, hypertension was the most common (23.9 percent). The value of diagnosis with ultrasonic techniques is discussed.

82–0762. Low, J. A. **Maternal Smoking in Human Reproduction.** *Canadian Journal of Public Health* 72(6): 390-393, November-December 1981.

Maternal smoking, through the effects of nicotine on catecholamines, reduces maternal utero-placental blood flow. In addition, there may be a parallel decrease of fetal blood flow. These events reduce maternal-fetal blood gas exchange and, in conjunction with the decreased oxygen transport due to high carboxyhemoglobin levels, lead to fetal tissue hypoxia and metabolic acidosis. Nicotine readily crosses the placenta with a higher concentration and slower disappearance from the fetal compartment. It is postulated that nicotine stimulates the fetal autonomic nervous system with the release of catecholamines. Maternal smoking affects reproduction by raising perinatal mortality and retarding fetal growth. Current evidence suggests an association between maternal smoking and minor handicaps or delay of motor, cognitive, or language development. Maternal smoking is also associated with congenital anomalies such as encaphaly, cleft palate, cleft lip, and neural tube defects. A positive correlation exists between maternal smoking and fetal cyanide and thiocyanate concentrations, but the effect on the fetus is unknown.

82–0763. Naeye, R. L. **Factors in the Mother/Infant Dyad That Influence the Development of Infections Before and After Birth.** In: *Perinatal Infections.* Ciba Foundation Symposium 77 (New Series). Excerpta Medica, 1980, pp. 3-16.

Although several factors can influence fetal infections, so far there does not appear to be a relationship with the maternal use of tobacco. Other factors that have been examined include nutrition, coitus during pregnancy, the use of alcohol and drugs, and maternal age. However, cigarette smoking can reduce uteroplacental perfusion as well as oxygen transfer to the fetus, and may damage intrauterine blood vessels. Such effects can predispose mothers to abruptio placentae, placenta praevia, and large placental infarcts, but do not appear to affect the development or outcome of antenatal bacterial and viral infections.

82–0764. Scott, A.; Moar, V.; Ounsted, M. **The Relative Contributions of Different Maternal Factors in Small-For-Gestational-Age Pregnancies.** *European Journal of Obstetrics, Gynecology, and Reproductive Biology* 12: 157-165, 1981.

Maternal risk factors associated with intrauterine growth retardation in individual pregnancies and in the population as a whole were calculated. Smoking and hypertension without preeclampsia each trebled the individual risk, and made a substantial contribution to the overall population of small-for-gestational-age (SGA) babies. Moderate or severe preeclampsia gave a higher individual risk, but because of its low prevalence, the contribution to SGA pregnancies as a whole was low. In multiparous women the most important factor for the total population was the slow fetal growth rate demonstrated in previous pregnancies. Other less potent factors were low maternal height, weight, and weight gain in pregnancy. When adjustment had been made for maternal size, smoking, pathological factors, and siblings' birth weight, the effect of social class became insignificant. The remaining "causes" of SGA pregnancies are likely to be single factors of large effect such as major congenital abnormalities and uterine abnormalities. If the smoking factor could be prevented, the number of SGA pregnancies in this population would be reduced by about 40 percent. (Auth. Abs. Mod.)

82–0765. Socol, M. L.; Manning, F. A.; Murata, Y.; Druzin, M. L. **Maternal Smoking Causes Fetal Hypoxia: Experimental Evidence.** *American Journal of Obstetrics and Gynecology* 142(2): 214-218, January 15, 1982.

Cigarette smoking during pregnancy has been related to poor perinatal outcome. To investigate the changes in maternal and fetal arterial blood gases associated with smoking, five chronically catheterized pregnant rhesus monkeys were exposed to standard cigarette smoke (N=5) and nicotine-free herbal cigarette smoke (N=3). The only consistently reproducible and significant finding was a drop in fetal pO_2. The timing of the decline in pO_2 and the similar responses to standard and nicotine-free cigarette smoke make carboxyhemoglobin the most likely causative factor.

82–0766. U.S. Department of Health and Human Services. **A Technique for Rapid Epidemiologic Assessment—Nevada.** *Centers for Disease Control—Morbidity and Mortality Weekly Report* 31(5): 61-63, February 12, 1982.

Results of a Nevada birth survey demonstrate that certain types of potentially useful public health information can be obtained in a cost- and personnel-effective manner by a limited-sample telephone survey. A sample of 200 mothers who had delivered 2 to 5 months previously were surveyed by phone with a 1-page, standard grid, modular questionnaire. Only one mother contacted did not wish to participate. One important finding was that postpartum infections remain a frequently perceived problem, and deserve more intensive surveillance. It was also found that the infants of women who smoked during pregnancy (28.5 percent) weighed an average of 0.9 pounds less than those of mothers who denied smoking during pregnancy. The data collected on the exposure to alcohol, drugs, radiation, and smoking can be used to improve educational programs, particularly against smoking which apparently is still widespread in the study population in spite of the known health risks. Limitations of such a study include: (1) only persons with telephones who were at home during the day could be contacted, and (2) the small number of participants limits the kinds of analyses that can be done.

82–0767. Van Allen, M. I. **Fetal Vascular Disruptions: Mechanisms and Some Resulting Birth Defects.** *Pediatric Annals* 10(6): 31-50, June 1981.

Birth defects caused by vascular disruption of normal embryonic development are discussed and examples of some structural anomalies that are believed to be at least partly caused by vascular disruption are given. The discussion is arranged in units: (1) the uterine-placental unit, (2) the placental-fetal unit, and (3) the fetal unit. Heavy cigarette smoking appears to lead to a chronic reduction in the amount of placental blood flow available to the fetus resulting in small-for-gestational-age infants through nicotine-stimulated release of catecholamines, resulting in vasoconstriction of peripheral and uterine vascular beds. Nicotine and carbon monoxide in the maternal blood readily cross the placenta and may also cause direct damage to the fetus by vasoconstriction and anoxia. Pregnancies of women who smoke 10 or more cigarettes a day are complicated by increased spontaneous abortions and premature deliveries. Birth weight of their offspring is 0.5 to 1.0 pound lighter; the offspring can have postnatal growth deficiency, and their school performance is statistically poorer than that of the offspring of nonsmokers. These effects on the fetus would most likely be secondary to vascular insufficiency, fetal vasoconstriction, and anoxia as mediated by nicotine and carbon monoxide from maternal smoking.

See also, **82–0629, 82–0654, 82–0846**

OTHER DISEASES AND CONDITIONS

82–0768. Achkar, E. **Peptic Ulcer Factors Influencing Ulcer Occurrence.** *Abdominal Surgery* 23(5/6): V-VI, May-June 1981.

Factors that contribute to the pathogenesis of peptic ulcer are discussed. Included are environment, sex, heredity, psychosomatic factors, diet, smoking, coffee, alcohol, and the role of other disease. It appears that there is a 2:1 prevalence of peptic ulcer in smokers vs. nonsmokers; nicotine has no clear consistent effect on acid secretion; and cessation of smoking produces no increase in the healing rate of gastric ulcer. It is concluded that the pathogenesis of gastric ulcer is different from that of duodenal ulcer; drugs and stress play a more important role in gastric ulcer, whereas psychosomatic factors, heredity, and the presence of another chronic illness seem to play a more important role in duodenal ulcer.

82-0769. Axell, T.; Henricsson, V. **Leukoedema—An Epidemiologic Study With Special Reference to the Influence of Tobacco Habits.** *Community Dentistry and Oral Epidemiology* 9(3): 142-146, June 1981.

An epidemiologic study found that leukoedema was more common among users of tobacco than among nonusers. Among 20,333 individuals aged 15 years and above, examined in urban, suburban, and rural regions in the middle of Sweden, leukoedema was found in 48.9 percent. The sex difference, males 61.9 percent and females 35.9 percent, was highly significant ($p<0.001$). The condition was most prevalent in the age groups 25-34 and 15-24 years being 60.6 and 57.3 percent, respectively. Leukoedema was significantly more prevalent among individuals with some daily tobacco habit (60.0 percent) than among those without (36.3 percent) ($p<0.001$), being most prevalent among smokers. Leukoedema was most common among pipe smokers followed by cigarette smokers and snuff dippers. The lesion seemed to be related to the amount of smoking since it was most prevalent among heavy smokers, and the most clearly established lesions were found in this group. (Auth. Abs. Mod.)

82-0770. Bastiaan, R. J.; Reade, P. C. **The Prevalence of *Candida albicans* in the Mouths of Tobacco Smokers With and Without Oral Mucous Membrane Keratoses.** *Oral Surgery, Oral Medicine, Oral Pathology* 53(2): 148-151, February 1982.

Although tobacco smoking alters the pH of saliva and its oxidation-reduction potential, the present study found that the prevalence of the fungus *Candida albicans* in the oral cavity of 127 patients was not influenced by tobacco smoking. Other factors examined included sex, age, and oral mucous membrane keratoses. The finding of the fungus in swabs taken from the oral mucosa and saliva was not related to the presence of oral mucous membrane keratoses or to tobacco smoking. The prevalence of the fungus in the mouth was significantly greater in women than in men, as was its prevalence in the older age groups of women. (Auth. Abs. Mod.)

82-0771. Bock, F. G.; Repace, J. L.; Lowrey, A. H. **Nonsmokers and Cigarette Smoke: A Modified Perception of Risk. (Letters).** *Science* 215(4529): 197, January 8, 1982.

A previous article (*Science* 208(4443): 464-472, May 2, 1980) on the dangers of passive smoking is criticized. The exposure of passive smokers was stated to be equivalent to smoking 2 to 27 cigarettes of a very low-tar brand per day. The health consequences of smoking have been evaluated for smokers in general, but not for the 2 percent who smoke this particular low-tar brand. Thus the statement of equivalency is not considered valid. The original authors reply that an exposure—response relationship between tobacco smoke and risk on the basis of low-tar equivalents was not intended. However, comparison of passive smokers' exposure to that of low-tar cigarettes is appropriate; there is no safe level of cigarette smoke consumption, and the sidestream smoke of high-tar cigarettes is similar to the direct smoke of low-tar cigarettes. See also 80-1378.

82-0772. Bonini, S. **Smoking, IgE, and Occupational Allergy. (Letter).** *British Medical Journal* 284(6314): 512-513, February 13, 1982.

Confirmation of findings of an earlier report (*British Medical Journal* 283(6301): 1215-1217, November 7, 1981), that raised serum immunoglobulin (IgE) concentrations and increased risk of occupational allergy may represent another hazard to smokers, is presented. In a survey to assess damage induced by an occupational exposure to cosmetics, serum IgE levels were measured in 320 hairdressers and 201 controls. Lung function tests were also carried out. IgE concentration in the serum was correlated to age, presence of current or previous allergic symptoms, occupational exposure to cosmetics, and smoking. While total serum IgE was not influenced by age or cosmetic exposure, a history of allergy and smoking was significantly related to high IgE values ($p<0.05$). Statistical analysis showed that the effect of smoking on IgE levels was independent of the occurrence of clinical sensitization. Moreover, when allergic subjects were excluded, the 133 smokers still had higher IgE concentrations than the 182 nonsmokers (4.09 ± 1.56 vs. 3.69 ± 1.28 ln IgE; $p<0.05$). It is concluded that healthy subjects should be aware that smoking could increase their IgE levels. See 82-0507.

82-0773. Halonen, M.; Barbee, R. A.; Lebowitz, M. D.; Burrows, B. **An Epidemiologic Study of the Interrelationships of Total Serum Immunoglobulin E, Allergy Skin-Test Reactivity, and Eosinophilia.** *Journal of Allergy and Clinical Immunology* 69(2): 221-228, February 1982.

The effects of age, sex, and smoking habits on the interrelationships of total serum immunoglobulin E (IgE), allergy skin-test reactivity, and peripheral blood eosinophilia are reported for a general population sample from Tucson, Arizona. Allergy skin-test reactivity showed a direct linear relationship to total serum IgE, but this relationship was differentially affected by age and smoking habits. The frequency of positive skin-test reactivity for a given level of IgE was greater among females than that among males, but a comparison of female and male nonsmokers indicated that this difference was attributable to differences in smoking habits rather than sex. Subjects less than 16 and those over 45 years of age demonstrated reduced skin-test reactivity for a given level of IgE when compared with subjects 16 to 45 years of age. Restricting the analysis to nonsmokers diminished (but did not obliterate) this age effect. Those over 45 years of age (but not those less than 16) were shown to have reduced levels of IgE specific for Bermuda grass as compared with the 16 to 45 year age group. The skin of subjects less than 16 and those over 65 years of age was reduced in its capacity to respond to histamine. Thus the reduction in the skin-test reactivity of subjects less than 16 years of age appears to involve a reduction in their skin reactivity to mediators, whereas for those over 45 it is related to a reduction in IgE specific for common aeroallergens, and for those over 65 it is apparently related to both of these causes. The frequency of peripheral blood eosinophilia was also found to be directly related to total serum IgE. Skin test-negative subjects had the same frequency of eosinophilia as that in skin test-positive subjects at any given level of serum IgE. Males had higher rates of eosinophilia than did females for a given level of IgE, but this difference also became insignificant if the analysis was restricted to nonsmokers. The increase in eosinophilia among smokers as compared with nonsmokers with equivalent serum IgE levels implies that smoking may trigger immunologic reactions associated with eosinophilia. (Auth. Abs.)

82-0774. Kleinschmidt, E.-G.; Henning, L. **Die Schwelle des Elektrischen Geschmacks beim Diabetes Mellitus. [Electric Gustatory Threshold in Patients With Diabetes Mellitus.]** *Zeitschrift fuer die Gesamte Innere Medizin* 36(12): 407-411, June 15, 1981, German.

Comprehensive tests are described of electrically stimulated gustatory function in 82 patients with diabetes mellitus and 107 nondiabetic patients without any gustatory disturbance. The tongue was stimulated for 1 second with a unipolar electrode as cathode, and the threshold electric pulse was measured with an electrogustometer. The effects of age, sex, and smoking habit of the patients were determined for both groups. Generally, the stimulated threshold was found to be significantly higher in the diabetics than in the controls of the same sex and age (average 50 years). In diabetics, the threshold at the tongue's tip was higher than at the margins of the tongue, but the opposite relationship was found in nondiabetics. In both groups, the finding of a higher threshold in men than in women was correlated with smoking habits. The threshold was higher in smokers of more than 5 cigarettes/day than in nonsmokers. Forty percent of the males were smokers versus less than 20 percent of the females among the controls. The difference in thresholds between male and female diabetics was more pronounced, since none of the female diabetics were smokers.

82-0775. Maeda, K.; Harada, N.; Takamatsu, M. **Factor Analysis of Complaints of Occupational Cervicobrachial Disorder in Assembly Lines of a Cigarette Factory.** *Kurume Medical Journal* 27(4): 253-261, 1980.

Approximately 270 workwomen, engaged in assembly lines for cigarette rolling and packing, were evaluated for their work motions and postures which included repetitive use of the upper limbs and constrained postures of the trunk. Many of them suffer from Occupational Cervicobrachial Disorder, complaining of stiffness or languidness and pain in the neck, shoulders, and arms. Of this group, 117 received a health examination and completed a 58-item questionnaire, rating complaints such as stiffness and pain in the neck, shoulders, and arms; eyestrain; headache; and hand coldness. The questionnaire data were examined using factor analysis. The following conclusions were drawn about the four extracted factors of the complaints: (1) relatively heavy loads upon the left side of the upper limbs and trunk; (2) disturbances of the peripheral circulatory, sensory, and motor nervous functions; (3) mental stress and loads upon sense organs; and (4) loads from constrained postures of the trunk and lower limbs. Taking into account observations of postures and motions of the upper limbs of the workwomen in the assembly lines, factors 1, 3, and 4 appear to be the primary effects of the workloads; factor 2 is considered to be a secondary effect based on the pathophysiological processes of Occupational Cervicobrachial Disorder. (Auth. Abs.)

82-0776. Repace, J. L. **The Problem of Passive Smoking.** *Bulletin of the New York Academy of Medicine* 57(10): 936-946, December 1981.

Within recent years, substantial new evidence concerning the adverse health effects of passive smoking has emerged, leading to various attempts to deal with the problem. This new evidence indicates that the well-known health effects of smoking may also be suffered by nonsmokers who breathe tobacco-smoke-contaminated air. The health risks of passive smoking may include lung cancer as well as cardiovascular, respiratory, and symptomatic effects. Concentrations of tobacco smoke indoors are directly proportional to the smoker density and inversely proportional to the effective ventilation rate. Attempts to control smoking by ventilation are futile, requiring ventilation rates far in excess of what is economical, and are contrary to the current trend toward energy conservation in buildings. However, alternative measures which regulate or reduce the source have been proved effective. Measures can include restriction of smoking in public areas, separation of nonsmokers and smokers, and encouraging employees to quit smoking. (Auth. Abs. Mod.)

82-0777. Seffrin, J. R.; Grove, R. B. **Tobacco Use and Oral Health.** *Journal of School Health* 52(1): 59-62, January 1982.

Since both smoking and smokeless tobacco can damage oral health, it is important to inform students of the hazards through health education. In addition to the fairly well-known relationship of smoking to lung cancer, heart disease, and emphysema, smoking has also been associated with various oral cancers. Smokeless tobacco can lead to oral leukoplakia, which is considered a precancerous condition, and can also damage periodontal tissues and cause tooth abrasion. Sugar added to improve taste can promote dental caries, while nicotine can produce dependence. Chewers and dippers of smokeless tobacco may have socially unacceptable traits such as bad breath, discolored teeth, and a constant need to spit. Relevant health concepts that can be used with students include: regular cigarette smoking can result in serious dependency, all forms of tobacco use are harmful to present and future health, tobacco-induced diseases are often progressively debilitating and incurable, tobacco-caused oral disease can be disfiguring, and most oral disease in the United States can be prevented. Anxiety-producing educational strategies appear most effective when the final anxiety level in the learner is moderate, rather than high or low. The negative aspects of tobacco use should be explored first; and the anxiety level then reduced by addressing the more positive aspects, including the preventability of most disease and the role of personal responsibility for oral health. Recommendations for preventive actions may be given.

82–0778. Young, G. P.; St. John, D.J.B. **Smoking and Ulcer Healing. (Letter.)** *Gastroenterology* 82(1): 163, January 1982.

Data from an Australian study are cited as confirmation of research (*Gastroenterology* 80(6): 1451-1453, June 1981) which demonstrated a statistically significant adverse effect of smoking on ulcer healing with antacids. In a comparison of carbenoxolone sodium in positioned-release capsules (duogasterone) with placebo over 6 weeks, it was observed that ulcer healing occurred in 7 of 11 (64 percent) nonsmokers and 10 of 29 (35 percent) patients who were smoking at the time of entry into the trial. In the carbenoxolone-treated group, healing occurred in 5 of 6 nonsmokers and 7 of 14 smokers. Carbenoxolone appeared to promote healing by increasing mucosal resistance rather than by reducing gastric acidity. It was noted that smoking adversely affected carbenoxolone-induced healing, despite the different healing mechanism. Patients were advised to cease smoking, and ulcers healed in 6 of the 12 who ceased but in only 4 of 17 who did not. Thus, it seems apparent that smoking adversely affects healing not only in patients treated by an antacid but also in those treated with carbenoxolone, an effect which appears to diminish in patients who cease smoking. See also 81-1547.

See also, **82–0622, 82–0730, 82–0812**

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

82–0779. Ben-Sira, Z. **Latent Fear-Arousing Potential of Fear-Moderating and Fear-Neutral Health Promoting Information.** *Social Science and Medicine* 15E(2): 105-112, 1981.

The potential of fear-moderating and fear-neutral health information for arousing latent fears and consequently for generating a defensive response similar to that of manifest fear-arousing communications was studied. It was hypothesized that messages may have such a potential when they relate to a type of undesirable behavior (e.g., smoking) which is commonly believed to be linked with some incurable, frequently fatal consequences (e.g., cancer). The hypothesis gained support from an investigation carried out among a representative sample of Israeli Jewish adults, following televised antismoking and cancer screening campaigns. The data indicate (1) that a latent (not broadcast) fear-arousing content ("Smoking causes cancer") constitutes a central underlying factor of all manifest (broadcast) fear-moderating and fear-neutral content items; (2) that, in general, the salience of the latent content is greater than that of manifest items; however (3) the salience of both the latent and manifest contents decreases with the increase in health hazardous (undesirable) behavior (volume of smoking). Inferentially, data conform to conclusions from previous studies, which indicated that the arousal of distress due to the threat of a disease may generate a defensive rather than a coping response unless accompanied by a perception of feasible and efficacious ways of coping. Due to the underlying centrality of a latent threatening perceived reality, any message relating to the undesirable behavior may arouse latent fears, even if the formulations are in neutral or reassuring terms. Thus a message may result in a defensive response, independent of its formulation, unless it is efficacious in disproving latent, commonly shared threatening beliefs. (Auth. Abs.)

82–0780. Biener, K. **Genussmittel und Drogen der Innerschweizer Jugend. [Stimulants and Drugs, Consumption by Youth of Central Switzerland.]** *Schweizerische Rundschau fuer Medizin (Praxis)* 70(41): 1783-1786, October 6, 1981, German.

Secondary school students from various districts of the Uri Canton, and apprentices of the largest industrial enterprise in the Canton were interviewed about their smoking habits. Among the 576 students, aged 14 to 16 years, 28 percent of the boys and 24 percent of the girls were regular cigarette smokers, and 5 and 2 percent, respectively, smoked 20 or more cigarettes/day. Thirty-one percent of all students in that group had tried smoking before they were 10 years old. Three-quarters of the students had their first cigarette with a friend. Most of them had unpleasant recollections of their first smoke. Only 13 percent of the girls, but 27 percent of the boys, were found to be regular smokers in a sample of 236 students, aged 16 to 18 years. In the sample of 106 apprentices, 44 percent of the boys but only 5 percent of the girls were regular smokers.

82–0781. Brook, J. S.; Whitman, M.; Gordon, A. S. **Maternal and Personality Determinants of Adolescent Smoking Behavior.** *Journal of Genetic Psychology* 139(2): 185-193, December 1981.

The interrelationship between maternal determinants and personality attributes of male tobacco users was studied. Subjects were from white inner-city neighborhoods of Brooklyn, New York, where the main supporters of the family were in semiskilled or skilled occupations with an average education between ninth and eleventh grade. The 39 mothers and their 39 sons (16 to 20 years of age) were interviewed separately about their attitudes and the mother's child-rearing practices. The sons also responded to questions concerning their personality characteristics. In general, adolescents who identified with mothers who smoked were most likely to smoke themselves. Additionally, it was found that adolescent tobacco use depends on the coexistence of certain personality predispositions and maternal conditions. Thus, with regard to personality factors, male smokers tended to be less in control of their impulses, less responsible and autonomous, more rebellious, and more likely to engage in interpersonal aggression. With respect to the coexisting maternal conditions, the mothers of smokers tended to be less traditional and affectionate, and less likely to serve as models for their sons. (Auth. Abs. Mod.)

82-0782. Bryson, R.; Dicken, C. **Smoking Behavior of Scientists.** *Psychological Reports* 49(1): 55-62, August 1981.

Academic biologists, chemists, mathematicians, and psychologists were surveyed to examine smoking rates among the various groups of scientists and to compare publication differences associated with smoking status. Smoking rates among these groups of scientists were, in general, lower than national averages for all groups considered except female psychologists. Rates for biologists, chemists, and mathematicians were, for both sexes, exceptionally low. In four of five science samples, women were more likely to smoke than their male colleagues. Most of the difference was due not to more women having started smoking, but to fewer women smokers having stopped. When all institution levels were combined, the median articles for smokers was 41, for former smokers, 39, and nonsmokers, 38. (Auth. Abs. Mod.)

82-0783. Canada Health Survey. **The Health of Canadians. Report of the Canada Health Survey.** Ottawa, Canada, Statistics Canada, Catalogue 82-538E, 1981, 237 pp.

A wide variety of topics relating to the health of Canada's population are addressed including tobacco consumption, the use of alcohol, physical activity and fitness, the use of seatbelts, immune status, health problems and disability, emotional health, blood pressure, blood biochemistry, and health services and medication. Tabular data are included for each area discussed. The problem of smoking is reviewed, listing factors such as age, sex, region, major activity, social status (education, income, and occupation), exposure to smoking risks, the relationship between smoking and drinking, and physical health status. Smoking-related statistics are also included under other topics; for example, physical activity and fitness includes data on fitness level and mean estimated maximum rate of oxygen consumption in milliliters per kilogram-minute, by sex and type of cigarette smoker. Health services and medication lists females who use birth control and hormone pills, by age and type of smoker.

82-0784. Coetzee, A. M. **Die Rookgewoonte by en Gesondheid van Dokters Wat Aan die Universiteit van Pretoria Gegradueer Het. [The Smoking Habit Among and Health of Doctors Who Have Graduated at the University of Pretoria.]** *South African Medical Journal* 60(1): 31-34, July 4, 1981, Afrikaans.

All medical graduates of the University of Pretoria were sent a questionnaire about their health status and smoking habits, covering the period from 1947, when the first class qualified, to the end of 1975. Of the 2,636 doctors approached, 2,080 (79 percent, 1,878 men and 202 women) replied. The male study group comprised 656 smokers, 554 ex-smokers, and 668 nonsmokers, of whom 86 percent were of Afrikaans extraction. There was a significant difference in the average age between the three groups. Of the men 35 percent still smoke; 70 percent of these smoke more than 15 grams of tobacco a day. Replies to the questions "Do you suffer from (a) ischemic heart disease, (b) obstructive airways disease, or (c) any other disease or disability which requires or required treatment?" indicated, after correcting for age, that each disease category occurred significantly more often in male smokers and ex-smokers than in nonsmokers. The difference in heart disease was also significant when the reported abnormalities in ECGs of about 70 percent in each group were compared. (Auth. Abs. Mod.)

82-0785. Dawley, H. H., Jr.; Morrison, J.; Carrol, S. **The Effect of Differently Worded No-Smoking Signs on Smoking Behavior.** *International Journal of the Addictions* 16(8): 1467-1471, 1981.

Positively vs. negatively worded "no smoking" signs were investigated in an attempt to determine the effectiveness of controlling smoking in a hospital setting. Unobtrusive measurements of smoking behavior were taken in the main lobby before and after the placement of positively and negatively worded "no smoking" signs. The first phase of the experiment assessed the baseline smoking rate in the lobby as a control condition. Following baseline, the ashtrays were removed for the two experimental conditions. Negative signs were installed and the smoking rate again assessed. Following that, the positive signs were put up and the proportion of smokers noted. Apart from the installation of signs and the removal of ashtrays, there were no attempts to enforce a no-smoking policy during the two experimental conditions. Data for each of the three conditions were based on six observations each. The proportions of smokers under the three phases of the experiment are given in tabular form. It can be seen that there was a reduced incidence of smoking in both experimental sign conditions compared to baseline. Further, it appears that the positive signs were more effective than the negative signs. The results clearly showed that there was less smoking under the experimental sign condition. Positively worded signs appeared to produce better compliance than negatively worded signs. (Auth. Abs. Mod.)

82-0786. Epstein, L. H.; Ossip, D. J.; Coleman, D.; Hughes, J.; Wiist, W. **Measurement of Smoking Topography During Withdrawal or Deprivation.** *Behavior Therapy* 12(4): 507-519, September 1981.

Smokers apparently change their smoking behavior when cigarette nicotine levels are changed but not when experiencing withdrawal or deprivation. Smoking behaviors were measured during two methods for changing serum nicotine, providing subjects with high- and low-nicotine cigarettes, or varying intercigarette intervals. The results showed adjustments in smoking behaviors, including puffs, cigarette duration, and puff duration, when cigarettes that vary in nicotine delivery were used, but not when serum nicotine was modified by withdrawal (gradual decrease of the number of cigarettes smoked) or deprivation. These results suggest inhalation patterns of individual cigarettes may not be sensitive to fluctuations in serum nicotine, while they are sensitive to cigarettes that vary in nicotine yield and draw resistance. (Auth. Abs. Mod.)

82–0787. Finau, S. A.; Stanhope, J. M.; Prior, I.A.M. **Kava, Alcohol and Tobacco Consumption Among Tongans With Urbanization.** *Social Science & Medicine* 16(1): 35-41, 1982.

The prevalence of kava (*Piper methysticum*), alcohol, and tobacco consumption in urban and rural areas of the Tonga Islands is described. Current kava consumption was males 48 percent and females 1 percent, with prevalence significantly higher among rural males. Current alcohol consumption was restricted almost exclusively to the urban male population. Tobacco consumption also showed a significant male predominance. There was a significantly higher total tobacco consumption in the urban population. Urban smokers smoked more manufactured cigarettes, while rural smokers preferred "European" or "Tongan" roll-your-own tobacco. Concordance of the three habits was evident among males, with 27 concordant positive (expected = 5.98) and 46 concordant negative (expected = 6.60). It appears that kava, a traditional Pacific beverage, may have lost ground to alcohol as urban Tongans adopt a more cosmopolitan lifestyle. Kava, if proved relatively harmless, may be promoted as a less unhealthy alternative to tobacco and alcohol. (Auth. Abs. Mod.)

82–0788. Friedman, G. D.; King, M.-C.; Klatsky, A. L.; Hulley, S. B. **Characteristics of Smoking-Discordant Monozygotic Twins.** In: Gedda, L.; Parisi, P.; Nance, W. E. (Editors). *Twin Research 3. Part C: Epidemiological and Clinical Studies.* New York, Alan R. Liss, Inc., 1981, pp. 17-22.

Discordant smokers of monozygotic twin pairs were unlikely to smoke more than one pack per day, usually smoked low-tar cigarettes, and started smoking at a later age. These smokers were more apt to regularly drink alcohol, coffee, cola, smoke marihuana, take estrogens, and add salt to their food. Tranquilizer use was reported less often by the smokers. The nonsmoking twins tended to have more formal education, exercise more, and be more concerned with physical fitness. The smokers were slightly taller and thinner on the average, and lefthandedness was more frequent. Compared with their nonsmoking counterparts, smoking twins show less increase in coronary heart disease than the general population.

82–0789. Greitemeyer, M. **Zur Risikobereitschaft von Rauchern und Nichtrauchern. [Risk Taking Readiness by Smokers and Nonsmokers.]** *Die Medizinische Welt* 32(46): 1730-1733, November 13, 1981, German.

Hypothetical differences between smokers and nonsmokers regarding their attitude toward unspecified risks and the health risks of smoking were studied in a representative sample of West German college students, 113 nonsmokers and 122 regular cigarette smokers (who had smoked at least 5 days per month during the past 6 months). The students, 62 percent of them male, average age 22.9 years, were surveyed by appropriate questionnaire. Statistical analysis showed a general trend toward a higher readiness of risk taking by smokers compared with nonsmokers. Significantly, the smokers are more impulsive in health areas and more willing to take health risks than nonsmokers, while nonsmokers are more strongly health-oriented than smokers. Also statistically significant was the difference between smokers and nonsmokers in their assessment of the health risks of smoking. Specifically, smokers estimated the risks of lung cancer, laryngeal cancer, chronic bronchitis, circulatory disorders, and cirrhosis of the liver to be lower for people who smoke than did the nonsmokers.

82–0790. Goldberg, S. R.; Spealman, R. D. **Maintenance and Suppression of Behavior by Intravenous Nicotine Injections in Squirrel Monkeys.** *Federation Proceedings* 41(2): 216-220, February 1982.

Nicotine appears to be a contributing factor in maintaining cigarette smoking, but experimental evidence for its reinforcing effects is scarce. Indeed, it has been suggested that in some situations nicotine may have noxious properties, which limit smoking behavior. These ideas were explored by comparing the effects of intravenous injections of nicotine on the behavior of squirrel monkeys under two experimental procedures. Under a fixed-interval schedule of nicotine self-administration, responding was well maintained by injections of 30-300 μg/kg of nicotine. Nicotine-maintained responding could be reduced by presession treatment with the nicotine antagonist, mecamylamine, or by substitution of saline for nicotine. In a second experiment, responding was maintained under a two-component fixed-ratio schedule of food presentation in which responses during one component (punishment component) also resulted in injections of 10-30 μg/kg of nicotine. Nicotine markedly suppressed responding during the punishment component but not during the alternating nonpunishment components. The suppressant effects of nicotine could be reversed by presession treatment with either mecamylamine or the antianxiety drug chlordiazepoxide, or by substitution of saline for nicotine. Nicotine had pronounced effects both as a reinforcer and as a punisher; the nature of the effects depended on the schedule under which nicotine was administered. (Auth. Abs.)

82–0791. Gonzalez, C. A. **A Comparison of the Personality Characteristics of the Smoker and the Ex-Smoker.** Doctoral Dissertation, United States International University, School of

Human Behavior, San Diego, University Microfilms International 81-17019, 1981, 107 pp.

Differences between the personality characteristics of 30 smokers and 30 ex-smokers were examined to identify any consistent traits related to successful cessation of smoking which might prove useful in programs for the reduction or cessation of smoking. The two groups were tested by the Sixteen Personality Factor Questionnaire, designed to measure the main dimensions of personality; the Internal-External Locus of Control Scale, designed to measure a respondent's expectations of how much rewards or punishments are dependent or independent of individual behavior; and the Smoking Survey, a questionnaire on cigarette smokers' needs, habits, and experience. Results indicate that smokers have expressive, adventurous, impulsive, carefree, and tough-minded personalities, and are more likely to be group-dependent and "joiners." Ex-smokers appear to be sober, reflective, and cautious; also more sensitive, astute, and self-sufficient, preferring to make their own decisions. Smokers tend to find smoking a pleasure and to smoke when they feel relaxed, while ex-smokers smoke for arousal and to reduce negative effect, and are stimulated by the sensorimotor pleasure of smoking rituals. It may be therapeutically useful to differentiate which individuals among people who still smoke maintain characteristics generally associated with ex-smokers.

82-0792. Griffiths, R. R.; Henningfield, J. E. **Experimental Analysis of Human Cigarette Smoking Behavior.** *Federation Proceedings* 41(2): 234-240, February 1982.

A laboratory setting that permits relatively unobtrusive measurement of naturalistic cigarette smoking behavior in humans was used for studies in which volunteer subjects participated in daily sessions lasting up to 3 hours. The first study showed that the intervals between consecutive cigarettes were relatively stable, both within and across sessions. An analysis of puffing for a single cigarette revealed that as the cigarette was smoked, interpuff intervals tended to become longer while puff durations became progressively shorter. The second study showed that cigarette deprivation increased the tendency for subsequent smoking as reflected in latency to smoke and number of cigarettes and puffs. In the third study no change in smoking behavior was detected when subjects were informed that they would subsequently be exposed to a period of cigarette deprivation. The final study demonstrated the responsiveness of cigarette smoking to a form of dose manipulation: decreasing the concentration of tobacco smoke delivered by using ventilated cigarette holders produced increases in the rate of puffing and total numbers of puffs. These studies demonstrate the sensitivity of this preparation and suggest that it may be valuable for exploring a range of pharmacological and behavioral variables that control human cigarette smoking. (Auth. Abs.)

82-0793. Griffiths, R. R.; Henningfield, J. E.; Bigelow, G. E. **Human Cigarette Smoking: Manipulation of Number of Puffs per Bout, Interbout Interval and Nicotine Dose.** *Journal of Pharmacology and Experimental Therapeutics* 220(2): 256-265, February 1982.

In a residential research ward, cigarette smoking was studied during 12-hour daily sessions in 7 volunteer human subjects with histories of regular smoking. Puff-to-puff delivery of cigarette smoke was held relatively constant by instructing and monitoring subjects in taking uniform puffs and by specifying and timing the duration of holding the smoke in the lungs (5 seconds) and the duration of the interpuff interval (25 seconds). When a modest response requirement of riding a stationary exercise bicycle for 1 minute was required before each smoking bout consisting of 8 uniform puffs, stable patterns of smoking emerged in which smoking bouts were evenly spaced within and across sessions. When the number of puffs per bout was experimentally manipulated both across and within sessions, increases in puffs per bout resulted in increases in interbout intervals. When the interbout interval was experimentally manipulated both across and within sessions and the number of puffs per bout was free to vary, increases in interbout interval resulted in increases in puffs per bout. When both nicotine dose (0.2-1.6 mg per cigarette) and puffs per bout were varied across sessions in the same experiment, changes occurred in interbout interval in response to manipulation of puffs per bout but not nicotine dose. Although nicotine dose did not influence interbout interval or puffs per hour, subject ratings of cigarette strength tended to increase with dose. (Auth. Abs.)

82-0794. Hay, D. R. **Cigarette Smoking, 1981 New Zealand Population Census. (Letter).** *New Zealand Medical Journal* 95(700): 52-53, January 27, 1982.

Although there has been a decline in the percentage of both male and female smokers in New Zealand since 1976 (425,000 have quit), census reports for 1981 indicate that in most age groups, those who do smoke are heavier smokers. These findings are similar to those of the United States, and lower than for most European countries. Since 1976, the percentage of New Zealanders who smoke has declined from 40 to 35 percent for males, and from 32 to 29 percent for females. In general, however, the percentage of heavy smokers has increased in most age groups in women except for those aged 15 to 19, where there has been a drop from 25 to 23 percent of those who smoke. A comparison of the various 1981 age groups with those who were 5 years younger in 1976, shows a reduction in heavy smoking by both men and women except for those under 35 years. It is suggested that physicians, who have reduced their smoking dramatically, can provide positive help for pregnant women, women on the pill, and for the large numbers of heavy smokers who are high risk candidates for coronary heart disease and lung cancer, by emphasizing the benefits of smoking cessation. It is also noted that more smoking cessation programs should be made available.

82-0795. Hunter, S. M.; Baugh, J. G.; Webber, L. S.; Sklov, M. C.; Berenson, G. S. **Social Learning Effects on Trial and**

Adoption of Cigarette Smoking in Children: The Bogalusa Heart Study. *Preventive Medicine* 11(1): 29-42, January 1982.

A variety of social and environmental factors were found to affect the initiation of cigarette smoking among a study group of children and adolescents in a totally biracial community, Bogalusa, Louisiana. As part of a major program on cardiovascular risk factors in children, the objective of this cigarette smoking study was to collect reliable and valid data within a conceptual structure incorporating principles of diffusion—adoption and social learning. The principles can be construed as decisionmaking under uncertainty. The study tested one aspect of the diffusion adoption model at an early age in a community setting. During 1976-1977 a survey was conducted on 3,014 children, ages 8-17 years, to determine factors associated with trial and later adoption of cigarette smoking. Parental, sibling, and peer smoking behaviors were combined into one environmental scale. The scale was higher among children who tried smoking than among those who did not (1.44 vs. -0.99, $p<0.001$). Beliefs about social influences such as peer acceptance or parental approval contribute to current behavior after trial. The mean sources for a social reinforcement scale were 1.38 for current smokers, 0.87 for noncurrent smokers, and 0.69 for "never" smokers ($p<0.001$). Distinct race and sex differences were seen, which reinforce the impact of social learning on attitudes and beliefs toward cigarette smoking. White children demonstrated a greater parental effect while black boys and girls showed a greater peer and sibling effect. Thus, the number of people who smoke in a child's environment influences trial, while beliefs about the rewards of smoking influence current behavior. A better understanding of the diffusion—adoption principle should help in developing rational methods for intervention in cigarette smoking or prevention of cigarette smoking at an early age. (Auth. Abs. Mod.)

82–0796. Jackson, N.; Carlisi, J.; Greenway, C.; Zalesnick, M. **Age of Initial Drug Experimentation Among White and Non-White Ethnics.** *International Journal of the Addictions* 16(8): 1373-1386, 1981.

To assess ethnicity, drug use, and related attitudes, a 196-item questionnaire was developed in which respondents from four inner-city communities (Baltimore, Detroit, Cincinnati, and Providence) were asked to report the age at which they first tried any of the following: cigarettes, inhalants, alcohol, marihuana, barbiturates, codeine, methadone, cocaine, hallucinogens, amphetamines, Valium, and heroin. Comparisons in age of initial drug experimentation among ethnic groups and races were made. Results indicated significant differences in actual age of experimentation among ethnic groups, as well as differences in other general patterns relating to age of first drug use. Generally, cigarettes were the first substance tried with initial experimentation occurring at the junior high school level. Comparison of whites to nonwhites showed little difference in ages of initial drug experimentation, although cigarettes were first tried at a younger age by the White Different Group (parents of different ethnic backgrounds), and were tried at a significantly lower age than the average for the total population ($p<.0025$). It was concluded that ethnicity, more than mere race, was related to age of first use of drugs.

82–0797. Jacobson, B. **The Ladykillers. Why Smoking Is a Feminist Issue.** London, Pluto Press, 1981, 143 pp.

Women are the new smokers. While smoking rates among men have fallen continuously over the past decade, women have been taking up the habit. This is reflected in the increasing numbers of women dying from disease and cancer. The reasons why women take up smoking and the pressures on them to continue are discussed. The many social factors involved in women's smoking are sifted through, from starting to smoke in grade school as a display of emancipation, continuing to smoke as a way of being in control of emotions, then as an adult, afraid to stop for fear of gaining weight, and finally, failing attempts to quit from lack of confidence to carry through. The relationship between smoking and changes in the position of women is also examined, showing how women are being exploited by the tobacco industry. A final section, a guide to giving up, helps readers to understand their own smoking habit as well as giving practical advice on the various methods available. (Auth. Abs. Mod.)

82–0798. Jokiel, M. **Popular Conceptions and Misconceptions About Cancer.** In: Hobbs, P. (Editor). *Public Education About Cancer. Recent Research and Current Programmes.* UICC Technical Report Series, Volume 62, Geneva, 1981, pp. 1-15.

A survey of public attitudes and knowledge about cancer conducted among study populations in Warsaw, and Poland in general, found only a modest belief that cigarette smoking might be associated with cancer. The survey covered several topics, including the fear of cancer and confidence in treatment methods, knowledge of and behavior toward the prevention and early detection of cancer, and changes in public opinion over 14 years toward selected problems concerning cancer. It was found that 63 percent of respondents believed that cigarette smoking is a cancer risk. The opinion was less common among younger people, among men, among people with less education, and among cigarette smokers. Smoking was more common among men (78 percent) than among women (25 percent). It was suggested that people smoke due to insufficient awareness of smoking's dangers or because they do not accept the information. Antismoking campaigns should take these two attitudes into account.

82–0799. Kozlowski, L. T. **The Determinants of Tobacco Use: Cigarette Smoking in the Context of Other Forms of Tobacco Use.** *Canadian Journal of Public Health* 72(6): 396-401, November-December 1981.

All forms of tobacco consumption deliver nicotine to the central nervous system (CNS); however, the effect of the form of tobacco use on the chemical senses, and its social convenience, influences the choice of how the nicotine is delivered. Chewing tobacco and cigarettes may differ in their CNS effect and taste, but may be equivalent in their biological rewards. The current unfortunate bias for cigarettes (possibly the most toxic means of tobacco use) may be a result of cultural and psychosocial considerations rather than the nicotine bolus effect. Modes of tobacco use have changed over time; the antispitting laws of the late 1800s and early 1900s reduced the convenience of chewing tobacco, the then-preferred form. The ease of accommodation, such as cigarette use in an industrial society (faster than pipes), and the improvement of a portable source of fire influenced the popularity of the cigarette. The cigarette is the "fast food" of tobacco, and while cigarettes may not produce stronger addictions, their convenience has made it easier for more individuals to become addicted. It is rare to find tobacco used as an intoxicating drug in industrialized societies; thus, the "greater addictiveness" of cigarettes due to the nicotine bolus-delivery theory as opposed to sociocultural considerations is unlikely.

82–0800. Krishna, K. P. **Tobacco Smoking Among College Students.** *Indian Journal of Social Work* 41(2): 177-183, July 1980.

Psychosocial factors among college students, including demographic attributes of tobacco smokers, personality factors underlying smoking, reasons for continuing the habit, and attitude toward smoking, were investigated. Smokers who are unmarried, younger in age, and belonging to upper and middle economic strata show more personal problems. They suffer from adjustment problems as well as high levels of anxiety and neuroticism. Nonsmokers are more restrained, ascendent, sociable, and thoughtful. Mild and heavy smokers have similar attitudes towards smoking, and although they claim they can stop the habit, responses from the majority in these two groups indicate that they did not succeed in spite of their attempts. (Auth. Abs. Mod.)

82–0801. Langinvainio, H.; Kaprio, J.; Koskenvuo, M.; Tarkkonen, L. **Structural Analysis of Smoking, Alcohol Use, and Personality Factors in MZ and DZ Twin Pair Relationships.** In: Gedda, L.; Parisi, P.; Nance, W. E. (Editors). *Twin Research 3. Part C: Epidemiological and Clinical Studies.* New York, Alan R. Liss, Inc., 1981, pp. 23-35.

Dizygotic (DZ) cotwins of discordant pairs differed more in smoking and drinking habits than monozygotic (MZ) twins. In DZ twins, the twin who smoked and drank had done so longer and consumed more than the MZ twin who smoked and drank. In all concordant twin pairs, the smoking—personality correlation coefficient was strong. In MZ pairs, smoking and life dissatisfaction had statistically significant estimates of fit: smoking, extroversion, and neuroticism were statistically significant in DZ, but not in MZ, pairs. Alcohol and cigarette use were related. Neuroticism, extroversion, and experienced stress were more common among smokers than nonsmokers.

82–0802. Lavrova, I. G.; Orlov, Yu. M.; Malaya, E. V. **Vliyaniye Psikhologicheskoy Ustanovki na Kureniye, Sotsiometricheskogo Statusa, Tekhniki Obshcheniya i Potrebnosti v Obshchenii na Nikotinizm Studentov. [Effects of Mental Attitudes on Smoking of Students, and of Their Sociometric Status, Affiliation Modalities, and Affiliation Needs on Smoking.]** *Gigiena i Sanitariya* 8: 49-51, 1981, Russian.

Results are published of a survey of sociopsychological characteristics of the smoking and nonsmoking students, male and female, in their first through fifth year of college. Specially developed questionnaires and tests were administered. The attitude of students toward smoking is shown to have a decisive effect on their smoking; there were four times more students with positive than with negative attitudes among the smokers. Among the male students, however, this ratio is only 2:1, while among the female students it is higher than 5:1. The attitude toward smoking is greatly influenced by the sociometric factor: the male students with a high sociometric status have a higher probability of positive attitudes than those with a low status. Also, male students with a high status are more likely to be smokers than those with a low status. Probability of smoking of the subjects with a high need of affiliation (relating to others) is higher than of those with a low need. It is concluded that the characteristics of human relations are responsible for smoking because they determine attitudes toward smoking.

82–0803. LeCompte, W. A. **The Ecology of Anxiety: Situational Stress and Rate of Self-Stimulation in Turkey.** *Journal of Personality and Social Psychology* 40(4): 712-721, April 1981.

Twelve community behavior settings in Ankara, Turkey, were ranked with high agreement by 30 judges (average $r = .80$, $p < 0.001$) according to the amount of situational stress, defined as evaluative apprehension and uncertainty, generated by each setting. The rate of hand-to-face or body (self-stimulation) behavior was observed systematically in stressful compared with relaxed settings. Analysis by a stepwise multiple regression procedure showed higher rates in stressful settings, $F(1,587) = 9.33$, $p < 0.01$. This finding was successfully replicated 1 year after the original study with new samples of settings, observers, and observed individuals, $F(1,351) = 7.38$, $p < 0.01$. When smoking, sex, and the Smoking X Sex interaction are taken out of the variance in self-stimulation, both stress and the Stress X Smoking interaction remain significantly correlated with the criterion variable; smoking acted as a suppressor variable. The findings seem consistent with earlier studies which observed smoking and talking in patients and found smoking was more tied to person variance, whereas talking varied more across specific settings.

82–0804. Morison, J. B. **Smoking Habits of Winnipeg School Students, 1960-80.** *Canadian Medical Association Journal* 126(2): 153-154, January 15, 1982.

Four smoking surveys of Winnipeg students, grades 5-12, were conducted in 1960, 1963, 1968, and 1980. A comparison of the data showed that the proportion of students of all ages and both sexes who smoked regularly increased between 1960 and 1968 and decreased after 1968. The decrease was greater for boys. By 1980 more girls than boys in grades 7 to 12 smoked. Although in 1980 the overall proportion of students who smoked was lower, the relative risk of a student's smoking if either parent smoked was somewhat greater than in 1960. In both surveys, the incidence of maternal smoking appeared stronger than that of paternal smoking, especially for girls. In 1960, 73 percent of fathers and 50 percent of mothers smoked compared with 54 and 41 percent, respectively, in 1980. It was concluded that these findings were consistent with those of many recent surveys of the smoking habits of students.

82-0805. Mougne, C.; MacLennan, R.; Atsana, S. **Smoking, Chewing and Drinking in Ban Pong, Northern Thailand.** *Social Science & Medicine* 16(1): 99-106, 1982.

The unusual pattern of oral cavity and respiratory tract cancers in Northern Thailand led to this descriptive study of smoking, drinking, and chewing in a rural community in Chiang Mai Province. A high proportion of adults were smokers and both males and females had very high estimated lifetime tobacco tar exposures. Variation in additives mixed with tobacco in traditional cigars and differences in smoking may be related to different respiratory cancer patterns in males and females. Few manufactured cigarettes were smoked, mainly by young men and those of higher socioeconomic status. Betel nut chewing, found commonly in other parts of Southeast Asia, was found only among the old; but chewing of a fermented tea-leaf preparation was common in all age groups, and was generally accompanied by smoking. Drinking of alcoholic beverages was rare and infrequent. (Auth. Abs.)

82-0806. Ockene, J. K.; Benfari, R. C.; Nuttall, R. L.; Hurwitz, I.; Ockene, I. S. **Relationship of Psychosocial Factors to Smoking Behavior Change in an Intervention Program.** *Preventive Medicine* 11(1): 13-28, January 1982.

Indications are that stress levels and "psychosocial assets" sometimes affect the smoking behavior of adults. The relationship of stress and the psychosocial assets of adjustment, belief in personal control, and social support to the modification of smoking behavior was studied in a sample of 169 male smokers, aged 35 to 57, enrolled in a heart attack prevention program. Smokers were categorized as Continuing Successes, Recidivists, or Nonstoppers, depending on whether they stopped smoking and maintained cessation, stopped smoking and relapsed, or never stopped during the initial intervention phase. The participants were followed for 2 years. Variations in the following factors were used to significantly discriminate among the groups of smokers: stress, security, social support, number of cigarettes smoked at baseline and when smoking heaviest, and measures of belief in personal control. Discriminant function analysis demonstrated that the likelihood of being a Continuing Success is increased when there are, in combination (in decreasing order of importance): a high expectation of success; few cigarettes smoked upon entry into the program; low stress; ease of prior cessation attempts; a long period of prior abstinence; and a high degree of personal security. The results suggest that it is possible to predict which participants in a smoking control program will have problems with cessation and maintenance of cessation; and that smoking control programs can target intervention toward specific individual needs. (Auth. Abs. Mod.)

82-0807. Pomerleau, O. F. **Why People Smoke: Current Psychobiological Models.** In: Davidson, P. O.; Davidson, S. M. (Editors). *Behavioral Medicine: Changing Health Lifestyles.* New York, Brunner/Mazel, Inc., 1980, pp. 94-115.

Three models of the smoking habit are reviewed, with their implications for research, treatment, and social policy. The Social Learning Model explains smoking as a habit learned via social reinforcements, usually peer pressure. Eventually tolerance of the unpleasant effects occurs, and positive reinforcement from generalized external stimuli propels the habit. Smoking is difficult to modify due to its own immediate reinforcement (nicotine), the delay in negative health consequences, and because the habit is overlearned. The Nicotine Addiction Model proposes that smoking is an escape response to nicotine withdrawal in an addictive cycle. An internal regulatory mechanism provides the stimulus, and smokers who consciously control their smoking habit must tolerate levels of withdrawal. The Opponent Process Model combines elements of both models. A central nervous system mechanism is postulated which operates homeostatically to reduce departures from affective equilibrium. Smoking is positively reinforced by producing pleasure, and negatively reinforced by withdrawal, setting up an addictive cycle. Should a smoker quit, the B-state, withdrawal, will grow weaker, but the A-state, smoking pleasure, will grow stronger in the equilibrium, thus facilitating resumption of the cycle. The three models presented can be used to provide a general framework and theory for research. Since smoking consists of a complex interaction between overt behavior, autonomic responses, and subjective states, parallel measurements on all three levels of functioning should be made.

82-0808. Poole, A. D.; Dunn, J.; Sanson-Fisher, R. W.; German, G. A. **The Rapid-Smoking Technique: Subject Characteristics and Treatment Outcome.** *Behaviour Research and Therapy* 20(1): 1-7, 1982.

Abstinence/nonabstinence were employed as the independent variable and subject (S) characteristics as the dependent variables to discriminate between subjects who were abstainers or nonabstainers following their participation in a smoking treatment program utilizing rapid-smoking. There is a lack of consistency in findings regarding S characteristics and the effects of smoking-control programs. Research workers appear to assume that abstinence and reduction in smoking rate are merely different points in the same continuum, and therefore seek to correlate S variables with percentage reductions in smoking rate. It is argued that abstinence and reduced smoking are more appropriately viewed as potentially discrete outcomes. Conceptualizing outcome in this manner, a discriminant function analysis was employed to determine whether it is possible to differentiate between Ss who do, or do not, abstain following participation in a treatment program involving rapid-smoking. It was found that it is possible to discriminate between the two groups of Ss. It is suggested that discriminant function analysis might be used more widely for examining subject characteristics and treatment outcome. (Auth. Abs. Mod.)

82-0809. Public Health Reports. **Effect of People's Education on Their Health Habits and Views of Personal Health.** *Public Health Reports* 97(1): 88, January-February 1982.

A 1979 telephone interview study found several relationships between people's level of education and health habits and views; among them a finding that people with less education were more likely to smoke and to use cigarettes with high tar and nicotine. The survey involved 3,000 people aged 20 to 64. Health practices also varied by age and sex.

82-0810. Roy, M.; Chakraborty, A. K. **Smoking and Drug-Abuse Among the Newly Admitted Students of Medical Colleges in West Bengal.** *Indian Journal of Public Health* 25(1): 30-35, January-March 1981.

A survey of 557 male and 148 female students who were admitted to medical colleges of West Bengal in 1979 revealed that 98 or 17.6 percent of the male and none of the female students had the experience of smoking. Only 18 or 3.2 percent of the males were regular smokers. Average number of cigarettes smoked by a regular smoker was 5.5/day. Drug abuse was found in 12 or 2.2 percent of the male students, and only 4 or 0.7 percent used dependence-producing drugs regularly. Smoking and drug abuse were more common among students from urban areas (23.1 percent) than among those from rural areas (8.4 percent). A rising trend of regular smoking and drug abuse was noted with increase in income of the parents. A regular smoking habit was absent among those students whose parents' income was less than Rs. 500/-, and regular drug abuse was absent among those whose parents' income was less than Rs. 1000/-. Education of the parents was not related to smoking and drug abuse in the students. (Auth. Abs. Mod.)

82-0811. Safer, M. A. **An Evaluation of the Health Hazard Appraisal Based on Survey Data From a Randomly Selected Population.** *Public Health Reports* 97(1): 31-37, January-February 1982.

In a study to identify the population most likely to benefit from the Health Hazard Appraisal (HHA) questionnaire, 268 telephone interviews were conducted in Milwaukee, Wisconsin. The HHA is used as a health counseling tool. Smoking is included as a risk factor, along with age, sex, race, educational attainment, family income, and residential area. It was determined that people under 60 are more likely to smoke than those over 60: cigarette and alcohol consumption were correlated. Men were more likely to report smoking than women. Because younger subjects respond more to bodily symptoms than numerical risk estimates, programs using the HHA may prove more effective in persons 60 years and older.

82-0812. Sardar, Z. **Sheesha Cancer.** *New Scientist* 93(1293): 459-460, February 18, 1982.

Sheesha smoking reportedly carries greater health risks than cigarette smoking. A research team from King Abdul Aziz Medical College analyzed blood samples from 96 healthy Saudi males and found that levels of carbon monoxide (CO) were higher in sheesha smokers than in cigarette smokers. Sheesha smokers also suffered more frequently from headaches, dizziness, blurred vision, and palpitations than did cigarette smokers. These findings support the thesis that although nicotine from jurak (tobacco, banana pulp, and molasses) smoke is dissolved in the water of the pipe, CO and the tar contents that are insoluble in water remain unaltered. Tobacco smokers adjust the number of cigarettes, the frequency of puffing, and their depth of inhalation to a predetermined dose of nicotine depending on the degree of addiction. Theoretically, cigarettes high in nicotine would be smoked less. In sheesha smoking, the abundant smoke from burned jurak contains other carbonaceous material and more CO. Because it is inhaled more deeply and frequently as a result of the low level of nicotine, sheesha is considered to be more dangerous than cigarettes.

82-0813. Schachter, S. **Urinary pH and the Psychology of Nicotine Addiction.** In: Davidson, P. O.; Davidson, S. M. (Editors). *Behavioral Medicine: Changing Health Lifestyles.* New York, Brunner/Mazel, Inc., 1980, pp. 70-93.

Although in questionnaire studies smokers report smoking to relax or relieve anxiety, a close look at stress testing shows that nonsmokers perform as well as smokers. However, deprived smokers do poorly. Thus smoking does not reduce stress: smokers smoke to avoid the stress of withdrawal. Heavy smokers who switched to low-nicotine cigarettes smoked more, but not enough to bring dosage back to normal. These deprived smokers displayed irritability, inability to concentrate, ate more candy, and showed other withdrawal symptoms. Serious smokers do smoke more under stressful circumstances. Nicotine is readily excreted in low pH urine, but not in alkaline urine. Prolonged stress alone increases the acidity of urine, and thus, under stressful conditions, nicotine is more readily eliminated from the body. In an experiment with stressed subjects who were given a placebo or alkylating agent, smoking did not increase in response to stress in those subjects with high pH urine. Thus stress causes an increase in smoking for nicotine replacement and avoidance of withdrawal effects.

82–0814. Senior, S. L. **Study of Smoking Habits in Hospital and Attitudes of Medical Staff Towards Smoking.** *Canadian Medical Association Journal* 126(2): 131-133, January 15, 1982.

A study of smoking habits among the patients and staff of a hospital found that many smoking patients continued to smoke in the hospital, and that physicians were less likely to be smokers than other hospital staff. Patients were surveyed to determine if they smoked and if their doctors advised or ordered them to stop smoking in the hospital. Also, the smoking habits and attitudes toward smoking of the medical staff and other hospital workers were explored. Of 741 patients 37 percent were smokers, and of those who responded fully to a questionnaire, 86 percent continued to smoke in the hospital. Patients who were advised or ordered not to smoke (59 percent) were no more likely to stop smoking than those who were not so advised or ordered. Physicians were less likely to smoke than other hospital staff, and those who did smoke were much more likely not to smoke while in the hospital. Physicians appear to have a reasonable appreciation of the health hazards of smoking, and almost two thirds are in favor of greater restrictions on patients' smoking in hospitals. The ineffectiveness of their efforts is primarily due to hospital policies that are not in keeping with physicians' standards of practice and with established knowledge of the deleterious effects of smoking on health. (Auth. Abs. Mod.)

82–0815. Shiffman, S. **Relapse Following Smoking Cessation: A Situational Analysis.** *Journal of Consulting and Clinical Psychology* 50(1): 71-86, February 1982.

Factors and responses affecting relapse in ex-smokers were studied. Data on the antecedents of relapse crises (actual or near lapses in abstinence) were collected from 183 ex-smokers who called a relapse-counseling telephone hotline. Most relapse crises were associated with negative effects, particularly anxiety, anger, and depression. One-third of relapse crises were associated with positive feeling states and were frequently precipitated by other smokers, eating, and alcohol consumption. Withdrawal symptoms played a lesser role than expected: Half of the episodes occurred in the absence of symptoms. Ex-smokers' coping responses rather than situational antecedents distinguished relapse crises resulting in smoking from those in which abstinence was maintained. Ex-smokers who performed any coping response were more successful in forestalling relapse. A combination of cognitive and behavioral responses was most successful. Behavioral coping was subject to situational influences: Subjects who had been drinking alcohol were less likely to engage in behavioral coping, and depression diminished its effectiveness. Cognitive coping responses, which were less affected by these variables, may be critical components of ex-smokers' coping repertoires. (Auth. Abs. Mod.)

82–0816. Shryock, H. **We've Come a Long Way, Maybe, on the Matter of Cigarettes.** Mountain View, California, Pacific Press Publishing Association, 1981, 96 pp.

Information is presented about the characteristics and possible effects of smoking, and advice on how to quit the habit. The past history of smoking and its establishment as a major industry are discussed; the decreased rate of smoking among physicians is noted. Explanations are offered for why people smoke, including psychological and physiological factors. A section is devoted to smoking's possible hazards for women as well as for fetuses and infants. Economic costs to the individual and to society are described, including money spent on cigarettes, absenteeism from work, shorter lives, and health care costs. The overall health hazards of smoking are reviewed, and some of the responses and alternatives to smoking are mentioned including quitting, the use of tobacco substitutes, nicotine chewing gum, the manufacture of low-yield brands, and the use of filters. Advice on a self-regulated regime to quit smoking is also included.

82–0817. Singh, S.; Preet, K. **Drug Abuse Amongst School and College Students in Punjab.** *Child Psychiatry* 14(1): 5-11, January-March 1981.

Data from a drug use survey of 440 students (12 to 20 years of age) in Patiala, Punjab, showed that tobacco was the preferred drug; 30.63 percent had used tobacco at some time. Drug use patterns were established through questions on drug use ever in lifetime, in the last 30 days, frequency of use, age at onset, and drug most commonly used. Other drugs ever used included alcohol (25.67 percent), tranquilizers (4.95 percent), sedatives (4.95 percent), cannabis (4.05 percent), amphetamines (3.82 percent), and opium (3.15 percent). Of the 15.54 percent of students reporting current use of tobacco, 48 percent were regular smokers (more than 20 days during the past month). A similar pattern of alcohol use was shown. Although over 25 percent admitted alcohol use at some time, only about 14 percent were current users, only slightly less than current tobacco users. Current use of other drugs was very low, indicating that students experimented with these drugs, but few were regular users. Societal constraints (drinking and smoking by women are considered taboo) probably explain the significant sex differences reported, with males using more types of drugs. Use of tranquilizers and sedatives, however, was reported by more women than men. It is noted that a higher prevalence of use was found in students not living at home, indicating that experimentation may occur in less restrained surroundings.

82–0818. Stanaway, R. G.; Watson, D. W. **Smoking and Personality: A Factorial Study.** *British Journal of Clinical Psychology* 20(3): 213-214, September 1981.

Personality scores of smokers vs. ex-smokers vs. nonsmokers as measured by the Eysenck Personality Inventory (EPI) and the Sensation-Seeking (SS) Scale are compared. A sample of 119 males and 177 females, ages 17 to 65 (115 current cigarette smokers, 47 ex-smokers, 9 pipe and/or cigar smokers, 125 nonsmokers), completed a biographical smoking questionnaire, together with an EPI (form A) and the SS Scale (form 1).

Cigarette smokers were asked to complete two additional questionnaires. A significant relationship was found between smoking status and neuroticism (N), with cigarette smokers scoring higher than nonsmokers or ex-smokers. Cigarette smokers scored somewhat higher than nonsmokers on extraversion (E) and sensation-seeking (S) and lower on lie scale (L) but these differences were not significant. When age was controlled for in an analysis of covariance, the results for E and L were essentially the same as before. For N, the relationship with smoking status was somewhat less strong than before, although still significant. S was now found to be very significantly related to smoking status with smokers scoring higher than nonsmokers, and ex-smokers intermediate between the other groups. To correct for dependent variables, the analyses were repeated using factor scores derived from a combined analysis of the EPI and SS Scale. Factor 4, psychoticism, was the only factor score to be significantly related to smoking status, and was the most strongly related of all the personality measures investigated, with smokers obtaining the highest scores and nonsmokers the lowest. N and S both correlated somewhat with factor 4, and it was found that when factor 4 was introduced into the analyses as an additional covariate, N and S were no longer significantly related to smoking status. Further investigation of the relationship between personality and smoking motivation showed that (1) extraversion was coupled with affect—control smoking; (2) a link was apparent with social or social-confidence smoking, low sensation-seeking, and with the female sex; and (3) a favorable image of cigarette smokers is most readily accepted by highly neurotic individuals. The data suggest that extroverts are most inclined to use cigarettes for either stimulant or tranquilizing purposes.

82–0819. Tagliacozzo, R. **Smoking Behavior and Exposure to "Dissonant" Information.** *International Journal of the Addictions* 16(7): 1149-1159, 1981.

To determine smokers' receptivity to information on the health risk of smoking, a questionnaire was submitted to 514 young adults attending classes at a community college. The questionnaire included questions on the smoking history of the respondents and their beliefs and attitudes toward smoking, and a list of information items, several of which dealt with the health risk of cigarette smoking. To test whether smokers avoid exposing themselves to "dissonant" information, the respondents were instructed to go through the list a second time and to circle items important for smokers. The prediction that a larger percentage of smokers than of nonsmokers would fail to circle any of the items was verified. No difference was found between smokers who expressed a wish to stop smoking and smokers who did not. (Auth. Abs.)

82–0820. Walker, G. R., Jr. **Self-Awareness and Cigarette Smoking: The Interaction of Public and Private Determinants.** Doctoral Dissertation, University of Florida, Gainesville, University Microfilms International 81-24462, March 1981, 73 pp.

Competing predictions by different models of self-awareness are tested. Persons who smoke cigarettes and who hold negative public attitudes toward smoking (i.e., believe that others disapprove of smoking) took part in an experiment designed to test their willingness to smoke under different circumstances. The model of self-awareness espoused predicted that the presence of a mirror would lead to greater smoking when the subject was alone, but to less smoking when the subject was with another person. Data from the experiment showed no significant overall differences due to the experimental manipulations. Followup analyses, however, suggested that mirror presence increased smoking only when another individual was present. Results are discussed in terms of the different models of self-awareness. It is concluded that the model of self-awareness espoused was not supported, and that the pattern of results generally supports the Self-Consciousness Perspective. (Auth. Abs. Mod.)

See also, **82–0648, 82–0826, 82–0830, 82–0843, 82–0864, 82–0865, 82–0866, 82–0885, 82–0906**

SMOKING PREVENTION AND INTERVENTION

82–0821. Ahmed, M. B.; Hilton, T. F. **How To Help Patients Stop Smoking.** *American Family Physician* 25(2): 13[illegible]136, February 1982.

Family physicians, in the role of therapists and consultants, may have considerable success in counseling against smoking, an important aspect of preventive health care. Patients cannot easily dissociate themselves from the adverse effects of smoking when symptomatic or pregnant, thus providing an opportunity for intervention. Awareness of available facilities, techniques, social support systems, and medical interventions enables the physician to match a stop-smoking strategy to the patient's personality, thus affording the best chance for successful cessation. Various strategies are discussed and evaluated.

82–0822. Bartlett, E. E. **The Contribution of School Health Education to Community Health Promotion: What Can We Reasonably Expect?** *American Journal of Public Health* 71(12): 1384-1391, December 1981.

A review of traditional school health education programs shows that although these programs are effective in increasing knowledge, and somewhat effective in improving attitudes, they are largely ineffective in changing health behavior. Relative to antismoking education, the more encouraging attempts to modify teenage smoking have relied on: (1) focusing on immediate, rather than long-term threats to health, (2) small-group student-led discussions to identify ways of counteracting peer and media pressure to smoke, (3) high-involvement peer-led activities, and (4) close coordination with community resources. Health education programs are effective in providing knowledge, which is the basis for behavior change, but are rarely sufficient for such change. Intervention in destructive health habits requires far more than traditional didactic school programs. Smoking is an ingrained personal habit and is very resistant to change. School health education programs cannot achieve long-standing changes in lifestyle without support from the family, community, and health professionals.

82–0823. Baumgartner, K. **Smoking Cessation: Physician—Make a Stand.** *Canadian Medical Association Journal* 126(2): 101-102, January 15, 1982.

Physicians are urged to encourage smoking cessation among their patients who smoke, and those doctors who smoke should not do so in front of their patients. Providing health education to patients can take the passive form of displaying information material in physicians' offices. Such educational materials are available from several agencies and organizations. A more active approach involves counseling patients on the risks of smoking and the benefits of quitting. Doctors can also routinely ask patients about their smoking habits. Physicians see many patients each year, including smokers, and thus could help produce a noticeable number of ex-smokers. Doctors should also take a more prominent public profile against smoking.

82–0824. Best, J. A. **Mass Media, Self-Management, and Smoking Modification.** In: Davidson, P. O.; Davidson, S. M. (Editors). *Behavioral Medicine: Changing Health Lifestyles.* New York, Brunner/Mazel, Inc., 1980, pp. 371-390.

A self-management smoking cessation program, based on a television series and an accompanying self-help guide, apparently achieved good maintenance rates during followup. Postprogram rates of abstinence among the respondent sample population were measured at the end of the series, at 3 months, and at 6 months. Success levels showed little or no reduction over time. It was found that most preprogram subject variables examined did not relate to success of outcome at 6 months, including age, sex, education, age of beginning smoking, and earlier attempts to quit. Successful subjects, however, had a lower level of smoking at program start. Subjects who had already tried to quit by themselves tended to have greater success during the present program. Postprogram measures indicated that smoking behavior near the conclusion of the series and perceived probability of future success were highly predictive for long-term success. The number of shows viewed did not appear related to success at 6 months. Relapse appeared to be situation-specific. Stress was often associated with relapse. The preliminary but promising results of the project suggest that the potential uses of mass media in health lifestyle change deserve greater attention in the future.

82–0825. Farquhar, J. W.; Magnus, P. F.; Maccoby, N. **The Role of Public Information and Education in Cigarette Smoking Controls.** *Canadian Journal of Public Health* 72(6): 412-420, November-December 1981.

The origins, methods, and impact of the major forces responsible for the adoption and partial cessation of cigarette smoking in the United States during the 20th century are addressed. The phases of adoption, partial cessation, and industry counter-responses are described. Evidence is given for the beneficial role of education on a national, community, school, and small group level. The need for an idealized model for smoking control is discussed and examples of successful planned programs and recommendations for further research and action are presented. The voluminous educational efforts coming from the public and private sectors in the United States are referred to as the U.S. nationwide smoking reduction campaign and compared with the Norwegian National Campaign. As examples of community-based programs, the Stanford project and its successor, the Stanford Five City Project, are detailed and the Finnish North Karelia Project is summarized. Recommended action in the United States includes expansion of field research efforts and a total ban on all tobacco promotion coupled with much more education, general and targeted.

82–0826. Green, D. E. **Teenage Smoking: Immediate and Long-Term Patterns.** U.S. Department of Health, Education, and Welfare, National Institute of Education, Washington, D.C., November 1979, 267 pp.

Results from the 1978-1979 National Institute of Education survey on the smoking behavior of teenagers are reported. Long-term trend data is provided by comparing the results of similar surveys in 1968, 1970, 1972, and 1974. Tabulated data from the questionnaire (copy reproduced) are included and discussed for: trends in prevalence of teenage smoking, family patterns, peer patterns, working practices, educational aspirations, perception of the health threat, prediction of future smoking behavior, and the dynamics of teenage smoking. Indications are that teenage smokers are now well aware of the health threat of smoking and respect the rights of nonsmokers, but also believe that experimentation is all right as long as you don't get hooked, which is what too often happens to the experimenter. Characteristics of the teenagers and their environments reveal that some cannot be changed (household composition, educational level of parents); and others could be targets for change, such as the smoking practices of parents and the aspirations of the teenagers. It is felt that as the positive influences deterring teenagers from smoking continue and grow, the teenagers will consider more realistically and objectively how they will deal with the problem. In the second part of the report, results of a more experimental and exploratory study, designed to elicit the variables related to adolescent smoking that might be useful in making predictions about smoking patterns in young adulthood, are presented. Data from the Longitudinal Study 1974-1979 provide: (1) background information; (2) description of the sample, including a comparison of the followup respondents with the 1974 survey sample, characteristics of the 1979 respondents, and their smoking history; (3) predictors of smoking behavior, including predictors of change in subsamples by 1974 and 1979 smoking status; and (4) conclusions. Results seem to indicate that current young adult smokers are concerned about the health effects of smoking and have tried to quit, or would try if they thought there was an easy way to do so. Typical smokers had: older siblings and peers who smoked; different attitudes toward teenage smoking than teenagers who never started or were able to quit; and lower educational aspirations and attainment than nonsmokers, probably a reflection of lifestyle, rather than a cause—effect relationship. It is hoped that a better understanding of these precedents will help in the efforts to reduce initiation of smoking in teenagers.

82–0827. Greenberg, J. A.; Pollack, B. **Motivating Students To Not Smoke.** *Journal of Drug Education* 11(4): 341-359, 1981.

An intervention program is described which showed a definite ability to produce statistically significant changes in college students' responses to statements on the cognitive and attitudinal factors which the Theory of the Diffusion of Innovation deems important to the reduction and prevention of smoking. It also produced a preponderance of statistically nonsignificant changes in responses to questions regarding smokers' motivation to attempt cessation and to modify smoking behavior. In addition, it produced a nonsignificant reduction in smoking behavior itself. Special presentations on smoking and topics such as social approval, academic achievement, and career success were made to the experimental group. A 91-item questionnaire was administered to both the experimental group (n = 196) and the control group (n = 146), during the second and fourteenth weeks of the semester program. An analysis of covariance showed statistically significant changes in students' responses to cognitive and attitudinal statements. It is possible that a longer period of time would have yielded more significant changes. Also, the inclusion of other variables in the analysis of variance might have allowed for greater significance in the results. The theory suggests that factors such as socioeconomic and demographic status, smoking status, and ability to adopt new innovations could have explanatory power. It is recommended that this approach be further investigated, since it has been shown that it can be used for developing new educational strategies with potential for reducing smoking.

82–0828. Jason, L. A.; Mollica, M.; Ferrone, L. **Evaluating an Early Secondary Smoking Prevention Intervention.** *Preventive Medicine* 11(1): 96-102, January 1982.

Antismoking interventions among ninth grade students were found to decrease the rate of smoking among experimenting smokers, but had minimal effect among regular smokers. An entire class of ninth graders was involved in a treatment condition featuring either role-playing and discussion, or only discussion. While both treatments effectively reduced levels of smoking among early smokers, few changes were noted for youngsters manifesting long-term, more entrenched habits. Levels of smoking for children monitored in a control school remained about the same. These data suggest that early preventive programs might be most efficacious in curtailing smoking among school-age children. (Auth. Abs. Mod.)

82–0829. Kirk, C.J.C.; Woolcock, N. E.; Lund, V. J. **The Effect of Advice To Stop Smoking on Arterial Patients Assessed by Serum Thiocyanate Levels.** In: Greenhalgh, R. M. (Editor). *Smoking and Arterial Disease.* Bath, Great Britain, The Pitman Press, August 1981, pp. 118-121.

The smoking habits of 39 patients admitted for arterial assessment were reviewed by history taking and by measure of serum thiocyanate levels. Of the 39 patients, (9 female, 30 male) total ages ranged from 32 to 78 years (mean 61), 7 stated that they were not current smokers (6 had thiocyanate levels below 70μmol/L), and there were no cigar or pipe smokers. Of the smokers, 5 were light smokers (1 to 10 cigarettes/day), 19 moderate (11 to 20 cigarettes/day), and 8 heavy (>20 cigarettes/day). All but 8 proceeded to surgery with followup ranging from 2 to 19 months. From the time of admission, smoking was discouraged in various ways: (1) the consulting surgeon gave instructions and reasons for smoking cessation in a forceful manner; (2) smoking was discouraged on the wards; (3) followup appointment cards reiterated the no smoking warning in block capitals; and (4) the advice was reinforced at each successive visit. This trial resulted in 13 of 32 patients claiming to have stopped smoking; 11 were confirmed by thiocyanate values. Twenty patients were still smoking but one was smoking only one or two cigarettes per day. Of the 19 remaining, 15 claimed to have reduced their smoking levels, but there was no significant diminution of thiocyanate values in this group. It would appear that more credence can be given to a claim of complete cessation than to a claim of reduction of smoking.

82–0830. Lichtenstein, E.; Ransom, C. C., Jr.; Brown, R. A. **Effects of Counselor Smoking Status, Experience Level, and Treatment Modality on the Credibility of Smoking Cessation Programs.** *Journal of Drug Education* 11(4): 361-367, 1981.

One hundred and seventeen smokers were presented with descriptions of a smoking control program and a smoking counselor and were then asked to rate program, counselor effectiveness, and counselor empathy. The descriptions varied along three dimensions constituting a 3 x 2 x 2 factorial design: counselor smoking status (ex-smoker, current smoker, never smoker); counselor experience level; and group or individual treatment. There was a clear preference for group over individual therapy. The ex-smoker counselor was highly preferred, but overall credibility ratings for current or never smokers were still in the moderately effective/empathic range. There were no significant differences between the current and never smoker counselor, suggesting that a smoking counselor may still have a reasonable level of credibility. The counselor experience factor moderated the impact of counselor smoking status, but was significant only for the program effectiveness dimension. (Auth. Abs. Mod.)

82–0831. McAlister, A.; Puska, P.; Salonen, J. T.; Tuomilehto, J.; Koskela, K. **Theory and Action for Health Promotion: Illustrations From the North Karelia Project.** *American Journal of Public Health* 72(1): 43-50, January 1982.

The North Karelia Project in Finland illustrates the fundamental goals of health promotion. Specific activities of the project serve as examples of how concepts from the social and behavioral sciences can be applied to achieve estimated reductions in predicted risk of disease. Objectives included the following: (1) improved preventive services to identify persons at risk of disease; (2) information to educate people about their health; (3) persuasion to motivate people to take healthy actions; (4) training to increase skills of self-control, environmental management, and social action; (5) community organization to create social support and power for social action; and (6) environmental change to improve various unfavorable conditions. Goals were associated with pride and provincial identity of the population. Shop proprietors displayed signs reading "Do not smoke here—we are in the North Karelia Project" which fostered local patriotism. Support within the family was created by involvement wherever possible, i.e., enlisting the support of wives in their husbands' adherence to smoking cessation courses and informing them about how to deal with irritability on the part of a recent ex-smoker and how to be reinforcing. Public health concerns have made it easier to regulate promotion and marketing of products such as cigarettes. The results in North Karelia are not conclusive, but they are encouraging, and the investigation conducted there is an essential reference for future research in health promotion and disease prevention. (Auth. Abs. Mod.)

82–0832. McDermott, R. J.; Marty, P. J. **Athletes Score a Hit With Kids in Grades Four Through Eight.** *Journal of School Health* 52(2): 94-96, February 1982.

Some cancer may be related to attitudes and behavioral patterns, such as cigarette smoking, established early in childhood. Since athletes are excellent role models that school-age children can identify with, the Wisconsin Division of the American Cancer Society (ACS) initiated a program in the fall of 1978 called Wisconsin Athletes Against Cancer (WAAC). Designed to be implemented in grades four through eight, the program features specially trained University of Wisconsin athletes, who deliver antismoking, nutrition, and exercise messages in public, private, and parochial schools. Athletes work in pairs and whenever possible, represent both men's and women's sports. Sessions last 45 minutes and include a film to promote discussion. The action of tar from cigarette smoke is demonstrated with the "smoking lung." Athletes conclude the sessions by distributing ACS bookmarkers with special health messages. A teacher's resource packet is used as an adjunct to the program. The presentations seem to be well received and an evaluation is planned.

82–0833. Mettlin, C.; Sciandra, R.; Walsh, D.; Mirand, E. A. **Attitude and Knowledge of Public School Teachers Regarding Cancer Education.** In: Hobbs, P. (Editor). *Public Education About Cancer. Recent Research and Current Programmes.* UICC Technical Report Series, Volume 62, Geneva, 1981, pp. 34-38.

A survey of 1,065 elementary classroom and certified health teachers in the Buffalo, New York area was conducted by Roswell Park Memorial Institute to determine why a comprehensive cancer education curriculum was not available at lower grade levels. A 16-item questionnaire was devised to elicit attitudes toward cancer and the value of health education about cancer. There was a 60 percent response (N=639). Results showed that elementary school teachers are concerned about cancer, believe that something can be done, and that cancer education is an appropriate classroom topic, but feel poorly equipped for the task. Nearly two-thirds of respondents agreed that teachers have a responsibility to refrain from smoking in schools. Sixty-five percent feel that teachers could help prevent children from adopting the smoking habit. Many do not recognize smoking as the leading known cause of cancer. In response to the survey, an intervention program was developed. In the spring of 1980, a concise *Cancer Education Newsletter* was established and distributed to public school teachers to alert them to resources in their area.

82–0834. Milsum, J. H. **Lifestyle Changes for the Whole Person: Stimulation Through Health Hazard Appraisal.** In: Davidson, P. O.; Davidson, S. M. (Editors). *Behavioral Medicine: Changing Health Lifestyles.* New York, Brunner/Mazel, Inc., 1980, pp. 116-150.

The Health Hazard Appraisal (HHA) is a counseling tool for health professionals, designed to evaluate health lifestyles for stimulating health behavior change. All possible risks to an individual's health are quantitatively estimated and evaluated to anticipate the likelihood of mortality within the coming 10 years. Smoking habits and time since quitting are included as risks in major disorders such as cancer, heart attack, and pneumonia. Results of the computer evaluation of the HHA questionnaire are given as an "appraised age" (results of the health lifestyle) and an "achievable age" which can be reached by following lifestyle change recommendations. Followup studies have shown the HHA tends to be influential in reduction of alcohol consumption and weight, but not very effective in changing smoking behavior.

82–0835. National Interagency Council on Smoking and Health. **Smoking and the Workplace. Toward a Healthier Work Force.** New York, National Interagency Council on Smoking and Health, Fall 1980, 85 pp.

A guide is presented for persons, organizations, and representatives of management and labor interested in smoking prevention or cessation programs as a positive step to good employee health. Information included is the result of a planning meeting sponsored by the National Interagency Council on Smoking and Health with representatives of management, labor, National Interagency Council member agencies, and representatives of the academic and scientific community, which addressed the growing problem of the combined hazard of occupational exposure and smoking. The hazard, identified in the 1979 Surgeon General's report, generated a survey of 3,000 companies, results of which are summarized. In addition, reports and summaries from the planning meeting are provided, covering such areas as: (1) business gains from smoking cessation; (2) smoking cessation programs in occupational settings; (3) passive smoking effects; (4) positive effects of smoking cessation; (5) how to start a smoking control program; (6) National Interagency Council resources; (7) evaluation techniques; and (8) indoor clean air legislation.

82–0836. Northcott, H. C.; Jarvis, G. K. **Government Influence, Media Influence, and Quitting Smoking.** *Canadian Journal of Public Health* 72(6): 447-450, November-December 1981.

A survey of a representative sample of the adult population of Edmonton, Alberta showed that 16 percent of men and 14 percent of women had quit smoking in the past decade. An additional 7 percent of the male population together with 5 percent of the female population were smoking less. Those who had quit smoking or who were smoking less were more likely than nonsmokers or continuing smokers to say that government and media sources had influenced them to live a healthier life. A Toronto survey found that television was most often cited as the source of smoking and health information. The second choice for males was newspapers, while second choice for females was magazines. It is possible that those who quit smoking quit for reasons other than media influence and attribute effects after the fact to the government and the media. This caution notwithstanding, the findings suggest that government antismoking campaigns are perceived as having a positive impact. (Auth. Abs. Mod.)

82–0837. Padron, J. S. **Control del Tabaquismo en Uruguay. [Control of Tobacco Use in Uruguay.]** Ministerio de Salud Publica, Republica Oriental Del Uruguay, Direccion General de la Salud, Division Epidemiologia, Montevideo, Uruguay, 1981, 89 pp., Spanish.

The need for an established smoking control program in Uruguay is emphasized, in terms of present economic and health concerns. Smoking as an important cause of mortality, morbidity, disability, and as a negative factor in the economy is detailed. Smoking problems in Uruguay are analyzed via statistical data for lung cancer, chronic bronchitis, heart and vascular pathology, and tobacco consumption. The importance of tobacco dependency and quitting is shown. Educational activities accomplished at professional and nonprofessional levels are summarized, and remarks from outstanding international and national personalities and public health organizations are transcribed, including reactions of industrialized countries to the smoking epidemic in developing countries. Immediate and later objectives of a smoking control program in Uruguay, creation of a permanent control organization, and a National Honorary Committee integrated by delegates of the Ministry of the Public Health, School of Medicine, National Council of Education, and National Council of Physical Education are emphasized. (Auth. Abs. Mod.)

82–0838. Pechacek, T. F.; Danaher, B. G. **How and Why People Quit Smoking: A Cognitive-Behavioral Analysis.** In: Kendall, P. C.; Hollon, S. D. (Editors). *Cognitive-Behavioral Interventions. Theory, Research, and Procedures.* Personality and Psychology Series, Volume 21, New York, Academic Press, 1979, pp. 389-422.

The problem of smoking and attempts at intervention are summarized, the literature on smoking behavior and its resistance to long-term modification is reviewed, and a cognitive-behavioral model is applied to smoking control. Because of the complexity of the biochemistry of nicotine and other tobacco derivatives, pharmacological researchers do not understand the mechanisms of dependency. Additionally, a clearer understanding of the medical and pathological aspects of smoking has produced only moderate improvements in controlling the problem, leaving to behavioral sciences the task of developing more effective treatments. Of the programs developed, those demonstrating impressive long-term results when compared with the general trend are a combination of aversion and self-control procedures. Treatment recommendations are suggested but need to be tested by appropriate research. It is concluded that the most effective programs tend to be multidimensional, individualized, and based on sound rationale.

82–0839. Pederson, L. L. **Compliance With Physician Advice To Quit Smoking: A Review of the Literature.** *Preventive Medicine* 11(1): 71-84, January 1982.

The variety of roles that the physician can assume with patients who smoke cigarettes ranges from providing a model by being a nonsmoker, to active management of the quitting process. The literature dealing with effectiveness of physician advice with general practice, pregnant, pulmonary, and cardiac patients is presented. It is concluded that, in general, the more severe the disease and the more imminent the danger from continued smoking, the more likely patients are to comply with advice to quit. Statistical consideration of the interrelationships between patient and physician characteristics ultimately may lead to providing the most effective methods to the largest number of patients. Practical recommendations for physicians are suggested: (1) act as a model of a healthy lifestyle, (2) clarify the risk associated with smoking and risk reduction with cessation, (3) encourage abstinence by direct advice and suggestions, (4) refer patients to smoking cessation programs, and (5) prescribe and follow up the use of specific cessation and maintenance strategies. (Auth. Abs. Mod.)

82-0840. Pederson, L. L.; Baskerville, J. C.; Lefcoe, N. M. **Change in Smoking Status Among School-Aged Youth: Impact of a Smoking-Awareness Curriculum, Attitudes, Knowledge and Environmental Factors.** *American Journal of Public Health* 71(12): 1401-1404, December 1981.

The relationships between exposure to a smoking awareness curriculum, attitudes toward and knowledge about smoking, sociodemographic and smoking exposure characteristics, and change in smoking status over three years are examined. Children from 20 public schools in London, Ontario, Canada were tested first in 1975 when in grades 4-6, and again in 1978 when in grades 6-8. Half of the schools were provided with a smoking and health curriculum guide to be used by teachers in health education; teachers in the remaining 10 control schools did not receive the guide. Results were available for 1,682 children who responded on both occasions. Over the three year period, 4.5 percent of the students decreased their involvement with cigarettes, 56.6 percent reported no change, and 38.9 percent reported an increase. Exposure to the curriculum did not bear a significant relationship to change in smoking; however, changes in peer smoking, knowledge, and parental smoking were significant predictors. (Auth. Abs. Mod.)

82-0841. Ronan, G.; Ruane, P.; Graham, I. M.; Hickey, N.; Mulcahy, R. **The Reliability of Smoking History Amongst Survivors of Myocardial Infarction.** *British Journal of Addiction* 76(4): 425-428, December 1981.

Testing of 117 survivors of myocardial infarction indicates that the rapport achieved between patients and medical staff made reports of smoking history reliable in studies of smoking factors on survival rates. All subjects are participants in a long-term, regular followup study and secondary prevention program. All were given detailed advice and a booklet about smoking at the hospital and at followup visits, and were treated with a policy of early mobilization, return to work, and risk factor intervention. Reports of smoking were checked by blood carboxyhemoglobin levels. There was a low, and possibly zero, deception rate among the ex-smokers, reflecting a high degree of rapport achieved between patients and medical staff during an exceptionally long and regular followup period.

82-0842. Rothmeier, R. C. **Commitment to Change: Effects of Contractual and Scanning—Focusing Techniques on Smoking Behavior.** Doctoral Dissertation, University of Nebraska, Lincoln, University Microfilms International 81-22602, April 1, 1981, 90 pp.

Twenty-four residents of Lincoln, Nebraska, participated in a stop smoking program. All subjects smoked at least 70 cigarettes per week, and all expressed a need for help in stopping smoking, as previous self-initiated attempts had failed. Half of the subjects were treated by a commitment-enhanced program combined with the American Cancer Society (ACS) program, and half by the ACS program only. Two dependent measures were taken to assess changes in smoking behavior: (1) self-monitored smoking rates gathered on "wrap sheets," and (2) carbon monoxide levels in alveolar air. The flexibility scale of the California Psychological Inventory was also included. More subjects under commitment-enhancement (4 of 12) stopped smoking than subjects who received only the ACS program (1 of 12). This difference in abstinence was maintained throughout the 6-week followup period. Both ACS and ACS plus commitment-enhanced treatment groups significantly reduced their rates of smoking as reflected in both self-monitored rates and carbon monoxide levels. The analysis of subject flexibility differences was not significant.

82-0843. Schachter, S. **Self-Treatment of Smoking and Obesity.** *Canadian Journal of Public Health* 72(6): 401-406, November-December 1981.

Results of a survey show that in nontherapeutic populations, the rates of successful self-cure of cigarette smoking and obesity are considerably higher than reported in therapeutic literature. Two different populations (n=161) were interviewed for smoking and weight histories: (1) faculty members, graduate students and staff of the Psychology Department at Columbia University, and (2) a portion of the entrepreneurial and working population from the town of Amagansett. Among those subjects with a history of cigarette smoking (n=94), an overall 63.6 percent had succeeded in quitting smoking by the time of the interview. The successful quitting rates were similar for both populations. This showed a higher success rate than the approximately 10-25 percent of self-selected therapeutic populations who remain quitters 1 year after therapy. The nontherapeutic populations also showed greater success in weight loss. Higher cure rates from self-treatment than from therapeutic intervention have also been found for heroin and opiate addiction, as in populations of Vietnam veterans. One possible explanation for such differences is that people who cure themselves do not go to therapists, while the more difficult cases do. Also, studies of therapeutic effectiveness may describe only the results of a single attempt to quit smoking or other unwanted behaviors, whereas repeated attempts to quit among a given population may increase the success rate.

82-0844. Sneader, W. **"Stop-Smoking" Campaign in West of Scotland Pharmacies.** *Pharmaceutical Journal* 228(6159): 105, January 23, 1982.

Approximately 150 pharmacies in Scotland participated in a "stop-smoking" project sponsored by the Scottish Health Education Group and Action on Smoking and Health. Each participating pharmacist was provided with a kit containing appropriate posters, display stickers, badges for counter assistants, and display boxes containing 250 copies of a tabloid newspaper which provided easily assimilable information on how to attempt to stop smoking. An assessment of one-third of the participants, conducted after the project had run for 8 weeks, revealed that 95 percent of the interviewed pharmacists would take part in the project again; 23 percent felt the project was not very effective in changing customers' smoking habits. Pharmacies were found, on average, to have disposed of between 100 and 200 copies of the free newspaper. Indications are that the greatest impact was made on women over 30, from a working-class background. It was also evident that some three-quarters of the participating pharmacists and their assistants took an active role in the project by discussing smoking withdrawal with customers.

82-0845. Superintendent of Public Instruction. **Smoking and Health Guide for Elementary and Secondary Schools.** Olympia, Washington, State Office of Public Instruction, September 1980, 88 pp.

A working copy of the Washington State smoking and health teaching unit is presented. The guide was developed to assist teachers in planning an interesting and effective instructional program directed toward improving the health behavior of children and youth, as well as the presentation of facts. Included in the guide are conceptual statements for elementary, junior high, and senior high schools. Objectives for the selection of content and effective learning experiences, resources, and an appendix of sample materials and simple experiments are provided.

82-0846. Taylor, D. H. **Response to Anti-Smoking Intervention in Pregnancy.** In: Madden, J. S.; Walker, R.; Kenyon, W. H. (Editors). *Aspects of Alcohol and Drug Dependence.* Bath, Great Britain, Pitman Press, 1980, pp. 325-331.

Women who wish to give up smoking during pregnancy present a considerable demand for smoking cessation assistance. However, when this assistance was offered apart from the antenatal care system, uptake was low among those who most needed help. Of 426 expectant women at two antenatal clinics, those who smoked were referred to a short interview concerning smoking cessation. Of 88 appointments resulting from these interviews, only 25 were kept. The principal difference between those who attended and those who broke their appointments was educational level: attendance rate was only 27 percent for those who left school prior to age 17. Those who had refused to modify their smoking behavior in the initial interview were typically lower in socioeconomic class, by occupation head of household; did not believe smoking harmful during pregnancy; claimed low levels of smoking; and believed they would have little difficulty in stopping if so inclined. It appears that the provision of smoking cessation assistance for pregnant women should be within the antenatal care system.

82-0847. Timbrell, D. **Smoking or Health in the '80's.** *Canadian Journal of Public Health* 72(6): 370-372, November-December 1981.

Canadian society and government continue to develop responses to smoking and its health hazards. The proportion of regular smokers among the Canadian population has dropped from 42.8 percent in 1965 to 34.2 percent in 1979. Data about smoking and its effects will need effective educational presentation. It is known that the environment can be a motivating factor associated with smoking; however, as the number of nonsmokers continues to grow, there may be less social encouragement and physical access to smoking. A new lifestyle emphasis on health and preventive measures may promote the desire to quit or not begin smoking. Health education efforts can be intensified in schools and with the general public, and promotion and advertising can be used. School age children may be a source of special concern, since they seem to be taking up smoking at an earlier age than did previous generations. More young women than men appear to be taking up cigarette smoking. Government and the public can both work against smoking.

82-0848. Weis, W. L. **Can You Afford To Hire Smokers?** *Personnel Administrator* 26(5): 71-73, 75-78, May 1981.

Restrictions and actions by employers against smoking by employees can lead to improvements in absenteeism, morbidity and premature mortality, other insurance costs, productivity, property damage, and morale. Smokers tend to be absent more often than nonsmokers, and may incur higher medical care costs. Other insurance considerations may include accidental injury and workers' compensation costs. Productivity loss may stem from working time spent on the rituals and habits associated with smoking. Cigarette burns can damage furniture, the smoke can affect sensitive equipment and also the costs of maintenance and cleaning. Involuntary or passive smoking may adversely affect nonsmokers' health and morale. Action against smoking may help improve company operations. The total cost of employing smokers is estimated as $4,611 per smoker, per year.

82-0849. Weis, W. L. **"No Ifs, Ands or Butts"—Why Workplace Smoking Should Be Banned.** *Management World* pp. 39-40, 44, September 1981.

It is predicted that employers can expect to save about 4,789 dollars per year per smoker by adopting a two-pronged policy that restricts hiring to nonsmokers and prohibits smoking on company premises. This policy, if implemented properly, will result in a reduction in (1) personnel costs: same amount of work with 10 percent fewer employees; (2) maintenance: interior window washing, sweeping, repainting, and repairs to carpeting and draperies; and (3) insurance premiums: fire, health, accident, and disability. Employee morale should improve and clients will accept the new policy. Studies are cited supporting the hypothesis that damage to involuntary smokers is real and severe. A case of a smoking-free company program (Radar Electric of Seattle) is described. Since sick leave was considered a special wage subsidy for smokers, it was eliminated and no new smokers were hired. The program was successful, showing an impressive productivity impact as well as savings in company costs.

82-0850. Wilson, D.; Wood, G.; Johnston, N.; Sicurella, J. **Randomized Clinical Trial of Supportive Follow-up for Cigarette Smokers in a Family Practice.** *Canadian Medical Association Journal* 126(2): 127-129, January 15, 1982.

Recently, family physicians have begun to test the effectiveness of certain smoking-control strategies in primary care settings. A trial took place in two middle-class family practices in Hamilton, Ontario. Smoking patients (n = 211) over 16 years of age, between December 10, 1979, and May 27, 1980, were asked to complete a questionnaire which was designed to facilitate the recording of a comprehensive smoking history including: age at onset, consumption rate, insight into health factors, and motivation to quit. Short counseling sessions were provided reinforced with a pamphlet. Strategies reviewed included: cold turkey, self-monitoring, aversion techniques, acupuncture, hypnosis, group sessions, and nicotine gum. Patients were randomly assigned to a control or experimental group. Controls (n = 105) received no further support. Experimentals (n = 106) received appointments for 1-, 3-, and 6-month reviews. Results of the experiment showed that the type of cigarette influenced the outcome. Of those who smoked low-tar cigarettes, there was little difference in outcome; of those who smoked regular cigarettes the proportion who quit was almost 5 times greater in the experimental than in the control group ($p < 0.05$). The only other prognostic factor approaching statistical significance was sex ($p = 0.11$), success rate somewhat greater in men. Sex and type of cigarette were highly associated: 60 percent of men, compared with 25 percent of women, smoked regular cigarettes. Those who believed in their own willpower (one-third in experimental group) quit compared with 22 percent with a weak locus of control and 11 percent who were fatalistic. Surprisingly, in the experimental group, those with perceived health problems were less likely to quit ($p = 0.02$). Finally, only 5 percent in the experimental group who judged it extremely difficult to quit were able to. Results demonstrate that family physicians can play a key role in smoking control.

See also, **82-0777, 82-0816**

82-0851. Winkelstein, W., Jr.; Marmot, M. G. **Primary Prevention of Ischemic Heart Disease: Evaluation of Community Interventions.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 2. Palo Alto, California, Annual Reviews Inc., 1981, pp. 253-276.

Modification of ischemic heart disease (IHD) risk factors, including smoking, hypercholesterolemia, and high blood pressure, prior to clinical manifestations of the disease (primary prevention), is discussed. Five community intervention programs are evaluated: (1) the Hypertension Detection and Follow-Up Program under the auspices of the National Heart, Lung and Blood Institute of the National Institutes of Health which aimed to reduce high blood pressure in several communities; (2) the Chicago Coronary Prevention Evaluation Program which achieved modest reductions in smoking; (3) the Multiple Risk Factor Intervention Trial whose data are still incomplete; (4) the North Karelia Project in Finland which achieved a substantial net decline in smoking; and (5) the Stanford Heart Disease Prevention Project which also reduced cigarette smoking. The IHD reduction results of the five programs were inconclusive, not reported, or incompletely reported, except for the North Karelia Project, which achieved a modest IHD reduction. It is suggested that a better understanding of factors that influence blood pressure levels may make primary prevention feasible, while dietary changes may be a useful response to the hypercholesterolemia factor. Intensified efforts against cigarette smoking are recommended since smoking cessation favorably affects the risk of developing IHD.

82-0852. Wynn, R. A. **Hard Drugs? (Letter).** *Journal of the Medical Association of Georgia* 71(2): 124, February 1982.

The social and legal status of the use of tobacco products in this country, despite known health risks, is discussed. It is noted that unlike other (illegal) drugs, tobacco use is socially acceptable, although its effects are described as insidious and long-term, and no less devastating medically, economically, socially, and personally than other drugs. Because tobacco use can be causally related to more deaths than all other drug habits, and because the few legislated limitations on its use are not really enforced, more government intervention is urged. Possible areas could include licensing of outlets, higher taxes (to be used to treat tobacco-related illnesses), or dollar-for-dollar contributions by tobacco companies for advertising for and against tobacco use. Education of business leaders on the economic losses due to tobacco use by employees may also help. It is suggested that the economic facts of life may succeed in cases where the appeal to reason has failed.

SMOKING CESSATION METHODS

82-0853. Allen, R. F. **A Program for Personal and Cultural Change on Smoking. A Part of the Lifegain Program for Changing Our Health Cultures.** New York, Appleton-Century-Crofts, 1981, 39 pp.

The Lifegain System self-help module for quitting smoking is presented. The ultimate goal of the module is a self-designed smoking cessation program. By completing questionnaires and a planning guide, the reader evaluates the present health practices and health norms of his/her social environment. Smoking and health information is given, followed by an action-planning guide for developing and implementing a self-styled nonsmoking program. The steps in the process are: (1) understand the culture, (2) factfinding, (3) build supportive environments, (4) put plan into action, (5) keep track and tune in, (6) rewards and having fun, and (7) reach out to others.

82-0854. Carrasco, J. C. **Modificacion del Comportamiento y Habito de Fumar (Estudio Piloto). [Modification of Behavior and the Smoking Habit (Pilot Study).]** *Revista de Psicologia General y Aplicada* 34(157): 225-243, 1979, Spanish.

The possibility of applying deductive techniques in testing a model of learning theory through analysis and modification of the smoking habit was investigated. Five smokers participated voluntarily in a group which learned to analyze the eliciting stimuli and causal factors for each cigarette smoked; methods of controlling environmental stimulus; and strategies for modifying their train of thought. They were also instructed in relaxation techniques in a series of steps in an aversive program. During the first four treatment sessions, a contingency contract system was implemented. All participants quit smoking during the intensive phase of the treatment. Four continued without smoking, and were still abstinent after 3 months. These results appear to confirm the effectiveness and convenience of applying scientific concepts to the problems of daily living. New objectives for investigation in this area are suggested.

82-0855. Delahunt, J. P. **A Smoking Cessation Treatment Investigation: A Replication and Assessment of Therapist Effect.** Doctoral Dissertation, Purdue University, West Lafayette, Indiana, University Microfilms International 81-13667, December 1980, 142 pp.

A smoking cessation treatment package shown to be successful in achieving and maintaining smoking reduction and abstinence was replicated by developing a therapist manual and training a therapist to apply the treatment procedures. The therapist effect was assessed using the dependent measures of cigarette consumption and a group processes scale. Female subjects (n=24) who had smoked at least 20 cigarettes a day for a minimum of 1 year were assigned to two treatment groups and one "expectation" control group of eight subjects each. Treatment group one was led by a female therapist who was trained in applying the treatment procedures; group two was led by a male therapist who had conducted the combined treatment group in the previous study. Both therapists used the therapist manual developed for this study. Treatment subjects were given manuals that contained rationales for treatment, specific treatment procedures, and homework assignments. Each group met for six 1 1/2 hour sessions over a 3-week period. Followup data were collected at 10 days, 1 month, 3 months, and 6 months after the quit day. The principal dependent measure was the number of cigarettes smoked per day as a percentage of the baseline rate. While the untreated control group maintained a consistent mean smoking rate from baseline through 6-month followup, treatment group one produced an overall reduction rate of 38 percent and an abstinence rate of 25 percent. Treatment group two produced an overall reduction rate of 75 percent and an abstinence rate of 62.5 percent, indicating that the goal of replication was achieved. Evidence that differences in group processes between treatment groups contributed to the significant differences in treatment outcome was provided by data from the Group Environment Scale, and it was concluded that the more successful therapist was less controlling and more nondirective, which coincided with the theoretical purpose of the self-control and negative practice components of the treatment package. (Auth. Abs. Mod.)

82-0856. DiClemente, C. C. **Self-Efficacy and Smoking Cessation Maintenance: A Preliminary Report.** *Cognitive Therapy and Research* 5(2): 175-187, June 1981.

The measure of self-efficacy for smoking cessation maintenance was shown to have good internal consistency in a study which applied the construct of self-efficacy developed by Bandura to the problem of long-term maintenance of smoking cessation. A measure of self-efficacy for avoiding smoking was used to analyze the relationship between self-efficacy and subjects' ability to maintain posttreatment abstinence at a 5-month followup. Subjects, 29 male and 34 female, (mean age 35 years) were confirmed, heavy smokers who previously had quit smoking by three different procedures (aversion, behavior management, self-quitters). Subjects were administered the self-efficacy measure and a demographic and smoking history questionnaire an average of 4 weeks after quitting smoking. Maintenance was assessed at 5-month followup. Two-thirds of all subjects successfully maintained nonsmoking at followup with no group differences for success. Maintainers (n=42) did not differ from recidivists (n=21) on any demographic or smoking history variables. However, maintainers did show significantly higher self-efficacy scores than recidivists. (Auth. Abs. Mod.)

82–0857. Fee, W. M.; Stewart, M. J. **A Controlled Trial of Nicotine Chewing Gum in a Smoking Withdrawal Clinic.** *Practitioner* 226(1363): 148, 151, January 1982.

A smoking treatment program initially achieved greater cessation rates among subjects given nicotine chewing gum than among those given a placebo, but results during followup suggested a high relapse rate among the nicotine gum subjects. During the treatments, claims of smoking cessation were tested by blood carboxyhemoglobin measurements. Those who finished the courses received questionnaires at 3, 6, and 12 months posttreatment, inquiring whether they were still abstinent, or what their current smoking habits were. Nicotine gum appeared more effective than the placebo in promoting initial smoking cessation. However, the information collected during the 1-year followup period suggested that the nicotine subjects had a high relapse rate, especially for the first 3 months, and that it was greater than that of the placebo subjects up to, if not after, 6 months following the end of treatment.

82–0858. Griffith, E. E. **Biofeedback: A Possible Substitute for Smoking.** Doctoral Dissertation, Utah State University, Logan, University Microfilms International 81-21910, 1981, 200 pp.

A study of smokers given training in biofeedback found that some quit smoking and others reduced their consumption. First, the study investigated the individual and simultaneous physiological changes, i.e., electroencephalography, electromyography, heart rate, blood pressure, and skin temperature, that occurred during and immediately after the smoking of one cigarette. Second, the hypothesis that smoking frequency would decrease when individuals were trained via biofeedback procedures to increase 8-12 Hz occipital EEG activity as a substitute for smoking, was investigated. Three male, very heavy smokers' (35 or more cigarettes per day) and three male moderate smokers' (15-24 cigarettes per day) physiologies were monitored while smoking, while nonsmoking, and while they were provided with 8-12 Hz occipital EEG biofeedback training using a multiple baseline design. During the training period, when the smokers were given music feedback whenever they produced 8-12 Hz, four of the six smokers learned to increase the percent of time producing 8-12 Hz. Two of these four smokers were able to continue producing high levels of 8-12 Hz activity without the use of biofeedback equipment. These smokers had quit smoking completely at the end of a 6-month followup period and reported they were still not smoking at the 8-month followup period. The four smokers who could not increase their 8-12 Hz activity without the use of 8-12 Hz auditory feedback showed a decreased frequency of cigarette smoking at the 6-month followup period. Several possible hypotheses were given which might account for the cessation or reduction of smoking among the subjects. (Auth. Abs. Mod.)

82–0859. Hill, R. W. **Hypnosis: A Group Treatment for Smoking, Obesity and the Perception of Stress.** Doctoral Dissertation, Virginia Commonwealth University, Richmond, University Microfilms International 81-18963, April 1981, 108 pp.

The generalizability of an individual hypnotic treatment model for smoking to group treatment for smoking behavior, and the application of this model on treatment for obesity and self-perceived stress is examined. The generalizability of the hypnotic treatment model to group treatment of smoking behavior was confirmed. Subjects in the treatment group showed a significant reduction of smoking behavior as compared with the control group. The treatment group demonstrated a 76 percent success rate for the cessation of smoking as compared with a 4 percent success rate in the control group. High success rates were also achieved in the treatment for weight reduction and stress reduction. The results of the present study would indicate that the individual hypnotic treatment model will generalize to groups and will further generalize other populations for treatment of such conditions as obesity and stress. (Auth. Abs. Mod.)

82–0860. Howley, T. J. **A Comparative Evaluation of Aerobic Exercise and Self-Management Strategies in the Treatment of Cigarette Smoking.** Doctoral Dissertation, West Virginia University, Morgantown, University Microfilms International 81-18423, 1981, 111 pp.

Three therapeutic clinical intervention programs designed to assist cigarette smokers to reduce or quit smoking are evaluated. Thirty-six subjects, 7 males and 29 females (21 to 50 years), with a mean daily smoking rate of 26.5 cigarettes and a mean smoking history of 16.4 years, were recruited. All were randomly assigned to one of four experimental conditions: exercise alone, self-management, combination of exercise and self-management, and a delay of treatment control group. Subjects were provided treatment through two smoking clinics run at different times. Data were analyzed both as a control condition (prior to their treatment) and as treatment subjects (after treatment). An evaluation of the validity of self-reported information was based on compliance to exercise through measures obtained at pre- and postexercise testings, and smoking reduction or abstinence through analysis of saliva samples. Seven of the original subjects failed to complete the study. Results are consistent with previous research in smoking cessation in which greater short-term reduction in cigarette smoking was seen in all treatment conditions over the control condition at posttreatment, with gradual recidivism in the 6-month followup period. At posttreatment, subjects in all treatment conditions reported a significant reduction in cigarette smoking over the delay of treatment control. However, the treatment groups did not differ significantly from each other. It was concluded that: (1) exposing subjects to several treatment strategies may be less effective than training them in a simple treatment, (2) treatments involving fewer components may result in increased compliance, and (3) simple treatments may be more cost effective even with relatively low success rates.

82-0861. Hymowitz, N.; Lasser, N. L.; Safirstein, B. H. **Effects of Graduated External Filters on Smoking Cessation.** *Preventive Medicine* 11(1): 85-95, January 1982.

The use of graduated filters for smoking cessation did not yield significantly different quitting rates from control conditions in a study involving adult smokers. Quit smoking rates were compared in adults assigned randomly to one of three experimental conditions (Group 1, quit smoking on own; Group 2, placebo filters; and Group 3, graduated filters). Two months later, quit smoking rates for Groups 1, 2, and 3, respectively, were 21, 14, and 26 percent, differences which did not achieve statistical significance. These data fail to document the utility of graduated filters for smoking cessation. Over 60 percent of the initial quitters remained abstinent more than 1 year later, with long-term quitters revealing significant reduction in carbon monoxide and pulmonary symptoms, and improvement in midmaximal flow rate (MMF50). Continuing smokers, some of whom used Filter 4 (the strongest of four available progressively stronger filters) for over 1 year, did not reveal significant improvement of MMF50 and did not achieve changes in carbon monoxide and pulmonary symptoms of the same magnitude as successful quitters. (Auth. Abs. Mod.)

82-0862. Koman, S. L. **Treatment of Smoking Addiction: The Effect of Treatment Goal and Properties of Addiction on Achieving and Maintaining Smoking Modification.** Doctoral Dissertation, Duke University, Department of Psychology, Durham, North Carolina, University Microfilms International 81-24804, 1981, 275 pp.

The problem of relapse is examined with respect to the effect of the goal of treatment (abstinence versus reduction) and the properties of smoking addiction (physiological, behavioral, and affective) on the individual's ability to maintain quitting or reduction of smoking. Forty-five subjects, aged 20 to 61, participated in one of two treatment programs; both involved attendance at eight treatment sessions, six small group sessions, and two individual sessions, over a 5-week period. All were instructed in treatment techniques including self-monitoring, problem solving, decisionmaking, relaxation and imagery, and maintenance. The two programs differed in treatment goal; one was designed to produce and maintain smoking abstinence, the other, to reduce smoking. Findings indicate that although overall success in smoking reduction was superior in the abstinence group, it was unevenly distributed; some enjoyed total or near success while others did poorly. The controlled smoking group achieved and maintained a significant reduction in carbon monoxide content. However, relapses in other smoking factors were evident in all other cases. Elements involving affective concerns were most often associated with successful treatment, underscoring the role of smoking as a means of reducing negative affect and anxiety.

82-0863. Markelova, V. F.; Belitskaya, R. A.; Rudnev, S. G. **O Nekotorykh Mekhanizmakh Vliyaniya Akupunktury na Simpato-Adrenalovuyu Sistemu u Kuril'Shchikov Tabaka. [Concerning Some Mechanisms of Acupuncture Effect on the Sympathicoadrenal System of Smokers.]** *Zhurnal Nevropatologii i Psikhiatrii imeni S.S. Korsakova* 81(2): 117-122, 1981, Russian.

Daily excretion of catecholamines in urine was determined in healthy men and women, aged 20 to 60 years, who have been smoking 20 to 30 cigarettes/day for at least 7 years. Determinations were carried out in: (1) 15 smokers a few days before acupuncture treatment while they continued regular smoking; (2) 36 smokers in the 15- to 18-hour period of abstinence from smoking immediately preceding the treatment, and again after five acupuncture sessions of 15 to 30 minutes each; and (3) 20 healthy nonsmoking controls. The norepinephrine and dopamine (DA) contents in urine of the first group of smokers were found to be significantly higher than in the controls. After the treatment, 17 out of 36 smokers stopped smoking and 19 others reduced their smoking to 3 to 6 cigarettes/day. Simultaneously with the positive therapeutic effect in 17 patients, their epinephrine, norepinephrine, and DA excretions decreased to normal values. In the group of 19 smokers, excretion of the first two catecholamines and that of DOPA also became normal after treatment, while DA level remained significantly higher than normal. Thus, it was shown that acupuncture effect depends significantly on the initial functional activity of the sympathicoadrenal system.

82-0864. Marlatt, G. A.; Gordon, J. R. **Determinants of Relapse: Implications for the Maintenance of Behavior Change.** In: Davidson, P. O.; Davidson, S. M. (Editors). *Behavioral Medicine: Changing Health Lifestyles.* New York, Brunner/Mazel, Inc., 1980, pp. 410-452.

Maintaining abstinence after quitting smoking can be very difficult. Viewed from the perspective of relapse rates, it would appear that heroin, alcohol, and tobacco are equally addicting. Yet physical withdrawal symptoms are rarely cited as factors leading to relapse in use of these substances. Study data indicate that the major determinants of smoking relapse fall into only three categories: social pressures, coping with negative emotional states, and coping with interpersonal conflict (anger and/or frustration). For smokers, the primary setting for relapse was their home (44 percent), followed by the work environment (19 percent), restaurants and bars (13 percent), and parties (6 percent). Evening was most often cited as the time of relapse. Data show that a single slip usually turns into "full-blown" backsliding. Effective maintenance of a smoking cessation behavior change requires that the smoker be taught cognitive recognition and behavior analysis, subtleties and impacts of mini-decisionmaking, and coping responses to relapse stimuli. Behavioral training, which includes coping with abstinence violation, self-efficacy factors, lifestyle intervention, programmed relapses, defusing relapse stimuli, and minimizing the impact of slips into previous smoking behaviors, is discussed.

82–0865. Orleans, C. T.; Shipley, R. H.; Williams, C.; Haac, L. A. **Behavioral Approaches to Smoking Cessation—I. A Decade of Research Progress 1969-1979.** *Journal of Behavior Therapy and Experimental Psychiatry* 12(2): 125-129, June 1981.

A topical bibliography presented in a subsequent article (*Journal of Behavior Therapy and Experimental Psychiatry* 12(2): 131-144, June 1981) covering the past decade of research into behavioral approaches to smoking cessation is described. Using multiple searches including *Psychological Abstracts*, over 300 references were compiled from over 50 psychology, medicine, and related behavioral science journals. All references were comprehensively coded to each of 28 topical areas of interest to behavioral researchers and clinicians. Topics include specific behavioral smoking cessation techniques (e.g., aversive smoking, relaxation techniques, stimulus control), treatment problems (e.g., maintenance problems and procedures, treatment "side-effects"), and important research issues (e.g., predictors of treatment success, physiological correlates of smoking behavior, therapist and treatment factors influencing outcome). An index defining each topic and listing by number all articles related to each topic is included with the bibliography. With this index, users with specialized interests can quickly locate all citations concerning a particular topic or combination of topics. Directions for using the bibliography are given along with guidelines for extending and adopting it for personal use. (Auth. Abs.) See also 82-0866.

82–0866. Orleans, C. T.; Shipley, R. H.; Williams, C.; Haac, L. A. **Behavioral Approaches to Smoking Cessation—II. Topical Bibliography 1969-1979.** *Journal of Behavior Therapy and Experimental Psychiatry* 12(2): 131-144, June 1981.

A topical bibliography covering the past decade of research into behavioral approaches to smoking cessation is presented. Using multiple sources, including *Psychological Abstracts*, 335 references were compiled from over 50 psychology, medicine, and related behavioral science journals. All references were comprehensively coded for each of 28 topical areas of interest to behavioral researchers and clinicians. Topics include specific behavioral smoking cessation techniques (e.g., aversive smoking, relaxation techniques, stimulus control), treatment problems (e.g., maintenance problems and procedures, treatment "side-effects"), and important research issues (e.g., predictors of treatment success, physiological correlates of smoking behavior, therapist and treatment factors influencing outcome). An index defining each topic and listing by number all articles related to each topic is included with the bibliography. With this index, users with specialized interests can quickly locate all citations concerning a particular topic or combination of topics. A preceding article (*Journal of Behavior Therapy and Experimental Psychiatry* 12(2): 125-129, June 1981) gives guidelines for expanding this bibliography for personal reference use. (Auth. Abs.) See also 82-0865.

82–0867. Pederson, L. L.; Baldwin, N.; Lefcoe, N. M. **Utility of Behavioral Self-Help Manuals in a Minimal-Contact Smoking Cessation Program.** *International Journal of the Addictions* 16(7): 1233-1239, 1981.

A smoking cessation manual, *Break the Smoking Habit: A Behavioral Program for Giving Up Cigarettes* by O. F. Pomerleau and C. S. Pomerleau (Champaign, Illinois, Research Press, 1977) was found to be potentially useful, while no firm conclusions were reached for a manual *Become an Ex-Smoker* by B. G. Danaher and E. Lichtenstein (Englewood Cliffs, New Jersey, Prentice-Hall, Inc., 1978). The Danaher and Lichtenstein manual is described as presenting a program combining stimulus control procedures, deep muscle relaxation, and aversive conditioning. Much emphasis is put on maintenance of nonsmoking. The Pomerleau and Pomerleau manual also offers a broad spectrum approach, utilizing stimulus control, deep muscle relaxation, and covert conditioning. The effectiveness of these two commercially available manuals was evaluated under minimal therapist contact conditions. Forty cigarette smokers who volunteered to participate in a self-help quit smoking program were randomly assigned to one of three conditions: 18 to Pomerleau and Pomerleau's manual, 13 to Danaher and Lichtenstein's manual, and 9 to a waiting-list control. Six-month followups indicate that the former manual offers a useful treatment approach, while no firm conclusions can be made regarding the latter manual. (Auth. Abs. Mod.) See also 78-0370 and 80-0834.

See also, **82–0808, 82–0815**

TOBACCO PRODUCT ADDITIVES

82–0868. Bonnet, J.; Lebet, J.-P. **Method for Aromatizing Tobacco Smoke.** United States Patent No. 4,311,156, January 19, 1982, 3 pp.

A method of aromatizing tobacco smoke drawn through a cigarette mouthpiece is disclosed. The mouthpiece is characterized by using a porous magnesium silicate and/or magnesium silicate hydrate charged with aromatic substances selected from the group consisting of menthol and synthetic or natural tobacco extracts. The magnesium content is at least 4 percent, and preferably 8 to 25 percent by weight (with respect to dry weight). This method is intended to overcome problems that occur in the manufacture of mentholated cigarettes, making it possible to manufacture mentholated filter cigarettes without contaminating the production plant, and requires relatively small quantities of aromatized substances, giving the aromatized cigarettes a longer storability.

82–0869. Graefe, A. **Low-Tar Cigarettes—Trendsetters With Taste.** *Tabak Journal International* 6: 482, 484, December 1981.

A flavoring is described which may allow tobacco manufacturers to enhance the taste of weak tobaccos. The reduction of nicotine and tar in low-tar cigarettes, a subject of especially great interest in the United States, brings with it noticeable losses in taste, for which compensation must be provided. As one of the internationally leading manufacturers of flavorings, Haarmann & Reimer is the first producer in this sector to create an extremely adhesive basic flavoring which, while not increasing nicotine and only slightly raising the tar level, enhances taste many times over. Yet the employment of this "building block" provides the tobacco manufacturer with sufficient freedom to compose its own blends to accentuate and typify the total flavor impression of its cigarette. (Auth. Abs. Mod.)

82–0870. Hiroshi, M.; Matsue, S. **Flavorant Composition for Tobacco, Method for Producing the Same Tobacco Product Comprising Said Composition.** United States Patent No. 4,318,417, March 9, 1982, 7 pp.

A flavorant composition for tobacco which is produced by heating a certain synthesized inorganic ion-exchangeable compound containing zirconium or titanium metal together with tobacco flavorant in organic solvent to adsorb the flavorant to the exchanger is described. This flavorant composition may be applied advantageously to a filter cigarette so as to be added to the portion of the filter tip in the form of a triple-filter or a dual-filter. The flavorant in the flavorant composition having been added to a filter tip scarcely volatilizes when it has only been left as it is; however, such flavorant may be released readily from the composition by wet smoke passing through the filter tip when smoked, whereby the aroma and taste of the flavorant may effectively be produced. The amount of tobacco flavorant capable of producing moderate aroma and taste is far less than that of flavorant that has customarily been added to the portion of blended cut tobacco. The ion-exchanger used in the invention does not affect the essential aroma and taste of tobacco flavorants. (Auth. Abs.)

82–0871. Houminer, Y.; Grubbs, H. J. **Smoking Compositions.** United States Patent No. 4,312,368, January 26, 1982, 7 pp.

Tobacco and nontobacco smoking compositions which contain a heterocyclic-hydroxy-substituted carboxylate compound as a flavorant additive are described. In one of its embodiments, this invention provides tobacco compositions which contain a heterocyclic-hydroxy-substituted alkanoate flavorant additive such as ethyl 2-(2-butyl)-3-hydroxy-3-methyl-3-(3-pyridyl)-propionate. Under smoking conditions the heterocyclic-hydroxy-substituted alkanoate additive and its pyrolysis products flavor the mainstream and sidestream smoke. (Auth. Abs.)

82–0872. Kallianos, A. G.; Southwick, E. W. **7-Methyl-1,2-Benzopyrone as a Tobacco Flavorant.** United States Patent No. 4,319,585, March 16, 1982, 3 pp.

A process to ameliorate the restrictive conditions inherent in blending tobacco by utilizing a new class of additive materials is described. When applied to the tobacco or the tobacco product, the additives improve and enhance the flavor and aroma of the tobacco smoke emitted from these products thereby enhancing the pleasure derived by the consumer. The invention also provides a process for enhancing or otherwise improving the flavor and aroma of certain domestic, oriental, reconstituted, or synthetic tobaccos and blends thereof which may be deficient in said flavor or aroma. In addition, it provides a smoking product, such as cigarettes, cigars, or pipe tobacco, and a process for forming same whereby the flavor and aroma of the tobacco smoke include characteristics similar to those imparted by deertongue or tonka, and thus are enhanced during smoking.

82–0873. Mintz, J. **Does It Taste Like a Cigarette Should?** *Science 81* 2(9): 95-96, November 1981.

A large variety of additives are available for use in tobacco, but concern has been expressed that some may pose possible health hazards. Cocoa, licorice, and fruit juices appear to be the most common flavorings. Little is known of the possible effects of licorice and fruit, but in 1977 the National Cancer Institute stated that condensed tar from cocoa-flavored cigarettes caused more tumors in mice than did tar from unflavored cigarettes. Another flavoring, coumarin, has been shown to harm the liver and other organs of laboratory animals. Glycerol, used to keep tobacco moist, can generate acrolein during burning. Acrolein can impair normal clearing of lungs. Other additives may also be potentially dangerous.

82–0874. Mussinan, C. J.; Mookherjee, B. D.; Vock, M. H.; Schmitt, F. L.; Granda, E. J.; Vinals, J. F.; Kiwala, J. **Uses of Hexenol Derivatives in Augmenting or Enhancing the Aroma or Taste of Smoking Tobacco Compositions and Smoking Tobacco Articles.** United States Patent No. 4,319,586, March 16, 1982, 23 pp.

Processes for augmenting or enhancing the aroma or taste of a smoking tobacco are described. The process includes admixing with smoking tobacco an aroma or taste augmenting or enhancing quality of at least one substance selected from the following: (1) a composition of matter consisting essentially of cis-3-hexenal, cis-3-hexenyl-cis-3-hexenoate, trans-2-hexenal, and cis-3-hexenol; (2) a composition of matter consisting essentially of cis-3-hexenal, cis-3-hexenyl-cis-3-hexenoate, cis-3-hexenyl formate, and trans-2-hexenal; (3) a composition of matter consisting essentially of cis-3-hexenal, cis-3-hexenyl-cis-3-hexenoate, cis-3-hexenol, cis-3-hexenyl formate, and trans-2-hexenal; and (4) cis-3-hexenyl methyl ether. Smoking tobacco compositions produced according to the above process are detailed. Also described are smoking tobacco articles comprising a cylindrically shaped body of smoking tobacco; a wrapper in intimate contact with said cylindrically shaped body along its length; a filter in intimate contact with one end of said cylindrically shaped body; and in intimate contact with either said filter, said wrapper, or said cylindrically shaped body at least one substance selected from the additives listed above. (Auth. Abs. Mod.)

82–0875. Park, J. Y.; Yang, K. K.; Yu, K. K. **Pilt'o Ploge Mentol Ch'omgahyogwae Kwanhan Yon'gu. [A Study on the Effect of Menthol Added to the Filter.]** *Journal of the Korean Society of Tobacco Science* 1(2): 149-151, November 1979, Korean.

A study focuses on a method for adding menthol to both the filter part and the shred part of a cigarette at once, compared to adding menthol to the filter part only or to the shred part only. The first step described is to make a menthol filter plug by adding menthol to an acetate tow, and then to use this plug to produce the mentholated cigarette. To make the menthol filter plug, spray 76-92 mg of the 13 percent menthol liquid per 100 mm filter plug on the cellulose acetate tow, and then attach the menthol filter plug to the nonmenthol shred of a cigarette. The method is much easier and simpler for manufacturing mentholated cigarettes. Furthermore, it shows a great improvement in menthol distribution: 9 percent improvement right off the production, 4.3 percent after 1 week, and 1.3 percent after 4 weeks. A table is included which shows the menthol distribution at various aging periods by various methods for adding menthols. Comparatively, adding menthol to the filter part only shows better distribution of menthol at various aging periods than adding menthol to the shred part only; however, adding menthol by using a menthol filter plug has shown the most effective distribution of menthol.

82–0876. Patarra, S. F. **Cigarette With Removable Smoke-Modifying Cartridges.** United States Patent No. 4,319,590, March 16, 1982, 4 pp.

A cigarette having a removable smoke-modifying cartridge or cartridges in one end enabling the smoker to select whether to filter or otherwise modify the cigarette smoke is described. The invention embodies (1) a smoke-filtering cartridge snugly but slidably received in the mouthpiece or tip of the cigarette and provided with a pull string which extends to the free end of the mouthpiece; and (2) a flavor—introducing filter cartridge in the mouthpiece just beyond the smoke-filtering cartridge, also provided with a pull string. The flavor filter contains a volatile flavoring agent which adds a suitable flavor, such as menthol, lemon, or cherry to the smoke flowing through it.

82–0877. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **2-Oxabicyclooctane Derivatives.** United States Patent No. 4,231,940, November 4, 1980, 44 pp.

The use of 2-oxabicyclooctane derivatives to augment or enhance flavor and fragrance is described for consumable materials such as smoking tobaccos, as well as perfumes, perfumed articles, colognes, foodstuffs, chewing gums, toothpastes, and medicinal products. The derivatives and their cyclohexene alkyl and alkenyl carbinols and esters impart hay, fruity, and herbaceous aroma and taste before and during smoking in the mainstream and sidestream smoke. These additives may be used in smoking tobacco or in suitable substitutes. Other flavor and aroma substances may also be added, either separately or mixed with one or more of the additives presented. The derivatives may be added to the smoking tobacco material, the filter, the leaf, or the paper wrapper, or to a filter which forms part of the smoking article. A suggested proportion by weight of the additives to smoking tobacco material is between 50 ppm and 1,500 ppm. The additives may be applied by spraying or dipping, or under certain circumstances by a suitable applicator. In addition to smoking tobacco, the additives may also be used for other tobacco products formed from sheeted tobacco dust or fines.

82–0878. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Fabric Softener Compositions and Articles Containing 2-Oxabicyclooctane Derivatives and Processes for Preparing Same.** United States Patent No. 4,226,729, October 7, 1980, 30 pp.

Described is a fabric softening composition whose oxabicyclooctane derivatives are used to augment or enhance the flavors and/or fragrances of such consumable materials as perfumes, perfumed articles, colognes, foodstuffs, chewing gums, toothpastes, medicinal products, and smoking tobaccos. Specifically, in smoking tobacco, these derivatives enhance the clove bud-like characteristics by imparting a clove bud aroma and taste nuance prior to and on smoking in the mainstream and in the sidestream smoke, regardless of variations in the tobacco components of the blend or the nature of the filter used. This additive is also usable with tobacco substitutes, e.g., dried lettuce leaves. In addition to the oxabicyclooctane derivatives of the invention, additional flavoring additives may be added to the smoking tobacco material, to the filter, or to the leaf or paper wrapper. A specific list of synthetic compounds and natural oils is provided.

82–0879. Wilson, R. A.; Schreiber, W. L.; Mookherjee, B. D.; Kiwala, J.; Vinals, J. F.; Vock, M. H.; Stork, G.; Schmitt, F. L. **2,6,6-Trimethyl-α(iso)propenyl-1-cyclohexene-1-methanol and 1,3 cyclohexadiene-1-methanol.** United States Patent No. 4,255,293, March 10, 1981, 32 pp.

The advantageous properties for 2,6,6-trimethyl-α(iso)propenyl-1-cyclohexene-1-methanols and 1,3-cyclohexadiene-1-methanols and their stereoisomers and geometric isomers, insofar as their organoleptic properties are concerned, are described. A variety of aromas and flavors are possible with these compounds, depending on the selected compositions of the admixed synthetic materials, such as beta-methylcinnamaldehyde, eugenol, dipentene, etc., and natural oils. Prior to smoking, various sweet, hay-like, woody, or floral aromas are detected and upon smoking, various sweet, musty, woody aromas and flavors, as well as astringent nuances in the mainstream and sidestream smoke, are produced.

82–0880. Yoshida, T. **Methyl Substituted Oxobicyclo-4,4,0-Decane Derivatives, Process for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,320,772, March 23, 1982, 114 pp.

Described are methyl substituted oxobicyclo-4,4,0-decane derivatives and uses thereof for their organoleptic properties in augmenting or enhancing the aromas or tastes of perfumes, perfumed articles, foodstuffs, chewing gums, smoking tobaccos, toothpastes, medicinal products, chewing tobacco, and smoking tobacco articles, as well as processes for preparing same. The invention provides an organoleptically improved smoking tobacco product and appropriate additives. Manufacturing methods that overcome problems in creating or augmenting the specific desired sweet, fruity, berry-like, woody, and floral flavor characteristics of natural tobacco (prior to smoking and, on smoking, in the mainstream and in the sidestream), as well as cooling effects, are given. These properties may be readily controlled and maintained at the desired uniform level regardless of variations in the tobacco components of the blend. It further provides improved tobacco additives and methods whereby various desirable natural aromatic tobacco flavoring characteristics may be imparted to smoking tobacco products and may be readily varied and controlled to produce the desired uniform flavoring characteristics.

TOBACCO MANUFACTURING AND PROCESSING

82–0881. Abdallah, F. **Flavor—What Makes Tobacco Tobacco.** Paper presented at General Session of the four working groups, 7th International Tobacco Scientific Congress, CORESTA, Philippines, November 10-14, 1980, 11 pp.

Tobacco flavor is an end product of a combination of genetics, soil, climate, cultivating, curing, and production methods. Consumer tastes, tobacco technology, chemistry, blending, and flavorings further influence the flavor of cigarette smoke. The taste of mainstream smoke is due to complex mixtures of nonvolatile substances of large mass, present in small numbers, along with volatile odor compounds which are small in mass but present in large quantities, and special groups of taste modifiers and flavor potentiators. A direct correlation between components and flavor cannot be established. In a recent survey of the cigarettes sold in the United States, over 30 brands deliver 1.3±0.1 mg nicotine per cigarette, and among these, 19 brands deliver 12±1 mg tar per cigarette. However, the acceptability of these cigarettes in terms of sales ranges from best seller to near failure, regardless of the category of the brand—milds, lights, extra lights, or ultra lights. The complex nature of smoke is illustrated by the total particulate matter (8 percent by weight) in which there are over 500 substances contributing to its flavor. Efforts to correlate a component or group of components to flavor may be an oversimplification. Theories and problems of sensory testing with respect to product development, and the fate of nicotine during smoking, are discussed.

82–0882. Dale, R. W.; Rooney, J. J. **Smoking Products.** United States Patent No. 4,317,460, March 2, 1982, 7 pp.

Catalysts for the low temperature oxidation of carbon monoxide to carbon dioxide, used in smoking product filters, are described. The catalysts are carried upon a support which should be microporous. The catalysts may include mixtures of tin or tin compounds with other catalytic material. The catalysts may involve a Redox mechanism. The catalysts exhibit resistance to deactivation caused by contact with water. (Auth. Abs.)

82–0883. Keith, C. H.; Karstedt, B. D. **Smoking Compositions.** United States Patent No. 4,319,591, March 16, 1982, 5 pp.

Smoking compositions having improved taste and odor are prepared by incorporation of minor amounts of proteins and protein hydrolysates, such as egg albumen, gelatine, collagen, soybean protein, casein, zein, and gliadin. At least 80 percent of the weight is derived from aliphatic and heterocyclic amino acids. The compositions also yield a smoke which is less irritating to the smoker. Smoking products may be prepared entirely from the described smoking composition or from any desired combination of the composition with tobacco, reconstituted tobacco, or other tobacco replacements. (Auth. Abs. Mod.)

82–0884. Moser, I. S. **Smoking Article.** United States Patent No. 4,319,587, March 16, 1982, 6 pp.

The invention presented comprises an improved smoking article, such as a cigarette or cigar, having means for controlling quickly, accurately, and selectively, the extent of consumption of the smoking article, to assist a habitual smoker in voluntarily curtailing or terminating his smoking habit. The control means comprises a generally tubular outer wrapping on the article on which are marked, printed, or otherwise imposed, spaced indicia in the nature of a scale, extending longitudinally of the article, and an annular control indicator mounted telescopically on the article and slidable axially relative thereto. The indicia may be accompanied by contrasting designs or markings, to provide quick, accurate, and carefully selected voluntary control by the smoker of the extent of consumption of the smoking article. The annular control indicator contrasts visually relative to the smoking article. (Auth. Abs.)

See also, **82–0655**

TOBACCO ECONOMICS

82–0885. Canadian Tobacco Grower. **Smoking in Japan.** *Canadian Tobacco Grower* 29(4): 75, 77, April 1981.

Statistics on Japanese tobacco production, sales, imports, and consumption are presented graphically, as are the attitudes of smokers about quitting, and of Japanese adults about the rights of nonsmokers. Japan is the fourth largest producer of cigarettes in the world, behind China, the United States, and the Soviet Union. Although it is shown that for the period from 1967 to 1979, the percentage of male smokers declined slightly, there are still many more male smokers than females (three out of four men, and only one out of six women smoke) and those who smoke are smoking more. There has been an 81 percent per capita increase in total consumption since 1961, possibly due in part to the popularity of the filter cigarette, and the continued low cost of cigarettes. Spending patterns indicate the extent of the problem, as Japanese retail sales of cigarettes exceed figures for such staples as meat and rice. More than half of the smokers in a 1978 poll, in spite of health warnings, have no intention of quitting. In addition, general attitudes on "nonsmokers rights" showed that less than 30 percent of those questioned were in favor of the movement.

82–0886. Farmline. **Third World Smoke Signals Cheer Tobacco Growers.** *Farmline* 1(5): 10-11, August 1980.

Because of a steady decline in U.S. per capita tobacco use during the last decade, and the drastic reduction in sales to Japanese, Egyptian, and European markets, tobacco growers are looking for new markets in the Third World. However, competition from Brazil, Korea, Thailand, Malawi, and Zimbabwe, and the realization that Third World demand for tobacco won't necessarily translate into long-term economic salvation for the U.S. farmer, softens the growing optimism. Statistics reveal a per capita consumption drop from 4,345 to 3,967 cigarettes from 1963 to 1978, less tobacco used in the manufacture of cigarettes, and more foreign tobacco "filler" used in domestic cigarettes. Looking ahead, antismoking campaigns are certain to continue to impact on sales by 1 to 2 percent per year. It is speculated that by 1990, per capita use could decline as much as 25 percent.

82–0887. Howland, K. E. **Long-Term Trends Will Continue in '82.** *Tobacco Reporter* 109(1): 41-42, January 1982.

It is expected that in 1982 the world tobacco industry will experience a steady increase in consumption, a moderate increase in trade, and a further decrease of stocks held by manufacturers in major consuming nations. The production of cigarettes is expected to increase at a rate of roughly 2 percent, with the rate being larger in the more advanced developing nations of Asia and Latin America. Some developed countries may display little growth in cigarette consumption. In recent years, developing nations have acquired a larger proportion of tobacco production, exports, and consumption relative to the proportion held by developed and centrally planned nations. The exports of the U.S. tobacco trade are expected to increase in 1982. For 1982, the net contribution of tobacco to the balance of trade of the United States may be approximately 2.4 billion dollars.

82–0888. Koerner, M. **1981 Fine for German Cigarettes, But Taxes and Expenses Will Hurt in 1982.** *Tobacco International* 183(26): 17, 20, December 25, 1981.

The German cigarette industry experienced an estimated 2 percent growth in 1981, but tax/price increases may adversely affect consumption in the future. The industry has also faced rising labor costs, as well as high fringe benefits and higher costs for leaf tobacco and energy. A 5 percent price increase per pack, effective January 1, 1982, is expected to have only a moderate effect on sales. However, there is to be a tobacco tax increase of almost 40 percent in the middle of 1982, which will result in an average 33 percent price increase per pack. Since disposable incomes may be lower due to the effect of inflation, consumption may be adversely affected. German cigarette manufacturers anticipate a sales reduction of as much as 20 percent, as more smokers switch to less expensive hand-made or roll-your-own cigarettes. Border sales are expected to increase, since the same cigarette brands can be cheaper in neighboring nations due to a lower level of excise duties. By mid-1982, German cigarettes will be the most highly taxed consumer commodity. The number of German smokers has remained more or less stable, at approximately 18 million smokers, or roughly 38 percent of the adult population.

82-0889. Lewit, E. M.; Coate, D. **The Potential for Using Excise Taxes To Reduce Smoking.** National Bureau of Economic Research, Inc., Cambridge, Massachusetts, NBER Working Paper Series, Working Paper No. 764, September 1981, 44 pp.

The potential for reducing cigarette smoking through increases in cigarette excise taxes is examined by estimating the price elasticity of demand for cigarettes. Using information on individual smoking behavior for a sample of adults in the 1976 Health Interview Survey, the adult price elasticity of demand for cigarettes is estimated to be -.45. Moreover, the data show that price has its greatest effect on the smoking behavior of young males, and that it operates primarily on the decision to begin smoking regularly, rather than via adjustments in the quantity of cigarettes smoked by smokers. Therefore, if future reductions in cigarette smoking are desired, Federal excise tax policy can be a potent tool to accomplish this goal, but only in the long run. An excise tax increase, if maintained in real terms, would discourage smoking participation by successive cohorts of young adults; and those reduced smoking levels would be reflected in aggregate smoking as these cohorts mature. In the short run, however, the impact of an excise tax increase on aggregate cigarette consumption would be relatively small. (Auth. Abs.)

82-0890. Maxwell, J. C., Jr. **Maxwell Report: Strong Finish.** *Tobacco International* 183(26): 15, December 25, 1981.

The cigarette industry has continued to experience economic growth. It was estimated there would be a 2.6 percent increase in units in 1981. The consumption of low-tar cigarettes (15 mg tar or less), which had 48.5 percent of the market in 1980, is estimated to be over 60 percent for 1981, with ultralow-tar cigarettes (0 to 6 mg) at about 11 percent. The advisability of an increase in Federal excise taxes on cigarettes is discussed. It is argued that State and local governments presently receive about 4 billion dollars a year from taxes on tobacco. Consumption growth might be discouraged by a large increase in the Federal excise tax, resulting in reduced income for local governments from tobacco taxation.

82-0891. Maxwell, J. C., Jr. **Maxwell Cigar Report: Import Market Continues To Grow.** *Tobacco Reporter* 108(7): 52, 54, July 1981.

Cigar market statistics for 1980 indicate that premium cigars constitute one of the few growth factors in the industry. Volume for both large and small cigars declined from 1979 levels; from 4,265 million units to 3,952 million for large cigars, and from 1,433 million to 1,415 million for small cigars. However, the import market continues to grow, accounting for a little less than 3 percent of total unit volume but 14 percent of total retail dollar sales. John H. Swisher and Sons, marketers of King Edward cigars, continuously increases its share of the market. Other products doing fairly well include Consolidated Cigar's Dutch Masters, General Cigar's White Owl, American Brands' Antonio y Cleopatra, Bayuk's Phillies, and Lorillard's Eric group.

82-0892. Maxwell, J. C., Jr. **The Maxwell Report: Cigarette Sales Rose 2.6 Percent Last Year.** *Tobacco Reporter* 109(3): 42-43, March 1982.

The cigarette industry is expected to show a strong 2.6 percent gain in units by the end of 1981. Philip Morris has increased 6.0 percent, Reynolds is up about 3.6 percent, and the Liggett Group has increased 2.8 percent, primarily because of its generic products. It is interesting to note that Camel nonfilters are up some 2.6 percent, and the rate of decline is slowing for such brands as Pall Mall and Lucky Strike. The same occurred in Germany, where the market share of very low-tar and -nicotine products increased, but then reversed and gave way to more flavorful products. Low-tar cigarettes (6-15 mg) now account for nearly two-thirds of the market, with ultralow tars (0 to 6 mg) occupying 11 percent. State and local governments currently receive about 4 billion dollars annually from tobacco taxes. A proposed Federal excise tax increase is not recommended since the effect on consumption growth would mean a drop in revenue for State and local governments.

82-0893. Mazzone, J. S. **Low Tar and Nicotine Brands Counter Sales Dip in Brazil.** *Tobacco International* 183(26): 73, 78, December 25, 1981.

Cigarette sales in Brazil declined in 1981. Local manufacturers estimated that 1981 sales would show a 2.5 percent drop compared with 1980. In 1981, 139 billion cigarettes were to be produced compared with 142 billion in 1980. It is suggested that the reduced sales may have been due to economic recession. Price increases may also have had an effect. Low-tar and -nicotine brands are expected to increase their percentage of the Brazilian cigarette market in the future, as business competition continues in the low yield portion of the cigarette market. It is predicted that the low-tar brands, which were only 4.5 percent of sales in 1980, may achieve 25 percent in the future.

82-0894. Menzie, E. L.; Marshall, J. P. **The Canadian and U.S. Tobacco Production, Pricing, and Marketing Systems.** Department of Agricultural Economics, Extension Division, Cooperative Extension Service, Virginia Polytechnic Institute and State University, Blacksburg, Virginia, March 1981, 38 pp.

The Canadian and U.S. institutions used in allocating tobacco quota and in growing and harvesting, pricing, marketing, and selling tobacco are examined. Emphasis is given in the Canadian system to The (Province of) Ontario Flue-cured Tobacco Growers' Marketing Board. The U.S. institutions apply generally to flue-cured and Burley tobacco. The basic institutions for flue-cured are depicted in graphic form. The tobacco programs, output controls, production and price trends, and producer bargaining power are discussed. Data are presented on average size of quotas, number of grades, efficiency in use of sales-floor space, and cost of marketing. (Auth. Abs.)

82–0895. Miller, R. H. **Reduced Crop Expected in '82.** *Tobacco Reporter* 109(1): 43-44, January 1982.

Tobacco production in the United States showed increases during 1981, and cigarette output is expected to attain a record 726 billion pieces for 1981, or roughly 12 billion more than in 1980. Consumption of cigarettes is still moving toward low-tar brands. The industry continues to be affected by antismoking controversy and laws. Several States increased excise taxes during 1981. The increased tobacco production in 1981 as well as greater carryover has meant that total supplies for the 1981/82 marketing year are approximately 5 percent higher than last year. The flue-cured supply is about 3 percent greater in 1981/82 than in the previous year. Current supplies are almost equivalent to 3 years' consumption. An editor's note mentions that the U.S. Department of Agriculture has put the 1982 flue-cured marketing quota at the same level as in 1981, the effective 1982 quota reaching 979 million pounds. For Burley tobacco, the supply for 1981/82 is 9 percent greater than for the previous season. The 1982 marketing quota may be set at roughly the present season's 660 million pounds. The 1982 effective quota, however, may decrease. The tobacco program may face change in the future.

82–0896. Nelson, J. F. **Profits. Focus: The Tobacco Industry.** Richmond, Virginia, Bank of Virginia, Winter 1981, 16 pp.

The tobacco industry's contribution to the economy, past and present; the changing mix of manufactured products dictated by consumer preferences; and the strengths and weaknesses of our tobacco world markets are discussed. Sales of tobacco products generate a steady flow of tax revenues into Federal, State, and local coffers. At 8 cents per pack, the Federal tax poured 3.2 billion dollars into the Treasury, or almost 11 percent of all 1979 Federal excise taxes. Exports of tobacco and tobacco products totaled 2.15 billion dollars against imports of 463 million dollars in 1979, resulting in a record trade balance. In the area of production, consumer preferences for lower tar and nicotine is the most pronounced fall-out of the smoking and health issue. In 1970, only 3.4 percent of all cigarettes sold had less than 15 mg of tar; today the figure is almost 50 percent. Competition is intense in a field where 1 percent of the domestic market equals about 130 million dollars annually in revenue. Advertising expenditures for the top 20 brands (90 percent of total U.S. sales) exceed 500 million dollars. Cigarette sales abroad are growing at the rate of 4 percent per year in spite of protectionist obstacles posed by government-controlled markets. All companies eye the vast Chinese market with its estimated 200 million smokers; however, China's market is largely self-contained. Tobacco companies have shown resilience through diversification and a smokeless revival has become apparent. Among factors favorable to this industry segment are the development of milder products, more leisure time and outdoor activities, and smoking bans. Also discussed are the farming and auction sectors of the tobacco industry.

82–0897. Revue des Tabacs. **Nouvelles du Monde. [World News.]** *Revue des Tabacs* 55(312): 19-20, 23-25, 1980, French.

Statistical data, mostly for 1979, are presented on: (1) tobacco products manufactured in Denmark (1978), Canary Islands (1980), Greece, Mauritius, Ireland, Portugal, Switzerland, U.S.S.R., and the United States (1980, Philip Morris, Inc.); (2) tobacco products consumption and/or sales in the Federal Republic of Germany, Austria, Denmark (1978), Spain, Greece (including 1977-1979 prices), Ireland, Norway, Portugal, the United States (1980, Philip Morris, Inc.), and Zimbabwe (1980); and (3) tobacco export products from the Federal Republic of Germany and Brazil, and smuggling into Spain.

82–0898. Tabak Journal International. **Swedish Consumer Patterns Typical for Industrial Countries.** *Tabak Journal International* 6: 471, 474, December 1981.

The cigarette market in Sweden during 1980 was stagnant: tax increases and antismoking campaigns affected sales. The total consumption was 11,900 million cigarettes, which is the same volume as the previous year. Cigar and cigarillo consumption declined on the domestic market during 1980 to 155 million cigars/cigarillos, a 7 percent drop compared with 1979. The consumption of pipe and roll-your-own tobacco products rose by approximately 4 percent. The consumption of snuff in Sweden rose in 1980 by fully 3 percent to reach 3,665 tons. The consumption of chewing tobacco was unchanged. (Auth. Abs. Mod.)

82–0899. Tabak Journal International. **Statistics.** *Tabak Journal International* 6: 524-525, December 1981.

Production and import/export statistics for smoking and chewing tobacco and snuff are presented for selected countries worldwide. Production totals are listed yearly for 1976 through 1980, and import/export totals for 1978 through 1980. Changing patterns are reflected in the figures for some of the largest producers/importers of tobacco: (1) in the United States, where production dropped from 107.3 million kg in 1976 to 73.7 million kg in 1980, imports rose from 4 million kg in 1978 to 14 million in 1980, and exports rose from 2.5 million kg in 1978 to 8.4 million in 1980; (2) in the Netherlands, where production rose slightly from 23.7 million kg in 1976 to 25.3 million kg in 1980, exports rose from 3.4 to 8.3 million kg, and imports dropped from 14.8 to 2.2 million kg from 1978 to 1980; and (3) in the Federal Republic of Germany, production rose slightly in 1977 but fell again to a constant level at about 6.8 million kg.

82–0900. Tobacco Tax Council. **The Tax Burden on Tobacco. Historical Compilation.** Tobacco Tax Council, Inc., Volume 16, Richmond, Virginia, 1981, 185 pp.

Statistical information on past and present Federal, State, and local taxation of tobacco products, especially cigarettes, is presented. The three levels of government now exact revenues from cigarettes in the amount of over 6.6 billion dollars annually. Cigarette tax increases and the resulting increase in cigarette prices can affect cigarette purchases. A brief outline of highlights for the tax year ending June 30, 1981, is given, followed by tables listing Federal and State cigarette taxes from 1920 to 1981, the growth in cigarette excise taxes compared to the gross receipts of the growers, and per capita consumption of cigarettes from 1900 to 1981. Tax data for cigarettes and other tobacco products by Federal, State, and local governments, are included; each State and the District of Columbia listed separately, with information about the history and present status of their cigarette taxation. It is suggested that the tobacco information may be of use not only for the tobacco industry, but also for legislators, public administrators, and others.

82-0901. Weiss, E. A. **Australian Tobacco Manufacturers Tend Towards Diversification.** *Tabak Journal International* 6: 485-486, December 1981.

Consumption patterns for Australian smokers show a downward trend in cut-tobacco and cigar sales while cigarette consumption has remained almost constant for the last decade. Cigarette sales have been affected by: (1) the excise increase in the 1978 Australian budget; (2) increased unemployment which has reduced consumer spending; and (3) the growing influence of the antismoking lobby. As a result, the three main manufacturers (98.5 percent of the market)— Rothmans of Pall Mall, Philip Morris, and Amatil—have responded by introducing new and often cheaper brands; a wider choice of packs per brand, including the 25 cigarette pack; and introducing "mild" cigarettes as an option in all the most popular brands. Since advertising on the electronic media was phased out in 1974 through 1976, advertising has greatly increased in the print media, as has sponsorship of the arts and major sporting events. Because Australia's population is relatively static, the total market for cigarettes is likely to remain fairly constant in the short term. Therefore, the cigarette companies are diversifying into nontobacco areas, mainly package and fast foods, soft drinks, and wine.

LEGISLATION

82-0902. American Lung Association. **Legislative Addendum to Nonsmokers' Rights Program Guide.** New York, American Lung Association, 1980, 106 pp.

Data on State legislation and voluntary efforts to protect nonsmokers' rights, collected by questionnaire survey (copy included in appendix) on smoking and health activities, are presented. Of the 52 inquiries, 44 responses were received (85 percent). Information is included on what constitutes a basis for law, legislative strategies, examples of municipal/county/city legislation, samples of regulations and ordinances, petitions, fact sheets, and complaint forms. General guidelines for State legislation are discussed including bill sponsorship, write-in campaigns, press conferences, and the need for persistence. It is stressed that scientific facts be presented clearly and without exaggeration when conducting these activities. A section on smoking in the workplace discusses various surveys, a model Lung Association program, and an employee education program. Procedures for employees to follow in trying to establish smoke-free areas at work are outlined. Also included are a history of significant legislation concerning nonsmokers' rights and a detailed economic analysis of the effects of the Clean Indoor Air Act of 1978 on California governments.

82-0903. Bjartveit, K.; Lochsen, P. M.; Aaro, L. E. **Controlling the Epidemic: Legislation and Restrictive Measures.** *Canadian Journal of Public Health* 72(6): 406-412, November-December 1981.

Legislation and restrictions implemented by Norway to combat the smoking epidemic are discussed, noting that information, education, and treatment are also important measures. Restrictions must be generally acceptable to be successful, so public opinion should be surveyed, as was done in Norway, where results showed a positive attitude toward restrictions (probably reflecting information and education results, underscoring the importance of coordinating antismoking efforts). Provisions of the Norwegian legislative experience, which culminated in the 1975 Norwegian Tobacco Act, include: (1) a total ban on advertising of tobacco products, (2) a requirement that all packets carry health warnings, (3) a prohibition against selling or handing over tobacco products to persons under 16 years of age, and (4) permission for the Norwegian Ministry of Health and Social Affairs to issue regulations concerning the content, weight, filters, etc. of tobacco products. When compared with British data, program effectiveness is seen in Norwegian consumption figures which are far below the level for the United Kingdom: per capita consumption in Norway today corresponds with the figures the United Kingdom experienced more than 30 years ago; among males aged 60-69, the British lung cancer death rate is more than three times the Norwegian. The need for antismoking measures in developing countries (before consumption reaches the epidemic level) is also stressed.

82–0904. Klebe, E. R. **Actions of the Congress and the Federal Government on Smoking and Health.** Library of Congress, Congressional Research Service, Report No. 79-219 EPW, September 26, 1979, 61 pp.

A history of actions taken by the U.S. Congress and by the various Federal Government departments and regulatory agencies on the subject of smoking and health, for the period from the mid-1950s to the end of the 95th Congress in 1978, is presented. In response to the 1964 report of the Surgeon General's Advisory Committee, Congress has been the scene of numerous attempts, through legislation, to deal with the problem. Bills were passed requiring warning labels on cigarette packages, prohibiting cigarette advertising on radio and television, and providing funds to help deter childhood smoking. Efforts to curb smoking by a number of Federal regulatory agencies have augmented legislative action. The Department of Health, Education, and Welfare has assumed the role of gathering and disseminating information on the issue as well as initiating a broad program of public education, regulation, and research, to encourage people to stop or reduce smoking, to discourage youth from starting, and to protect the rights of nonsmokers for a smoke-free environment.

82–0905. Marks, L. **Policies and Postures in Smoking Control.** *British Medical Journal* 284(6313): 391-395, February 6, 1982.

The measures adopted for the control of smoking in the 15 nations of the Organization for Economic Cooperation and Development (OECD) are reviewed. All of the countries involved have antismoking policies, but these range from extensive and enforced legislation to abused voluntary agreements with the tobacco industry. The three most common themes in cigarette restriction are advertising bans in certain media, health warnings on cigarette packets, and restrictions on the content of allowed advertising. Fiscal measures are also widespread. Legislative actions range from Canada's Tobacco Restraint Act of 1908, which prohibits giving tobacco to those under 16 (generally accepted as unenforceable, and no later legislation exists), to Norway's tobacco act of 1973, which includes governmental control of tar, nicotine, and carbon dioxide levels. Voluntary codes demonstrate the same range, and present great difficulties in enforcement. Recently, various health organizations such as the Royal College of Physicians, disease associations such as the National Cancer Society, and numerous nonsmokers rights groups have taken up lobbying activities and other antismoking efforts. However, the unenforceable nature of much legislation makes "paper tigers" out of strong policies, and can even provide hidden benefits for the industry: the 1965 United Kingdom ban on television advertising resulted in prolific gift coupon schemes, and TV coverage of tobacco-sponsored sports events has led to more TV advertising than existed before the ban.

82–0906. Waterson, M. J. **Advertising and Cigarette Consumption.** London, England, The Advertising Association, 1981, 23 pp.

A cigarette advertising ban would deprive consumers of important product information. Countries that have imposed a ban on cigarette advertising have a much higher proportion of smokers who smoke nonfilter and high-tar brands than countries where product advertising is allowed. Advertising does not stimulate or maintain cigarette consumption levels. Research evidence shows that the influence of advertising expenditure on large mature consumer markets is negligible, other than at the brand level. In particular, detailed econometric studies of the United Kingdom cigarette market show that advertising expenditure has not had a significant influence on the total size of the cigarette market over the past 20 years. This evidence is reinforced by other studies of the effects of the cigarette advertising bans that are currently in force in a variety of countries. The most recent study concluded "every country in the world that has tried to reduce smoking by restricting tobacco advertising has been unsuccessful." Hence a cigarette advertising ban would be both futile and damaging to the interests of consumers, in addition to infringing seriously on the right of manufacturers to inform the public about legal products, and the right of consumers to full information about freely available products. (Auth. Abs.)

GENERAL

82–0907. British Medical Journal. **Tobacco Sponsorship of Sport: Think Again.** *British Medical Journal* 284(6313): 365, February 6, 1982.

The continued sponsorship of sports events in Britain by tobacco companies is criticized. Although cigarette advertising on television has been banned, the products are mentioned during commentary and appear on screen during sponsored sports events. There is a noticeable amount of such television coverage. Concealed advertising on television may be a source of concern. Although cigarette advertising in effect has apparently returned to television, no health warning accompanies it. Smoking becomes associated with healthy outdoor activity. Also, this link is presented to the attention of young people. It is suggested that other sponsors besides tobacco companies are available. An additional cigarette tax might raise enough money to cover current sports sponsorship by tobacco companies. The government should reconsider whether to allow sports sponsorship by tobacco companies.

82–0908. Campbell, P. **Nurses' Attitudes Toward Smoking and Non-Smoking Patients: A Semantic Differential Analysis.** Doctoral Dissertation, Teachers College, Columbia University, New York, University Microfilms International 81-22935, May 1981, 195 pp.

The dimensions of discrimination employed by practicing registered nurses (female, n = 333), in differentiating between smoking and nonsmoking female and male patients were evaluated for nurses from six New York City metropolitan area hospitals. Each nurse was provided with a brief description of a patient and asked to rate the patient on a 30-item semantic differential. Descriptions of patients were identical except for smoking behavior and gender; divided equally between a male smoker, a female smoker, a male nonsmoker, and a female nonsmoker. The nurses also completed a brief demographic questionnaire which provided data on their smoking behavior (nonsmoker, ex-smoker, light smoker, or moderate-to-heavy smoker). Four strong factors emerged when responses were subjected to a principal components analysis: (1) the bad patient, (2) the impotent patient, (3) the gentle patient, and (4) the dull and dirty patient. Scores on each factor were subjected to a three-way analysis of variance, with independent variables (1) patient's smoking behavior; (2) patient's gender; and (3) nurse's smoking behavior. Results showed: (1) smoking patients rated significantly higher than nonsmoking patients on the bad patient factor, regardless of gender of patient or smoking behavior of respondent; (2) female patients rated significantly higher than male patients on the impotent patient factor, regardless of smoking behavior of patient or respondent; (3) female patients rated significantly higher than male patients on the gentle patient factor by nonsmoking or light-smoking respondents, but not by respondents who were ex-smokers or who were moderate-to-heavy smokers; and (4) male patients rated significantly higher than female patients on the dull and dirty patient factor, regardless of smoking behavior of patient or respondent. The results are discussed in terms of person perception theory and with consideration of their implications for nursing practice, education, and research. (Auth. Abs. Mod.)

82–0909. Heneson, N. **Tobacco Versus Marijuana: What's the Score?** *New Scientist* 93(1295): 564, March 4, 1982.

In comparing the hazards of smoking cigarettes to those of marihuana, the National Academy of Sciences reports a similarity in the smoke of both, suggesting a strong possibility that prolonged marihuana use will lead to cancer of the respiratory tract and serious impairment of lung function. The Panel's report lists the hazards of marihuana as impairment of the brain's ability to control muscles, alteration of mood and perception, slowing of learning, interference with short-term memory, increasing the heart rate and sometimes blood pressure. However, these effects are reversible, and there is no evidence the drug induces physical dependence the way cigarettes do. Thus it is suggested that marihuana may be safer than cigarettes.

82–0910. Hornig, D. **Review Article: Metabolism and Requirements of Ascorbic Acid in Man.** *South African Medical Journal* 60(21): 818-823, November 21, 1981.

A study of ascorbic acid requirements appeared to indicate that smokers may need a higher ascorbic acid intake than nonsmokers. Study groups of smokers (consuming ≥ 20 cigarettes a day) and nonsmokers placed on diets low in vitamin C with varying levels of daily ascorbic acid supplements were measured for plasma and urinary metabolic results. For nonsmokers, it was calculated that a daily intake of 100 mg ascorbic acid was apparently needed to cover 95 percent of the male nonsmoking population. Higher total metabolic turnovers were needed in smokers to reach plasma ascorbate levels comparable to those of nonsmokers. It was calculated that a daily intake of 140 mg ascorbic acid was apparently needed to cover 95 percent of the male smoking population.

82–0911. Kozlowski, L. T.; Appel, C.-P.; Frecker, R. C.; Khouw, V. **Nicotine, a Prescribable Drug Available Without Prescription. (Letter).** *Lancet* 1(8267): 334, February 6, 1982.

The suggestion is made that it is irrational to treat nicotine gum as a prescribable drug while at the same time snuff and other tobacco products are treated as if the presence of nicotine and other health risks did not merit attention or regulation. The nicotine dose in both products is roughly the same, and is absorbed in the buccal mucosa, thus showing comparable pharmacokinetics. Canadian manufacturers caution against use of nicotine gum by pregnant or breast-feeding women and patients with certain heart conditions. Swedish snuff packages carry explicit warnings of nicotine as a nerve poison and other potential health injuries. Such concrete messages are better spurs to action than vague statements such as "snuff may be hazardous to your health." No warnings appear on Canadian, U.S., or United Kingdom snuffs, yet this is more hazardous than the controlled nicotine gum. Explicit and concrete health warnings should appear on all nicotine-containing products.

82–0912. Maugh, T. H., II. **Marijuana "Justifies Serious Concern."** *Science* 215(4539): 1488-1489, March 19, 1982.

The potentially harmful effects and some possible therapeutic uses of cannabis are discussed. Psychological and physiological effects are examined as well as the controversy involving paraquat contamination. The therapeutic potential for lowering intraocular pressure in glaucoma, the control of severe nausea and vomiting caused by carcinoma chemotherapy, and treatment of some central nervous system and emotional disorders as well as tumors are also considered. With the exception of the cannabinoids in one and tobacco alkaloids (nicotine) in the other, tobacco and marihuana smoke are qualitatively similar; marihuana smoke, however, contains about 50 percent more carcinogenic hydrocarbons. Preliminary research data suggest that exposure to combined marihuana and tobacco smoke produces more harmful effects than are produced by either alone. Greater investment in research on marihuana is recommended.

82–0913. National Research Council. **Indoor Pollutants.** Washington, D.C., National Academy Press, 1981, 552 pp.

Because air pollutants typically occur in concentrations and mixtures that are often episodic and vary over a wide range of time and from one space to another, human exposures are difficult to assess. There is little epidemiological evidence on the health effects of indoor pollutants. However, it is recognized that concentrations of some pollutants that already have primary ambient air quality standards exceed those standards. Although this report covers those pollutants that are liberated indoors, outdoor and indoor pollutants may interact chemically and physiologically. Pollutants such as radon, formaldehyde, asbestos and other fibers, tobacco smoke, indoor combustion (carbon monoxide), microorganisms and allergens, and moisture are presented but not ranked. As for tobacco smoke, it has been determined that 33 percent of the population smoke, and the rest are exposed to the smoke. An increased incidence of respiratory tract symptoms and functional decrements in children residing in homes with smokers as well as increased lung cancer rates among nonsmoking wives of smokers is now recognized. Coughing, headaches, nausea, and irritation of eyes, nose, and throat are among reported symptoms. Although many studies have measured various components of tobacco smoke indoors, total exposure has not been determined. Passive exposure may constitute an important source of respirable particles such as acrolein and formaldehyde, benzo(*a*)pyrene, and various trace metals. It is recommended that the Federal Government assess exposure in both residential and office buildings. Major health effects of contaminants should receive priority in the investigation but low-level acute and chronic complaints such as malaise and headache deserve careful study. Building factors should be investigated, including the effectiveness of air-cleaning systems in capturing pollutants. In the case of tobacco, smoking could be restricted to well-ventilated zones although public policy should clearly articulate that involuntary exposure ought to be minimized or avoided where possible.

82–0914. Robson, J. G.; Black, D.; Clark, D. H.; Curran, R. O.; Feroze, R. M.; Gillingham, F. J.; Horder, J.; Laws, J. W.; Strong, J. A.; Taylor, P. J. **Sponsorship of Sport by Tobacco Companies.** *British Medical Journal* 284(6313): 395-396, February 6, 1982.

The text of a letter signed by officials associated with several Royal Colleges and sent to the British minister for sport is presented, in which sports sponsorship by tobacco companies is criticized. The health hazards of smoking are mentioned. Tobacco company sponsorship of sports may tend to suggest an association in the minds of the young between smoking and healthy sports. It is argued that such sponsorship is a way of circumventing the ban on cigarette advertising on television and radio. Recommendations are given concerning any new government agreement on sports sponsorship by tobacco companies. Ideally such sponsorship should be banned. If this is not done, then the amounts the tobacco companies are permitted to spend on sponsorship should be progressively reduced, and any agreement should be limited to not more than 2 years, after which a new agreement should be made. The agreement should be monitored, and advertisements at and for events at which tobacco sponsorship is allowed to continue ought to carry a government health warning.

82–0915. Steinfeld, J. L. **Increasing Life Span; Changing Disease Patterns; Changing Life Styles: The Role of Government.** In: Harris, L. S. (Editor). National Institute on Drug Abuse Research Monograph 34, U.S. Department of Health and Human Services, Public Health Service, DHHS Publication No. (ADM)81-1058, February 1981, pp. 11-19.

Cigarette smoking which has been associated with cardiovascular diseases, cancer, and pulmonary disease, is a major health hazard and should receive continued attention. About 53 million Americans are regular smokers. In recent years the proportion of filtered cigarettes has increased while tar and nicotine levels have fallen. About 30 million Americans have stopped smoking cigarettes, and at present, the overall level of smoking among adults is 32.3 percent, the lowest level in 45 years. Cigarettes are not considered a food or drug and are not subject to regulation by a government department, except for regulation of advertising by the Federal Trade Commission. It is suggested that government should educate people about the hazards of tobacco and conduct research on disease processes caused by cigarette smoking. Behavioral research may also be needed.

AUTHOR AND ORGANIZATIONAL INDEX

Aaro, L. E., 82-0903
Abdallah, F., 82-0881
Aceto, M. D., 82-0646
Achkar, E., 82-0768
Adams, J. D., 82-0675
Adelstein, A. M., 82-0658
Agaj, A., 82-0693
Ahmed, M. B., 82-0821
Aittoniemi, K., 82-0703
Alderson, M. R. 82-0659
Aliperta, A., 82-0617
Allen, R. F., 82-0853
Allen, R. J., 82-0618
American Lung Association., 82-0902
Anderson, M. W., 82-0660
Aoki, K., 82-0684
Appel, C.-P., 82-0911
Arany-Fuzessery, K., 82-0610
Arber, F., 82-0718
Aroney, C., 82-0709
Ashley, M. J., 82-0648
Asire, A. J., 82-0692
Atsana, S., 82-0805
Auerbach, O., 82-0719
Axell, T., 82-0769
Ayre, D. J., 82-0676

Bailey, A., 82-0738
Balavadze, M. B., 82-0742
Baldwin, N., 82-0867
Balfour, D.J.K., 82-0619
Ball, K., 82-0751
Balmes, J. R., 82-0681
Baraniewski, K., 82-0732, 82-0733
Barbee, R. A., 82-0773
Barrett-Connor, E., 82-0722, 82-0730
Bartlett, E. E., 82-0822
Baskerville, J. C., 82-0840
Bastiaan, R. J., 82-0770
Baugh, J. G., 82-0795
Baumgartner, K., 82-0823
†Baumgartner Papier S.A., 82-0868
Bazin, M., 82-0720
Beaumont, J. J., 82-0661
Beck, G. J., 82-0694
Becker, M., 82-0698
Beeley, L., 82-0755, 82-0756
Belitskaya, R. A., 82-0863
Bellissimo, U., 82-0617
Ben-Sira, Z., 82-0779
Benarde, M. A., 82-0662
Benfari, R. C., 82-0806
Bennett, D. H., 82-0745
Benwell, M.E.M., 82-0619
Berenson, G. S., 82-0752, 82-0795
Best, J. A., 82-0824
Beton, D. C., 82-0745
Bevan, C., 82-0710
Biener, K., 82-0780
Bigelow, G. E., 82-0793
Biro, E., 82-0732, 82-0733
Bjartveit, K., 82-0903
Black, D., 82-0914
Bock, F. G., 82-0771
Boggio, V., 82-0725
Bolanowska, W., 82-0640
Bonebrake, F. C., 82-0726
Bonini, S., 82-0772
Bonnet, J., 82-0868
Booyse, F. M., 82-0721
Boreham, J., 82-0738
Boroujerdi, M., 82-0660
Bosse, R., 82-0715
Boutet, M., 82-0720
Bratush, C. M., 82-0641
Braud, M. L., 82-0725
Bray, C. L., 82-0745
British Medical Journal., 82-0907
Brittebo, E. B., 82-0663
Brockway, M., 82-0727
Brook, J. S., 82-0781
Broom, G., 82-0621
Brower, R. W., 82-0735
Brown, R. A., 82-0830
Bryant, L. R., 82-0746
Bryson, R., 82-0782
Buring, J. E., 82-0684
Burrows, B., 82-0773

Campbell, M. J., 82-0710
Campbell, P., 82-0908
Canada Health Survey., 82-0783
Canadian Tobacco Grower., 82-0885
Cardillo, M. E., 82-0712
Carlisi, J., 82-0796
Carrasco, J. C., 82-0854
Carrol, S., 82-0785
Carter, H. W., 82-0719
Cartwright, R. A., 82-0672
Caton, J. E., 82-0666
Cazamajour, F., 82-0607
†Celanese Corp., 82-0883
Celebic, A., 82-0711
Chakraborty, A. K., 82-0810
Chameaud, J., 82-0664
Chang, R. L., 82-0640
Chaudhary, B. S., 82-0753
Chen, C. B., 82-0624
Chianca, M. C., 82-0617
Chouteau, J., 82-0607
Chretien, J., 82-0664
Clark, D. H., 82-0914
Clarke, E. A., 82-0665
Clarke, P.B.S., 82-0620
Clayton, B., 82-0642
Cluette, J. E., 82-0728
Conte, D., 82-0889
Cockle, S. M., 82-0760
Coetzee, A. M., 82-0784
Coggins, C.R.E., 82-0695
Coleman, D., 82-0786
Comstock, G. W., 82-0747
Conney, A. H., 82-0640
Cooper, R., 82-0683
Corrao, W. M., 82-0699
Cotes, J. E., 82-0710
Criqui, M. H., 82-0722, 82-0727, 82-0730
Cumming, G., 82-0697
Cummings, A. J., 82-0760
Cunningham, D. G., 82-0757
Curran, R. O., 82-0914

Dalbey, W. E., 82-0666
Dale, R. W., 82-0882
Danaher, B. G., 82-0838
Daniel, M. L., 82-0723
Daniele, R. P., 82-0712
Darioli, R., 82-0718
Dass, T., 82-0711
Dauber, J. H., 82-0712
Dawley, H. H., Jr., 82-0785
Dean, A., 82-0621
Deanfield, J., 82-0724

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Deibel, P., 82–0758
Delahunt, J. P., 82–0855
DeLong, E. R., 82–0673
Deluca, P. P., 82–0623
Delucia, A. J., 82–0746
Delves, H. T., 82–0642
Delzell, E. S., 82–0673
Di Franza, J. R., 82–0691
Dicken, C., 82–0782
DiClemente, C. C., 82–0856
Dodsworth, H., 82–0621
Doll, R., 82–0649, 82–0667, 82–0668
Downing, R. W., 82–0622
Druzin, M. L., 82–0765
Dumas, J. P., 82–0725
Dunn, J., 82–0808

Eberhagen, D., 82–0625
Einolf, W. N., 82–0612
Emmerson, B., 82–0709
Enoch, H., 82–0623
Epstein, F. H., 82–0748
Epstein, L. H., 82–0786
Erickson, A. D., 82–0699
Essinger, A., 82–0718

Falletta, J. M., 82–0673
Faragher, E. B., 82–0745
Farber, E., 82–0669
Farmline., 82–0886
Farquhar, J. W., 82–0825
Fee, W. M., 82–0857
Feinleib, M., 82–0748
Ferguson, R. N., 82–0612
Feroze, R. M., 82–0914
Ferrone, L., 82–0828
Finau, S. A., 82–0787
Folts, J. D., 82–0726
Fox, K. M., 82–0724
Frank, C. W., 82–0753
Frecker, R. C., 82–0648, 82–0911
Friedman, G. D., 82–0788
Fujiwara, M., 82–0628

Gabriel, A., 82–0690
†Gallaher Ltd., 82–0882
Garfinkel, L., 82–0670
Garn, S. M., 82–0757
Garrison, R. J., 82–0748
Garrity, F. L., 82–0699
Gayle, T. M., 82–0700
German, G. A., 82–0808
Gessner, T., 82–0640
Gibel, W., 82–0671
Gielen, J. E., 82–0645
Gillingham, F. J., 82–0914
Glashan, R. W., 82–0672
Goerisch, I., 82–0696
Goerisch, V., 82–0696
Goldberg, J. D., 82–0753
Goldberg, S. R., 82–0790
Goldman, H., 82–0759
Gonzalez, C. A., 82–0791
Gordon, A. S., 82–0781
Gordon, J. R., 82–0864
Graefe, A., 82–0869
Graham, I. M., 82–0841
Granda, E. J., 82–0874
Green, D. E., 82–0826
Greenberg, J. A., 82–0827
Greenway, C., 82–0796
Greitemeyer, M., 82–0789
Griesemer, R., 82–0666
Griffith, E. E., 82–0858
Griffiths, R. R., 82–0792, 82–0793
Grove, R. B., 82–0777
Grubbs, H. J., 82–0871
Grufferman, S., 82–0673
Gudzinowicz, B. J., 82–0611
Gudzinowicz, M. J., 82–0611
Guerin, M. R., 82–0666
Guilland, J. C., 82–0725
Guyatt, A. R., 82–0697

Haac, L. A., 82–0865, 82–0866
Halonen, M., 82–0773
Hammer, R. E., 82–0759
Hannan, M. A., 82–0623
Harada, N., 82–0775
Harris, L. S., 82–0646
Haskell, W., 82–0727
Havel, R. J., 82–0743
Haxhiu, M. A., 82–0693
Hay, D. R., 82–0794
Hecht, S. S., 82–0624
Heinemann, G., 82–0625
Heiss, G., 82–0722, 82–0727
Heneson, N., 82–0909
Henning, L., 82–0774
Henningfield, J. E., 82–0792, 82–0793
Henricsson, V., 82–0769
Heyden, S., 82–0694
Hickey, N., 82–0841
Higashi, E., 82–0636
Higgins, I., 82–0698
Higgins, M. W., 82–0698
Hill, R. W., 82–0859
Hilton, T. F., 82–0821
Hinks, L., 82–0642
Hirayama, T., 82–0674
Hiroshi, M., 82–0870
Hocko, M., 82–0626
Hoffmann, D., 82–0616, 82–0675
Hofman, A., 82–0751
Hojnacki, J. L., 82–0728
Holdbrook, M. J., 82–0730
Holford, T. R., 82–0729
Holmes, M. A., 82–0697
Honda, K., 82–0677
Horder, J., 82–0914
Hornig, D., 82–0910
Houminer, Y., 82–0871
Howard, C. A., 82–0713
Howatt, W., 82–0698
Howland, K. E., 82–0887
Howley, T. J., 82–0860
Huber, G. L., 82–0728
Hubert, H. B., 82–0729
Hughes, J., 82–0786
Hulley, S. B., 82–0788
Hund, F., 82–0633
Hunter, S. M., 82–0753, 82–0795
Hurwitz, I., 82–0806
Hymowitz, N., 82–0861

Idle, M., 82–0738
Igarashi, S., 82–0627
Ikushima, S., 82–0628
Imai, H., 82–0677
†International Flavors and Fragrances Inc., 82–0874, 82–0877, 82–0878, 82–0879, 82–0880
Irwin, R. S., 82–0699
Ishiguro, S., 82–0615
Itani, S., 82–0636

Jackson, N., 82–0796
Jacobs, D. R., 82–0633
Jacobson, B., 82–0797
Jaeger, R. J., 82–0629
Jamasbi, R., 82–0685
Janus, J., 82–0630
Janzon, L., 82–0631
†Japan Tobacco and Salt Public Corp., 82–0870
Jarvis, G. K., 82–0836
Jason, L. A., 82–0828
Jenkins, R. A., 82–0700
Johnson, C. A., 82–0633
Johnston, M., 82–0757
Johnston, N., 82–0850
Jokiel, M., 82–0798
Jonathan, A., 82–0724

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Jones, B. R., 82–0730
Jones, N. L., 82–0701
Juan, H., 82–0632

Kado, M., 82–0702
Kaelin, A. C., 82–0760
Kaemmerlen, J. T., 82–0699
Kallianos, A. G., 82–0872
Kalliomaki, K., 82–0703
Kalliomaki, P.-L., 82–0703
Kaminska-Kosmider, A., 82–0630
Kane, J. P., 82–0743
Kannel, W. B., 82–0729, 82–0731, 82–0748
Kappenberger, L., 82–0734
Kaprio, J., 82–0801
Karstedt, B. D., 82–0883
Katz, T., 82–0612
Keast, D., 82–0676
Keith, C. H., 82–0883
Keller, J. B., 82–0698
Kessler, I. I., 82–0680
Kew, R. R., 82–0728
Key, C. R., 82–0713
Khouw, V., 82–0911
Kim, J. O., 82–0613
Kimm, S.Y.S., 82–0673
King, M.-C., 82–0788
Kinser, R. D., 82–0612
Kirk, C.J.C., 82–0829
Kirkinen, P., 82–0761
Kiwala, J., 82–0873, 82–0877, 82–0878, 82–0879
Klatsky, A. L., 82–0682, 82–0788
Klebba, A. J., 82–0650
Klebe, E. R., 82–0904
Kleinschmidt, E.-G., 82–0774
Klepping, J., 82–0725
Knapp, R. J., 82–0704
Kobara, Y., 82–0677
Koerner, M., 82–0888
Koman, S. L., 82–0862
Koskela, K., 82–0831
Koskenvuo, M., 82–0801
Kozak, J., 82–0705
Kozlowski, L. T., 82–0799, 82–0911
Kringlen, E., 82–0751
Krishna, K. P., 82–0800
Krol, J., 82–0733
Kuch, J., 82–0732, 82–0733
Kumar, R., 82–0620
Kunze, M., 82–0689
Kunzler, A., 82–0734
Kyokane, K., 82–0638

LaFuma, J., 82–0664
Lagace, R., 82–0720
Land, G., 82–0651
Landau, L. I., 82–0707
Landis, J. R., 82–0698
Langinvainio, H., 82–0801
Larme, P., 82–0631
Lasser, N. L., 82–0861
Lavrova, I. G., 82–0802
Laws, J. W., 82–0914
Leaman, D. M., 82–0735
Lebet, J.-P., 82–0868
Lebowitz, M. D., 82–0773
Leck, I., 82–0684
LeCompte, W. A., 82–0803
Lee, J. K., 82–0614
Lefcoe, N. M., 82–0840, 82–0867
Lehnert, G., 82–0678
Lewit, E. M., 82–0889
Liard, R., 82–0706
Lichtenstein, E., 82–0830
Liddell, F.D.K., 82–0679
†Liggett Group Inc., 82–0872
Lin, D., 82–0624
Lin, R. S., 82–0680
Lindell, S.-E., 82–0631
Lochsen, P. M., 82–0903
Low, J. A., 82–0762
Lowrey, A. H., 82–0771
Lu, C.-T., 82–0736
Lubiszewska, B., 82–0732, 82–0733
Luepker, R. V., 82–0633
Lund, V. J., 82–0829

Maccoby, N., 82–0825
MacLennan, R., 82–0805
Maeda, K., 82–0775
Magnus, P. F., 82–0825
Malaya, E. V., 82–0802
Manning, F. A., 82–0765
Markelova, V. F., 82–0863
Marks, L., 82–0905
Marlatt, G. A., 82–0864
Marmot, M. G., 82–0737, 82–0851
Marshall, J. P., 82–0894
Martin, A. J., 82–0707
Martin, B. R., 82–0646
Marty, P. J., 82–0832
Masse, R., 82–0664
Matsue, S., 82–0870
Matsui, Y., 82–0677
Matsushima, S., 82–0615
Matthay, R. A., 82–0681
Maugh, T. H., II., 82–0912
Maunder, L. R., 82–0694

Maxwell, J. C., Jr., 82–0890, 82–0891, 82–0892
May, E. L., 82–0646
Mazzone, J. S., 82–0893
McAlister, A., 82–0831
McDermott, R. J., 82–0832
McEvoy, L., 82–0651
McNamara, P. M., 82–0748
McNicol, M. W., 82–0738
Meester, G. T., 82–0735
Menzie, E. L., 82–0894
Merchant, J. A., 82–0708
Mermolja, M., 82–0693
Mettlin, C., 82–0833
Miller, G. H., 82–0682
Miller, R. H., 82–0895
Miller, W. J., Jr., 82–0683
Milsum, J. H., 82–0834
Mintz, J., 82–0873
Mir, M. A., 82–0749
Mirand, E. A., 82–0833
Mishkel, M., 82–0722
Mishra, S. K., 82–0634
Mitchell, C., 82–0709
Mitchell, J. A., 82–0759
Moar, V., 82–0764
Mollica, M., 82–0828
Momiyama, K., 82–0639
Monk, M. A., 82–0753
Monti, M., 82–0718
Mookherjee, B. D., 82–0874, 82–0879
Morgan, R. W., 82–0665
Morison, J. B., 82–0804
Morrison, A. S., 82–0684
Morrison, J., 82–0785
Moser, I. S., 82–0884
Mougne, C., 82–0805
Mowery, R., 82–0727
Mulcahy, R., 82–0841
Mulligan, J. J., 82–0728
Muramatsu, I., 82–0628
Muranaka, H., 82–0635, 82–0636
Murata, K., 82–0739
Murata, Y., 82–0765
Murray, D. M., 82–0633
Musk, A. W., 82–0710
Mussinan, C. J., 82–0874
Mustard, J. F., 82–0740
Myers, J. R., 82–0699

Naeye, R. L., 82–0763
National Academy of Sciences., 82–0637
National Interagency Council on Smoking and Health., 82–0835

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

National Research Council., 82-0913
Nelson, J. F., 82-0896
Nettesheim, P., 82-0666, 82-0685
Newman, A. M., 82-0665
Niederhauser, H. U., 82-0734
Nishi, K., 82-0638
Nishioka, H., 82-0638
Norioka, T., 82-0639
Northcott, H. C., 82-0836
Nunez-Cortes, J. M., 82-0688
Nuttall, R. L., 82-0806

Obata, K., 82-0684
Ockene, I. S., 82-0806
Ockene, J. K., 82-0806
Ohno, Y., 82-0684
Oldridge, N. B., 82-0741
Oleynikov, S. P., 82-0742
Oosone, A., 82-0639
Orleans, C. T., 82-0865, 82-0866
Orlov, Yu. M., 82-0802
Oshima, S., 82-0677
Osikowicz, G., 82-0721
Ossip, D. J., 82-0786
Ounsted, M., 82-0764

Padron, J. S., 82-0837
Paigen, B., 82-0640
Papadimitriou, J. M., 82-0676
Park, E. S., 82-0613
Park, J. Y., 82-0875
Park, K. H., 82-0613, 82-0647
Patarra, S. F., 82-0876
Pathak, D. R., 82-0713
Patrianakos, C., 82-0616
Pechacek, T. F., 82-0633, 82-0838
Pederson, L. L., 82-0839, 82-0840, 82-0867
Percy, C. L., 82-0692
Perdrizet, S., 82-0706
Perna, A., 82-0617
Perraud, R., 82-0664
Peto, R., 82-0668
Petzold, A. S., 82-0757
Phelan, P. D., 82-0707
†Philip Morris, Inc., 82-0871
Phillips, N. R., 82-0743
Pitner, T. P., 82-0612
Pocock, S. J., 82-0642
Podeur, G., 82-0607
Poe, R. H., 82-0711
Pollack, B., 82-0827
Pomerleau, O. F., 82-0807
Poole, A. D., 82-0808
Popovac, D., 82-0693
Pratt, P. C., 82-0694, 82-0708
Pratter, M. R., 82-0699
Preet, K., 82-0817
Prior, I.A.M., 82-0787
Pryszmont, M., 82-0744
Public Health Reports., 82-0809
Puska, P., 82-0831

Radek, J., 82-0721
Rahkonen, E., 82-0703
Rahlfs, V., 82-0625
Ramsdale, D. R., 82-0745
Ransom, C. C., Jr., 82-0830
Raviprakash, V., 82-0634
Raymond, T. L., 82-0746
Reade, P. C., 82-0770
Recio, L., 82-0623
Reinert, P., 82-0706
Repace, J. L., 82-0771, 82-0776
Revue des Tabacs., 82-0897
Rickels, K., 82-0622
Ridella, S., 82-0757
Robson, J. G., 82-0914
Ronan, G., 82-0841
Rooney, J. J., 82-0882
Roos, M., 82-0761
Rose, G., 82-0737
Rosner, B., 82-0715
Rossman, M. D., 82-0712
Rothmeier, R. C., 82-0842
Rotman, H., 82-0698
Roy, M., 82-0810
Ruane, P., 82-0841
Ruberman, W., 82-0753
Rudnev, S. G., 82-0863

Saceda, P.G.-B., 82-0688
Safer, M. A., 82-0811
Safirstein, B. H., 82-0861
Saito, Y., 82-0639
Salonen, J. T., 82-0831
Samet, J. M., 82-0713
Samkoff, J. S., 82-0747
Sanson-Fisher, R. W., 82-0808
Sardar, Z., 82-0812
Sato, A., 82-0677
Schachter, E. N., 82-0694
Schachter, S., 82-0813, 82-0843
Schievelbein, H., 82-0625
Schmitt, F. L., 82-0874, 82-0877, 82-0878, 82-0879
Schrag, S. D., 82-0713
Schreiber, W. L., 82-0879
Sciandra, R., 82-0833
Scott, A., 82-0764
Seffrin, J. R., 82-0777
Selikoff, I. J., 82-0686
Selwyn, A., 82-0724
Senior, S. L., 82-0814
Serwecinska, T., 82-0732, 82-0733
Shamberger, R. J., 82-0641
Shaper, A. G., 82-0642
Shiffman, S., 82-0815
Shipley, M. J., 82-0737
Shipley, R. H., 82-0865, 82-0866
Shishova, A. M., 82-0742
Shryock, H., 82-0816
Sicurella, J., 82-0850
Silverberg, E., 82-0652
Simecek, C., 82-0714
Singh, S., 82-0817
Sklov, M. C., 82-0752, 82-0795
Sneader, W., 82-0844
Snowden, C. B., 82-0748
Socol, M. L., 82-0765
Sottiurai, V., 82-0736
Southwick, E. W., 82-0872
Sparrow, D., 82-0715
Spealman, R. D., 82-0790
Sprecker, M. A., 82-0877, 82-0878
Sprince, H., 82-0643
Srinivasan, S. R., 82-0752
St. John, D.J.B., 82-0778
St. Leger, A. S., 82-0716, 82-0717
Stack, D. J., 82-0728
Stanaway, R. G., 82-0818
Stanhope, J. M., 82-0787
Steenland, K., 82-0640
Steffens, G. L., 82-0608
Steinbrunn, W., 82-0734
Steinfeld, J. L., 82-0915
Stewart, M. J., 82-0857
Stork, G., 82-0879
Strong, J. A., 82-0914
Sugawara, S., 82-0615
Superintendent of Public Instruction., 82-0845

Tabak Journal International., 82-0898, 82-0899
Tagliacozzo, R., 82-0819
Takamatsu, M., 82-0775
Tamada, T., 82-0635, 82-0636
Tanner, N.S.B., 82-0690
Tarkkonen, L., 82-0801
Taylor, D. H., 82-0846
Taylor, P. J., 82-0914
Thomas, B. J., 82-0737
Timbrell, D., 82-0847
Tirlapur, V. G., 82-0749

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Tjalve, H., 82-0663
Tobacco Tax Council., 82-0900
Topping, D. C., 82-0685
Topping, D. L., 82-0644
Treffert, D. A., 82-0653
Trell, E., 82-0631
Tripathi, H. L., 82-0646
Troitino, R. P., 82-0688
Tuomilehto, J., 82-0831
Turcotte, H., 82-0720
Turner, J.A.McM., 82-0738
Tweed, W. A., 82-0750
Tyroler, H. A., 82-0727

U.S. Department of Health and Human Services., 82-0654, 82-0655, 82-0687, 82-0766
U.S. Department of Labor, 82-0687
University of Kentucky., 82-0609

Vaaranen, V., 82-0703
Van Allen, M. I., 82-0767
van Cantfort, J., 82-0644
Vandenbroucke, J. P., 82-0751
Van Vunakis, H., 82-0738
Varela, A. S., 82-0688
Verhoek, W. G., 82-0684
Vinals, J. F., 82-0874, 82-0877, 82-0878, 82-0879
Vincek, W. C., 82-0646
Vock, M. H., 82-0874, 82-0877, 82-0878, 82-0879
Vollmer, R. T., 82-0708
Voors, A. W., 82-0752
Vutuc, Ch., 82-0689

Wadden, R. A., 82-0618
Wald, N. J., 82-0738
Wale, C. J., 82-0642
Walker, G. R., Jr., 82-0820
Walker, M., 82-0642
Wallace, R. B., 82-0722
Walsh, D., 82-0833
Wang, H. H., 82-0673
Ward-Booth, R. P., 82-0690
Ward, C., 82-0745
Ward, E., 82-0640
Waterson, M. J., 82-0906
Watson, D. W., 82-0818
Webber, L. S., 82-0752, 82-0795
Weg, J. G., 82-0698
Weilepp, M., 82-0696
Weinblatt, E., 82-0753
Weis, W. L., 82-0848, 82-0849
Weiss, E. A., 82-0901
Weiss, N. S., 82-0661
Weiss, S. T., 82-0715
Weiss, W., 82-0662
Wennmalm, A., 82-0754
Whitman, M., 82-0781
Wiist, W., 82-0786
Williams, C., 82-0865, 82-0866
Wilson, A.G.E., 82-0660
Wilson, D., 82-0850
Wilson, R. A., 82-0879
Wingard, D. L., 82-0656
Winkelstein, W., Jr., 82-0851
Winters, T. H., 82-0691
Wo, Y.I.S., 82-0647
Wojtulewicz, L., 82-0733
Wood, A. W., 82-0640
Wood, G., 82-0850
Woolcock, N. E., 82-0829
Wynn, R. A., 82-0852

Yang, C.-F., 82-0736
Yang, K. K., 82-0875
Yarnell, J.W.G., 82-0716, 82-0717
Yoshida, T., 82-0880
Young, G. P., 82-0778
Young, J. L., Jr., 82-0692
Yu, K. K., 82-0875

Zalesnick, M., 82-0796
Zamorano, T., 82-0657
Zarins, C. K., 82-0736
Zyryaeva, L. A., 82-0742

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

SUBJECT INDEX

ABORTION
- maternal smoking effect, 82–0758
- spontaneous, maternal smoking relationship, 82–0755

ABSENTEEISM
- cost benefit through no smoking policy, 82–0849

ACCIDENTS
- workplace cost benefit through no smoking policy, 82–0849

ACETALDEHYDE
- possible toxicity from smoking, 82–0643

ACETYLCHOLINE
- nicotine comparison on release and metabolism of arachidonic acid, 82–0632

ACROLEIN
- respirable particle of tobacco smoke, 82–0913

ADENOCARCINOMA
- etiology, possible synergism of environmental factors and smoking, 82–0681
- lifetime cigarette smoke-exposed rats, occurrence, 82–0666
- passive smoking as factor in nonsmoking wives of smokers, 82–0674, 82–0678
- smoking habit factor in occurrence of varying cell types of lung neoplasms, 82–0677

ADOLESCENTS
- alcohol, tobacco, and drug use in India, 82–0817
- antismoking program for ninth grade students, 82–0828
- ethnic patterns in age of drug use initiation, 82–0796
- initiation of cigarette smoking, social factors, 82–0795
- personality and maternal determinants in son's tobacco use, 82–0781
- reduction of smoking via increased excise tax, 82–0889
- saliva thiocyanate as chemical measure of smoking, 82–0633
- school health education programs, smoking cessation effectiveness, 82–0822
- serum lipid and lipoprotein levels, smoking effects, 82–0752
- smoking awareness curriculum impact, 82–0840
- smoking habits in Switzerland, 82–0780
- smoking habits in the United States, 1978-1979, 82–0826
- Washington State smoking and health teaching guide, 82–0845

ADVERTISING
- advertising ban, cigarette consumption relationship, 82–0906
- annual expenditures for the top 20 cigarette brands, 82–0896
- Australian tobacco companies, 82–0901
- cigarette ads aimed at women, 82–0797
- cigarettes, prohibition on radio and television, 82–0904
- Norway, bans on tobacco products, 82–0903
- smoking control measures in 15 countries, review, 82–0905
- sponsorship of sports events by tobacco companies in Britain, 82–0907, 82–0914

AFRICA
- tobacco consumption increase, possible growth area for U.S. companies, 82–0886

AGE FACTORS
- alcohol consumption and smoking influence on blood lead concentrations in middle-aged men, 82–0642
- allergic reactivity, with smoking, 82–0773
- asbestos and smoking dosage effects on interval from exposure to death from lung cancer, 82–0679
- coronary risk factor in middle-aged men, 82–0751
- occupational cosmetic exposure, allergy history, total serum IgE, and smoking correlations, 82–0772
- response to mortality risk factors, health hazard appraisal questionnaire, 82–0811
- statistical variable, HDL cholesterol study, 82–0727
- 21-year-old asthmatics, smoking incidence, 82–0707

AGRICULTURAL PRACTICES
- early topping effects on tobacco leaf nicotine and tar content, 82–0607
- maleic hydrazide residues, reduction by combination with a *N*-benzyldinitroaniline, 82–0608
- tobacco handbook, University of Kentucky, 82–0609

AIR POLLUTION
- carbon monoxide, indoor hospital levels, 82–0618
- health impairment effects, 82–0913
- lung function impairment in children, effect on, 82–0717
- natural gas combustion and tobacco smoke, combined effects, 82–0629
- Nonsmokers' Rights Program, American Lung Association, 82–0902
- occupational hazard with smoking, 82–0835
- passive smoking health hazards and possible solutions, 82–0776

ALCOHOL CONSUMPTION
adults in Canada, smoking relationship, 82–0783
blood lead concentrations effect, smoking relationship, 82–0642
cardiovascular vs. noncardiovascular mortality according to daily intake, smoking relationship, 82–0737
coronary heart disease risk reduction, smoking and dietary factors, 82–0730
correlation with hypertension, smoking effect, 82–0722
monozygotic vs. dizygotic twins, smoking and personality factors, 82–0801
mortality in abstainers vs. moderate drinkers, 82–0682
pancreatic cancer risk, 82–0680
smoking correlation, 82–0811
twins, smoking relationship, 82–0788

ALDEHYDES
possible acetaldehyde toxicity from smoking, 82–0643

ALKALOIDS, TOBACCO
combustibility of tobacco, positive correlation, 82–0610

ALLERGY
indoor pollutant relationship, 82–0913
skin-test reactivity, total serum IgE, blood eosinophilia, and smoking interrelationships, 82–0773
total serum IgE, occupational cosmetic exposure and smoking correlations, 82–0772

AMINO ACIDS
nicotine administration in liver, reduced leucine incorporation, 82–0627

ANGINA PECTORIS
prognosis for smokers vs. nonsmokers vs. ex-smokers, 82–0729
treatment, smoking effects, 82–0723

ANTISMOKING CAMPAIGNS
Canada, governmental and media influence on smoking cessation, 82–0836
Canada, health education programs, 82–0847
Chile, proposed health education, 82–0657
fear-moderating vs. fear-neutral health information effects, 82–0779
North Karelia Project, 82–0831
Norway, legislation and restrictive measures, 82–0903
physicians' role, 82–0823
Scottish pharmacies, 82–0844
smoking control measures in 15 countries, review, 82–0905
state and local legislative recommendations, American Lung Association, 82–0902
suggested intervention methods, 82–0852
tobacco use and oral health education, 82–0777
United States, programs, 82–0825
United States, effect on tobacco consumption and the tobacco industry, 82–0886
Wisconsin Athletes Against Cancer program, 82–0832

ANTISMOKING MATERIALS
cancer prevention education program for elementary schools, 82–0833
distribution in Scottish pharmacies, 82–0844
manuals for therapists and subjects, 82–0855
two self-help manuals, evaluation of effectiveness, 82–0867
Washington State elementary and secondary schools, teaching guide, 82–0845
Wisconsin Athletes Against Cancer program, 82–0832

ANXIETY
college students, smoking habit relationship, 82–0800
ecological study of stress and rate of self-stimulation in Turkey with smoking as a suppressor, 82–0803
latent fear arousal of antismoking and cancer screening campaigns, 82–0779
smoking relationship, urine pH and nicotine withdrawal factor, 82–0813

AORTA
endothelial aortic bovine cells, nicotine effect, 82–0721
endothelium permeability in rats, cigarette smoke effect, 82–0720

ARECA CHEWING
betel nut consumption in Ban Pong village, Thailand, 82–0805
oral cancer incidence in Sri Lanka, relationship, 82–0690

ARIZONA
allergy skin-test reactivity, total serum IgE, eosinophilia, and smoking interrelationships, 82–0773

AROMATIC AMINES
analysis in cigarette smoke, 82–0616

AROMATIC HYDROCARBON CONTENT
marihuana vs. tobacco smoke, 82–0912

AROMATIC HYDROCARBONS
activation in tracheobronchial epithelial cells, 82–0669
tobacco and marihuana analysis via gas chromatography and mass spectrometry, 82–0611

ARRHYTHMIA
occurrence during myocardial infarct rehabilitation, smoking relationship, 82–0734

ARTERIAL DISEASES
cessation of smoking among coronary bypass surgery patients, 82–0735
cessation of smoking counseling and reinforcement in patients, 82–0829

HDL-cholesterol and smoking as risk factors, 82–0718
percutaneous transluminal angioplasty benefits in smokers vs. nonsmokers, 82–0736
prognostic index prior to valve replacement, smoking role, 82–0745

ARTERIES
atherogenesis of coronary vessels in smokers vs. nonsmokers, 82–0719

ARTERIOSCLEROSIS
mechanisms involved, smoking effect, 82–0740
presence in coronary arteries, smokers vs. nonsmokers, 82–0719
smoking relationship, conference proceedings, 82–0648
smoking relationship, literature review, 82–0739

ARYL HYDROCARBON HYDROXYLASE
antipyrene metabolism, seasonal variation, 82–0640
fluorimetric and radiometric assays for human placentas, 82–0760
lung induction with cigarette smoking in rat, 82–0645

ASBESTOS
Australian waterside workers, exposure effects, 82–0709
exposure-death interval, dosage and smoking effects, 82–0679
exposure effects, smoking as multiplicative factor, 82–0687
indoor pollutant, 82–0913
lung cancer risks and mortality rates, smoking relationship, 82–0686

ASIA
tobacco consumption increase, possible growth area for U.S. companies, 82–0886

ASTHMA
smoking incidence in 21-year-old asthmatics, 82–0707

ATTITUDES
adolescent smoking relationship, 82–0826
cessation of smoking factor, 82–0850
cessation of smoking methods, topical bibliography, 82–0865, 82–0866
cessation of smoking, psychosocial factors, 82–0806
cessation of smoking to improve employee productivity, 82–0848
college students in the Soviet Union, smoking habit relationship, 82–0802
Diffusion of Innovation Theory, college smoking cessation intervention program, 82–0827
education level vs. smoking habits, 82–0809
ex-smokers, relapse factors, 82–0815
hospital patients and staff, smoking habits in hospital, 82–0814
Japanese smokers' intentions to quit and general feelings on nonsmokers' rights, 82–0885
nurses' perceptions of smoking behavior of patients, 82–0908
positively vs. negatively worded "no smoking" signs, effect on smoking behavior, 82–0785
public and private self-awareness effect on smoking habit, 82–0820
public disbelief in association of cancer and smoking in Poland, 82–0798
public responses to smoking hazards, 82–0915
risk-taking and health practices in German smoking vs. nonsmoking students, 82–0789
smokers' reluctance to receive health risk information, 82–0819
smokers' response to counselor smoking status in cessation program, 82–0830
smoking awareness curriculum effect, 82–0840
social factors in initiation of cigarette smoking, 82–0795
teachers, cancer education, 82–0833
televised smoking cessation program, subject variables, 82–0824
twins, factors in smoking discordance, 82–0788

AUSTRALIA
respiratory morbidity in Brisbane waterside workers, 82–0709
tobacco industry diversification, 82–0901

AUTONOMIC NERVOUS SYSTEM
acute tobacco intoxication effects, 82–0725

AVERSIVE THERAPY
combination with self-control for effectiveness, 82–0838

BACTERIA
tracheobronchial clearance and/or detoxification, smoking effect, 82–0699

BEHAVIOR, ANIMAL
learning, nicotine effect on self-stimulation, 82–0620
nicotine administration effect on maintenance and suppression of squirrel monkey responses, 82–0790

BEHAVIOR, HUMAN
analysis of abstainers and nonabstainers following rapid-smoking treatment, 82–0808
Canada Health Survey information, 82–0783
cessation of smoking, psychosocial factors, 82–0806
cessation of smoking to improve employee productivity, 82–0848
cessation of smoking, topical bibliography, 82–0865, 82–0866
college students in the Soviet Union, factors related to smoking habit, 82–0802
combined techniques in smoking cessation, 82–0854

education level vs. smoking habits, 82–0809
ex-smokers, relapse factors, 82–0815
Health Hazard Appraisal system for evaluating and modifying health lifestyles, 82–0834
hospital patients and staff, smoking incidence, 82–0814
maintenance of smoking abstinence, coping with relapse factors, 82–0864
male vs. female mortality, smoking and behavioral factors, 82–0656
modification of smoking habit, resistance, 82–0838
passive smoking hazard, possible solutions, 82–0776
patients, nurses' perceptions of relative to sex and smoking behavior, 82–0908
personality traits of smokers vs. ex-smokers, 82–0791
positively vs. negatively worded "no smoking" signs, effect on smokers, 82–0785
psychobiological models of the smoking habit, 82–0807
psychological and physiological factors in smoking, 82–0816
psychological effect of intervention, 82–0751
public and private self-awareness effect on smoking habit, 82–0820
public disbelief in association of cancer and smoking in Poland, 82–0798
risk-taking and health practice attitudes in German smoking vs. nonsmoking students, 82–0789
self-designed smoking cessation program, 82–0853
self-efficacy relationship to maintenance of smoking cessation, 82–0856
situational stress study in Turkey, smoking as suppressor variable, 82–0803
smokers' reluctance to receive health risk information, 82–0819
smoking rates among scientists, publishing relationship, 82–0782
social factors in initiation of cigarette smoking, 82–0795
televised smoking cessation program, subject variables, 82–0824
twins, smoking and alcohol use, personality factors, 82–0801

BENZO(*A*)PYRENE
indoor pollutant, 82–0913
inhibition of carcinogenesis with butylated hydroxyanisole, 82–0660
metabolism, measure of induced aryl hydrocarbon hydroxylase activity, 82–0640
mutagenicity inactivation by human saliva, 82–0638

BIOASSAY
aryl hydrocarbon hydroxylase in human placenta, 82–0760
nicotine and cotinine extraction method for gas chromatography, 82–0635
plasma nicotine microextraction method, 82–0636
tobacco smoke inhalation dosimetry model, 82–0700
vasopressin content of guinea pig hypothalamus and neurohypophysis, smoking effects, 82–0630

BIRTH DEFECTS
maternal smoking effect, 82–0758
vascular disruption, smoking relationship, 82–0767

BIRTH WEIGHT
maternal smoking effect, 82–0755, 82–0757, 82–0758, 82–0764, 82–0767
Nevada birth survey, maternal smoking effects, 82–0766

BLADDER NEOPLASMS
coffee consumption and smoking as risk factors, 82–0684
occupation and smoking as risk factors, 82–0672
smoking-associated mortality, United States, 1960-77, 82–0650

BLOOD
cell count and volume, smoking and oral contraceptive effects, 82–0621
nicotine treatment in stressed rats, plasma corticosterone, 82–0619

BLOOD CHEMICAL ANALYSIS
nicotine treatment in stressed rats, plasma corticosterone, 82–0619
plasma lipoprotein levels in stumptailed macaques after smoke exposure, 82–0746

BLOOD CHOLESTEROL
atherogenic risk factor, smoking relationship, 82–0718
exercise and smoking effect, 82–0723
nicotine effect in rats, 82–0639
plasma lipoprotein levels in stumptailed macaques after smoke exposure, 82–0746
socioeconomic status and lifestyle relationship, 82–0727

BLOOD CIRCULATION
coronary flow and thrombosis in dogs, nicotine effects, 82–0726
pulse, vascular tonus, smoking effects, 82–0744
reduced oviductal and uterine in rats, nicotine effect, 82–0759

BLOOD COAGULATION
vascular prostaglandins vs. platelet thromboxane formation, nicotine effects, 82–0754

BLOOD LIPIDS
nicotine effect in rats, 82–0639
plasma lipoprotein levels in stumptailed macaques after smoke exposure, 82–0746

- serum and lipoprotein levels relationship, smoking association, 82–0743
- socioeconomic status and lifestyle relationship, 82–0727

BLOOD PLATELETS
- aggregation, nicotine effect, 82–0632
- thrombosis and coronary flow in dogs, nicotine effects, 82–0726
- vascular prostaglandins vs. thromboxane formation, nicotine action, 82–0754

BLOOD PRESSURE
- acute tobacco intoxication effects, 82–0725
- exercise and smoking effect, 82–0723
- nicotine effect in rats, 82–0639

BLOOD SUGAR
- exercise and smoking effect, 82–0723

BLOOD VESSELS
- atherogenesis of coronary arteries, smokers vs. nonsmokers, 82–0719
- cessation of smoking among coronary artery bypass surgery patients, 82–0735
- injury to vessel wall, smoking effect, 82–0740

BODY WEIGHT
- nicotine effect in rats, 82–0639

BRAIN
- blood vessels and circulation, smoking effects, 82–0744
- determination of 1-methylhistamine binding sites in rat brain tissue, 82–0704
- nicotine binding and disposition in rats, 82–0646
- nicotine treatment in stressed rats, 5-hydroxyindole levels, 82–0619
- self-stimulation, nicotine effect, 82–0620

BRAZIL
- cigarette sales decrease, 82–0893

BRONCHI
- bacterial clearance and/or detoxification, smoking effect, 82–0699

BRONCHIAL DISEASES
- indoor pollutants relationship, 82–0913
- obstructive airway illness, smoking as a risk factor, 82–0698

BRONCHIAL EPITHELIUM
- radiation exposure from alpha emitters in cigarette smoke, 82–0691

BRONCHIAL MUCOSA
- cigarette smoke, properties and influence on bronchial membranes, 82–0705
- smoking alterations in mucogenesis, 82–0617

BRONCHIAL NEOPLASMS
- incidence in East Germany, smoking relationship, 82–0671
- smoking-associated mortality, United States, 1960-77, 82–0650

BRONCHITIS
- bacteria clearance and/or detoxification, smoking effect, 82–0699
- cotton textile workers, 82–0708
- disease and smoking rates among Hispanics vs. Anglos in New Mexico, 82–0713
- passive smoking as factor in nonsmoking wives of smokers, 82–0674
- passive smoking relationship in Czechoslovakia, 82–0714
- simple vs. wheezy in children, parental smoking relationship, 82–0706
- smoking-associated mortality, United States, 1960-77, 82–0650

CADMIUM
- kidney concentration with zinc and selenium in smoking and nonsmoking deceased patients, 82–0641

CAFFEINE
- pancreatic cancer risk, 82–0680

CANADA
- cessation of smoking, randomized trial in Hamilton, Ontario, 82–0850
- governmental and media antismoking campaigns, influence on smoking cessation, 82–0836
- health education programs, 82–0847
- Ontario youth smoking habits, predictors, 82–0840
- public health survey information, 82–0783
- smoking habit, health consequences, conference proceedings, 82–0648
- tobacco production, pricing and marketing system, 82–0894
- Toronto study, carcinoma of the cervix, 82–0665
- Winnipeg students' smoking habit 1960-1980, 82–0804

CANNABIS DERIVATIVES
- pharmacology, absorption, metabolism, and excretion, 82–0611

CARBOHYDRATES
- tobacco smoke and its constituents, effect on metabolism, 82–0644

CARBON DIOXIDE
- catalyzed production from carbon monoxide, 82–0882

CARBON MONOXIDE
- indoor pollutant, 82–0913
- inhalation levels with nicotine and cholesterol, effect on plasma lipoprotein composition, 82–0728

- less hazardous cigarettes, risk reduction, Surgeon General's report, 82–0655
- lipid and carbohydrate metabolism, possible effects, 82–0644
- maternal blood, effect on fetus, 82–0767
- natural gas combustion and tobacco smoke, effects, 82–0629
- sheesha smokers vs. cigarette smokers, 82–0812
- site-specific absorption in respiratory tract, 82–0697

CARBON MONOXIDE CONTENT
- catalyzed oxidation to carbon dioxide, 82–0882
- indoor hospital levels, 82–0618

CARBOXYHEMOGLOBIN LEVELS
- blood concentration relationship to smoking habit, 82–0631
- fetal hypoxia relationship in rhesus monkey, 82–0765
- fetus of smoking mothers, 82–0629
- lipid and carbohydrate metabolism, possible effects, 82–0644
- maternal bloodstream, nicotine effect, 82–0762
- primary vs. secondary pipe smokers, 82–0738

CARCINOGENESIS
- inhibition of DNA adducts in the lung with butylated hydroxyanisole, 82–0660
- nitrosamines, formation and metabolism, 82–0614
- nitrosonornicotine in the rat, 82–0663
- respiratory tract, host and environmental factors, 82–0685
- smoking-related cancers, "state of the art" review, 82–0669

CARCINOGENS
- alpha emitters in cigarette smoke, 82–0691
- marihuana vs. tobacco smoke, and combined effect, 82–0912
- mutagenicity inactivation by human saliva, 82–0638
- *N*-nitrosamines, levels in snuff and saliva of snuff dippers, 82–0675
- tobacco and other factors in cancer etiology, 82–0668
- tobacco smoke, combustion temperature correlation, 82–0610

CARCINOMA, BRONCHOGENIC
- host and environmental factors, 82–0685

CARDIOVASCULAR DISEASES
- alcohol abstainers vs. moderate drinkers, smoking effect, 82–0682
- aortic endothelium and myocardial capillary permeability in rats, cigarette smoke effects, 82–0720
- declining mortality, decreased proportion of smokers, 82–0731
- ischemic heart disease primary prevention programs, 82–0851
- occurrence in moderate vs. heavy vs. nondrinkers, smoking relationship, 82–0737
- risk reduction via less hazardous cigarettes, Surgeon General's report, 82–0655
- smoking-associated mortality, United States, 1960-77, 82–0650

CARDIOVASCULAR SYSTEM
- acute tobacco intoxication effects, 82–0725
- prostaglandin and nicotine interaction, 82–0754

CATECHOLAMINES
- acute tobacco intoxication effects, 82–0725
- excretion in smokers vs. nonsmokers after acupuncture treatment, 82–0863
- excretion of epinephrine, norepinephrine, and metabolites, smoking effect, 82–0625
- nicotine effect, 82–0767
- nicotine effects in placental blood flow and carboxyhemoglobin levels via catecholamine stimulation, 82–0762
- nicotine effects on coronary blood flow and thrombosis in dogs, 82–0726

CELLS, EPITHELIAL
- endothelial aortic bovine cells, nicotine effect, 82–0721
- endothelium, reduced protection with nicotine, 82–0632
- population and morphology of respiratory epithelium, smoking effects, 82–0693

CENTRAL NERVOUS SYSTEM
- benzodiazepine use in anxiety treatment, coffee consumption and smoking effects, 82–0622
- nicotinic stimulation effect on responses of fowl rectum, 82–0634

CESSATION OF SMOKING
- abstinence maintenance, coping behavior, 82–0864
- analysis of abstainers and nonabstainers following rapid-smoking treatment, 82–0808
- angina pectoris patients, prognosis effect, 82–0729
- antenatal program for pregnant women, 82–0846
- arterial occlusion, post surgical benefits, 82–0736
- atherosclerosis risk reduction, 82–0740
- cardiovascular mortality risk reduction, 82–0731
- cognitive-behavioral model, 82–0838
- combined behavior modification techniques, 82–0854
- commitment-enhancement interviews effect, 82–0842
- coronary disease risk reduction, 82–0750
- counselor smoking status impact on program effectiveness, 82–0830
- Diffusion of Innovation Theory in college students, 82–0827
- employee health and productivity benefits, 82–0848
- family physician counselor role, 82–0821
- gastric ulcer healing rate, effect, 82–0768

government health campaigns and media influence in Canada, 82–0836
Health Hazard Appraisal system, effectiveness, 82–0834
higher self-cure rates compared to therapy, 82–0843
ischemic heart disease risk reduction, 82–0851
Japanese smokers' attitudes, 82–0885
long-term maintenance, measured by self-efficacy construct, 82–0856
low rates among coronary artery bypass surgery patients, 82–0735
lung neoplasms risk effect, 82–0681
ninth grade students, experimenting vs. regular smokers, 82–0828
North Karelia Project, 82–0831
occupational program guide, 82–0835
personality traits as success factor, 82–0791
physicians' role, 82–0823
psychobiological models of smoking behavior, implications for quitting, 82–0807
reliability of self-reported smoking history, rapport factor, 82–0841
school health education programs, effectiveness evaluation, 82–0822
smoking control program goal, recidivism effects, 82–0862
success prediction through individual psychosocial factors, 82–0806
ulcer healing effect, 82–0778
women's smoking habits and quitting method, 82–0797

CESSATION OF SMOKING, METHODS
  acupuncture, catecholamine excretion following treatment, 82–0863
  aerobic exercises and self-management, 82–0860
  aversion and self-control, 82–0838
  biofeedback training, 82–0858
  counseling and reinforcement for arterial patients, 82–0829
  counseling in a family practice setting, 82–0850
  family physician, individualized stop-smoking strategy, 82–0821
  graduated external filters, 82–0861
  hypnosis treatment for groups, 82–0859
  nicotine chewing gum, effectiveness, 82–0857
  physicians, counseling various patient groups, 82–0839
  scaled article to help control consumption, 82–0884
  self-cure vs. therapy, 82–0843
  self-help module for self-designed quitting program, 82–0853
  self-regulated cessation program, 82–0816
  television series and self-help guide, 82–0824
  therapist effect assessment, 82–0855
  topical bibliography, 82–0865, 82–0866
  two self-help manuals, evaluation of effectiveness, 82–0867

CHEWING TOBACCO
  factors favorable to development of smokeless products, 82–0896
  statistical data from selected countries, production and import/exports, 82–0899
  Sweden, consumption in 1980, 82–0898

CHILDREN
  health education about cancer prevention in elementary schools, 82–0833
  initiation of cigarette smoking, social factors, 82–0795
  lung function impairment caused by smoking in pregnancy and/or respiratory infections, 82–0717
  prevention of smoking program, Wisconsin Athletes Against Cancer, 82–0832
  respiratory morbidity, household smoking as factor, 82–0716
  respiratory tract impairment with indoor pollutants, 82–0913
  rhabdomyosarcoma, relative risk from paternal smoking, 82–0673
  smoking awareness curriculum impact, 82–0840
  Washington State smoking and health teaching guide, 82–0845

CHILE
  proposed antismoking campaign, 82–0657

CHROMIUM
  urinary levels in smoking vs. nonsmoking stainless steel welders, 82–0703

CIGAR SMOKING
  nicotine, cotinine, and carboxyhemoglobin levels, 82–0738

CIGARETTE SMOKE
  alpha emitters, carcinogenic relationship, 82–0691
  aromatic amines analysis, 82–0616
  cartridges for filter and flavor selection, 82–0876
  flavone and butylatedhydroxyanisole vs. condensate microsomal activation, 82–0647
  health risks, marihuana comparison, 82–0909
  inhalation bioassay model, 82–0700
  isolation and identification of two C_{20} seco-cembranoids, 82–0612
  nicotine delivery, manipulation experiment, 82–0793
  physical and chemical properties, influence on bronchial membranes, 82–0705
  respiratory tract neoplasms in rats exposed daily over a lifetime, 82–0666
  whole vs. gas phase, effect on lung compliance and residual volume in rats, 82–0695

CIGARETTES
  ethnic patterns in age of initiation of smoking, 82–0796
  Sweden, consumption in 1980, 82–0898

SUBJECT INDEX

CIGARETTES, HIGH-TAR
- cessation of smoking factor, 82–0850
- Kreyberg I and II tumors vs. controls, associations, 82–0689
- pathological changes in exposed mice, 82–0676

CIGARETTES, LOW YIELD
- smoke inhalation levels and cholesterol intake effect on plasma lipoprotein composition, 82–0728
- Surgeon General's report on the changing cigarette, 82–0655

CIGARETTES, LOW-NICOTINE
- Brazil, increase in popularity, 82–0893
- compensatory smoking behavior, 82–0786

CIGARETTES, LOW-TAR
- Brazil, increase in popularity, 82–0893
- cessation of smoking factor, 82–0850
- exposure equivalency of passive smokers to high-tar sidestream smoke, 82–0771
- flavor enhancement, 82–0869
- market growth since 1970, 82–0896
- 1981 market share, sales trends, 82–0892

CIGARS
- 1980 Maxwell Report statistics, 82–0891
- Sweden, consumption in 1980, 82–0898

COCARCINOGENS
- 2-aminoanthracene and cigarette smoke, 82–0623
- radon daughter products and tobacco smoke, combined effects in rats, 82–0664
- respiratory tract, host and environmental factors, 82–0685

COFFEE CONSUMPTION
- anxiety treatment with benzodiazepines vs. placebo, effect with smoking, 82–0622
- bladder cancer association, smoking effects, 82–0684
- pancreatic cancer etiology, smoking effect comparison, 82–0662

COGNITION
- dissonance in smokers toward health risk information, 82–0819

COMBUSTION TEMPERATURE
- carcinogenic substances formed in cigarettes, correlation, 82–0610

CONTRACEPTIVES, ORAL
- blood cell counts and volume, smoking effects, 82–0621
- serum lipids and lipoprotein levels in black vs. white adolescent girls, smoking relationship, 82–0752

CORONARY DISEASE
- alcohol consumption, smoking, and diet as risk factors, 82–0730
- angina pectoris, prognosis for smokers vs. nonsmokers vs. ex-smokers, 82–0729
- causal relationship of cigarette smoking, 82–0750
- cessation of smoking among artery bypass surgery patients, 82–0735
- diet and smoking as risk factors, Oslo study, 82–0751
- exercise program to minimize risks, smoking effect, 82–0723
- incidence among siblings, possible risk factors, 82–0748
- ischemic heart disease primary prevention programs, 82–0851
- ischemic heart disease, smoking and other risk factors in Moscow population, 82–0742
- ischemic heart disease, smoking as risk factor, 82–0732, 82–0733
- monozygote twins, smokers vs. nonsmokers, 82–0788
- Mormons vs. non-Mormons, prevalence, 82–0651
- myocardial infarct, prognosis comparison between 1960s and 1970s patients, 82–0753
- nicotine effects on coronary blood flow and thrombosis in dogs, 82–0726
- nicotine relationship, 82–0738
- prevention counseling recommended in New Zealand, 82–0794
- prognostic index prior to valve replacement, smoking role, 82–0745
- rehabilitative program participation, smoking relationship, 82–0741
- smoking-associated mortality, United States, 1960-77, 82–0650

COST BENEFITS ANALYSIS
- company savings with no smoking policy, 82–0849

COTININE
- pipe smokers' levels, 82–0738
- plasma concentration, smokers vs. nonsmokers, extraction method for gas chromatography, 82–0635

COUNSELING
- arterial patients, effect on cessation of smoking, 82–0829
- cessation of smoking strategies in family practice medicine, 82–0850
- family physicians, antismoking advice as preventive health care, 82–0821
- nurses, smoking effect in pregnancy, 82–0758
- physicians, effectiveness strategies, 82–0839
- smoking prevention in high risk candidates in New Zealand, 82–0794

SUBJECT INDEX

CZECHOSLOVAKIA
passive smoking at home vs. chronic bronchitis prevalence, 82–0714

DEMOGRAPHIC FACTORS
cancer mortality rates among black men, 82–0683
college students, smoking habit relationship, 82–0800
cultural determinants in form of tobacco use, 82–0799
ethnic patterns in age of initial drug experimentation, 82–0796
morbidity and mortality in developed countries vs. developing countries, smoking effects, 82–0649
Mormons in Missouri and Utah, morbidity comparison, 82–0651
mouth neoplasms, Sri Lanka vs. United Kingdom, 82–0690
U.S. cancer statistics, 82–0692
urbanization effect on smoking habit, Tonga Islands, 82–0787

DIABETES
taste threshold in smoking vs. nonsmoking, healthy vs. diabetic patients, 82–0774

DIET
alcohol consumption in coronary heart disease risk, smoking relationship, 82–0730
ascorbic acid requirements in smokers, 82–0910
coffee drinking vs. bladder cancer, smoking effects, 82–0684
coronary heart disease, risk reduction, 82–0751
food vs. tobacco, health effects of nitrosamine, 82–0637
maternal smoking, relationship to pregnancy outcome, 82–0758
smoke inhalation and cholesterol intake effects on plasma lipoprotein composition, 82–0728
twins, smoking relationship, 82–0788

DNA
carcinogen interaction, 82–0669
inhibition of benzo(*a*)pyrene diolepoxides: DNA adducts in the lung with butylated hydroxyanisole, 82–0660

DRUG ABUSE
ethnic patterns in age of initiation, 82–0796

DRUG METABOLISM
benzodiazepine use in anxiety treatment, coffee consumption and smoking effects, 82–0622
carbenoxolone and antacids, smoking effect, 82–0778

DUST
cotton worker exposure, bronchial lesions association, 82–0708
cotton worker exposure, byssinosis relationship, 82–0694

DYSPNEA
Australian waterside workers, 82–0709

ECONOMICS
Australian tobacco industry diversification, 82–0901
benefits of occupational smoking control programs, 82–0835
Brazil, cigarette sales decrease, 82–0893
business losses as antismoking incentive, 82–0852
cigarette sales up for 1981, 82–0892
company savings with no smoking policy, 82–0849
continued growth in the cigarette industry, 82–0890
cost of employing smokers, 82–0848
costs to individual and society of smoking, 82–0816
Federal, State, and local tobacco tax data, 82–0900
high 1981 production, expected trends for 1982, 82–0895
Japan, tobacco production, sales, imports, consumption, 82–0885
potential of excise tax to reduce cigarette consumption, 82–0889
regulation, programs, data of tobacco industry in United States and Canada, 82–0894
shifting consumption patterns worldwide, effects on U.S. tobacco industry, 82–0886
statistical data, tobacco production and import/exports worldwide, 82–0899
tobacco industry status report, 82–0896
tobacco products, world manufacturing, consumption, and sales, 1978-1980, 82–0897
tobacco programs, 82–0609
United States, cigar imports and sales, 82–0891
Uruguay, smoking control and health care costs, 82–0837
West Germany, rising costs and taxes, effects on cigarette consumption, 82–0888
world tobacco industry, expected trends for 1982, 82–0887

ELECTROCARDIOGRAPHY
hypoxemia effects in chronic obstructive airways disease patients, 82–0749

ELECTROENCEPHALOGRAPHY
biofeedback training for reduction or cessation of smoking, 82–0858

EMBRYO
reduced conceptus cell number in rats, nicotine effect, 82–0759

EMPHYSEMA
cigarette particulates, effect on lungs and chronic obstructive diseases, 82–0701
Hispanics vs. Anglos in New Mexico, disease and smoking rates, 82–0713
incidence among smoke-exposed mice, 82–0676

smoking-associated mortality, United States, 1960-77, 82-0650

ENZYME ACTIVITY
aryl hydrocarbon hydroxylase in human placenta, 82-0760
aryl hydrocarbon hydroxylase inducibility in human lymphocytes, antipyrene metabolism, 82-0640
blood and bronchoalveolar lymphocytes in smokers and nonsmokers with pulmonary sarcoidosis, 82-0712
cyclo-oxygenase, inhibition with nicotine, 82-0754
lactic acid dehydrogenase enhancement and inhibition, 82-0647
lung lysozyme concentration in rats after cigarette smoke inhalation, 82-0702
ontogenic variation in rat liver, lung, and kidney with low doses of benzo(*a*)pyrene and cigarette smoke condensate, 82-0645

ENZYMES
aryl hydrocarbon hydroxylase, 82-0640, 82-0760
cyclo-oxygenase, 82-0754
cyclo-oxygenase inhibition, nicotine effect, 82-0632
lactic acid dehydrogenase, 82-0647
lung lysozyme in rats, 82-0702
serum angiotensin-converting enzyme, 82-0712

ESOPHAGEAL NEOPLASMS
smoking-associated mortality, United States, 1960-77, 82-0650

ESOPHAGUS
nitrosonornicotine-induced tumors in the rat, 82-0663

ETHNIC GROUPS
age of initiation of drug experimentation, 82-0796
black men, cancer mortality rates, 82-0683
Hispanics in New Mexico, lower respiratory disease and smoking rates, 82-0713
serum lipid, lipoprotein levels in adolescents, smoking and race factors, 82-0752

EX-SMOKERS
adults in Canada, 82-0783
analysis of abstainers and nonabstainers following rapid-smoking treatment, 82-0808
angina pectoris patients, prognosis compared with smokers, 82-0729
carcinoma of the cervix, reduced risk, 82-0665
ischemic heart disease risk factors compared with smokers and nonsmokers, 82-0742
personality factors compared with smokers and nonsmokers, 82-0818
personality traits compared to smokers, 82-0791
relapse factors, 82-0815
reliability of self-reported smoking history, rapport factor, 82-0841

FEAR
moderating and neutral health information effects, 82-0779

FEDERAL REPUBLIC OF GERMANY
college students, risk-taking and health practice attitudes in smokers vs. nonsmokers, 82-0789
tobacco tax increase, expected effects on consumption, 82-0888

FETAL GROWTH
hypertension and smoking risk factors, 82-0761
maternal smoking effect, 82-0762, 82-0764

FETAL MORTALITY
infections vs. maternal smoking, 82-0763
maternal smoking effect, 82-0758

FETUS
aryl hydrocarbon hydroxylase, low activity in rat, 82-0645
carboxyhemoglobin level, maternal smoking effect, 82-0629
hypoxia, catecholamine release and placental blood flow, nicotine role, 82-0762
hypoxia in rhesus monkey, cigarette smoke exposure effect, 82-0765
vasoconstriction and anoxia resulting in birth defects, 82-0767

FILTERS
catalysts for oxidizing carbon monoxide to carbon dioxide, 82-0882
cessation of smoking, external use, 82-0861
glycol monacetin toxicity in animals, 82-0626
increase in smoking in Japan, relationship, 82-0885
menthol addition, 82-0875
smoke-modifying cartridges, 82-0876

FINLAND
North Karelia Project for health promotion, 82-0831

FUNGI
Candida albicans oral prevalence, smoking relationship, 82-0770

GAS PHASE, CIGARETTE SMOKE
chemical composition analysis via gas chromatography and mass spectrometry, 82-0611

GERMAN DEMOCRATIC REPUBLIC
bronchial and lung neoplasms, smoking relationship, 82-0671

GLYCOLS
glycol monoacetin toxicity in animals, 82-0626

SUBJECT INDEX

GYNECOLOGIC NEOPLASMS
carcinoma of the cervix, smoking relationship, 82–0665

HEALTH EDUCATION
antismoking programs in Canada, 82–0847
antismoking campaign proposed in Chile, 82–0657
cancer prevention for elementary schools, 82–0833
Health Hazard Appraisal system for evaluating and modifying health lifestyles, 82–0834
knowledge of smoking effects on health in 21-year-old asthmatics, 82–0707
Lifegain System, self-help module for smoking cessation, 82–0853
North Karelia Project, 82–0831
physicians' role, 82–0823
prevention of smoking, Wisconsin Athletes Against Cancer program, 82–0832
smoking awareness curriculum effect on youth's smoking habit, 82–0840
smoking cessation effectiveness evaluation, 82–0822
smoking control components, value, 82–0825
tobacco use and oral health information programs, 82–0777
Washington State elementary and secondary schools, smoking and health teaching unit, 82–0845

HEALTH PRACTICES
Canada Health Survey information, 82–0783
health hazard questionnaire, smoking responses, 82–0811
risk-taking attitudes, and perception of dangers of smoking in German college students, 82–0789

HEART
myocardial capillaries permeability in rats, cigarette smoke effect, 82–0720

HEART DISEASES
angina pectoris, prognosis for smokers vs. nonsmokers vs. ex-smokers, 82–0729
incidence among siblings, possible risk factors, 82–0748
ischemic, smoking as risk factor, 82–0732, 82–0733

HEART FUNCTION
electrocardiographic effects of hypoxemia in chronic obstructive airway disease patients, 82–0749

HEART RATE
acute tobacco intoxication effects, 82–0725

HEMATOCRIT
blood cell count and volume, smoking and oral contraceptive effects, 82–0621

HEMODYNAMICS
acute tobacco intoxication effects, 82–0725
cessation of smoking benefits, 82–0736
myocardial oxygen requirement assessment, smoking effect, 82–0724

HEMOGLOBINS
blood cell counts and volume, smoking and oral contraceptive effects, 82–0621

HEREDITY
predisposition to coronary heart disease among siblings, 82–0748

HETEROCYCLIC COMPOUNDS
tobacco flavorant additive, 82–0871

HORMONES
vasopressin content of guinea pig hypothalamus and neurohypophysis, smoking effects, 82–0630

HYPERTENSION
alcohol vs. smoking effect, 82–0722
birth weight effect, maternal smoking relationship, 82–0764
diagnosis, possible interference by patient smoking, 82–0625
risk in pregnancy, smoking effects, 82–0761
smoking relationship, 82–0742

HYPNOSIS
cessation of smoking treatment, 82–0859

HYPOXEMIA
electrocardiographic effects in chronic obstructive airway disease patients, 82–0749

HYPOXIA
nicotine and carbon monoxide effect on fetus, 82–0767
rhesus monkey fetus, cigarette smoke exposure effect, 82–0765

IMMUNE SYSTEM
reactions associated with eosinophilia, smoking relationship, 82–0773

IMMUNOGLOBULINS
total serum IgE relative to occupational cosmetic exposure, age, allergy history, and smoking effects, 82–0772
total serum IgE, skin-test reactivity, eosinophilia, and smoking interrelationships, 82–0773

INDIA
Patiala, Punjab, drug abuse survey of students, 82–0817
West Bengal, smoking among medical students, 82–0810

INFANT MORTALITY
maternal smoking effect, 82–0755, 82–0762

SUBJECT INDEX

INFANTS
Apgar scores, maternal smoking effects, 82–0757
respiratory symptoms and parental smoking relationship, 82–0706

INFORMATION SERVICES
topical bibliography, smoking cessation methods, 82–0865, 82–0866

INTELLECTUAL DEVELOPMENT
retardation in infants of smoking mothers, 82–0757, 82–0767

ISCHEMIA
electrocardiographic determination, smoking effect, 82–0724

ISRAEL
Jewish urban adults, latent fear arousal in antismoking and cancer screening campaigns, 82–0779

JAPAN
tobacco production, sales, consumption patterns, 82–0885

KIDNEY DISEASES
cadmium, selenium, and zinc levels in cancer, hypertensive, and renal patients, 82–0641

KIDNEY NEOPLASMS
smoking-associated mortality, United States, 1960-77, 82–0650

KIDNEYS
enzyme induction with benzo(*a*)pyrene and cigarette smoke condensate in rat, 82–0645

LABELING
health warnings on cigarette packages, 82–0904
Norway health warning requirement, 82–0903
smoking control measures in 15 countries, review, 82–0905
warnings recommended for nicotine-containing products, 82–0911

LACTATION
nicotine and other drugs, possible effects on neonate, 82–0756

LARYNGEAL NEOPLASMS
incidence in smoking and nonsmoking patients, 82–0696
smoking-associated mortality, United States, 1960-77, 82–0650

LARYNX
benign disease vs. malignant disease, smoking relationship, 82–0696

LEAD
alcohol consumption and smoking influence on blood concentrations, 82–0642

LEARNING
nicotine effect on self-stimulation in rats, 82–0620

LEGISLATION
advertising ban, cigarette consumption relationship, 82–0906
Federal, State, and local tobacco tax data, 82–0900
Nonsmokers' Rights Program guide, American Lung Association, 82–0902
Norwegian Tobacco Act, 82–0903
possible smoking control in Canada, conference proceedings, 82–0648
smoking control measures in 15 countries, review, 82–0905
tobacco tax increase effects on consumption in West Germany, 82–0888
United States, smoking and health, 82–0904

LIP NEOPLASMS
smoking-associated mortality, United States, 1960-77, 82–0650

LIPIDS
serum levels, smoking effects in adolescents, 82–0752
tobacco smoke and its constituents, effect on metabolism, 82–0644

LIPOPROTEINS
cholesterol intake and smoke inhalation effects, 82–0728
exercise and smoking, 82–0723
serum levels, smoking effects in adolescents, 82–0752
triglyceride and cholesterol levels relationship, smoking association, 82–0743

LIVER
enzyme induction with benzo(*a*)pyrene and cigarette smoke condensate in rat, 82–0645
nicotine administration, reduced leucine incorporation, 82–0627

LIVER CIRRHOSIS
smoking-associated mortality, United States, 1960-77, 82–0650

LUNG DISEASES
parenchymal, Australian waterside workers, smoking factor, 82–0709
sarcoidosis, enzyme activity correlation with blood and bronchoalveolar lymphocytes, 82–0712

LUNG FUNCTION
bacterial clearance and/or detoxification, smoking effect, 82–0699

breathing mechanics and ventilatory functions, smoking effects, 82–0705
children, household smoking as factor in respiratory morbidity in Wales vs. western England, 82–0716
children, smoking in pregnancy and respiratory infections factors, 82–0717
cigarettes vs. marihuana, health risks, 82–0909
cotton dust exposure, 82–0708
cotton dust exposure vs. smoking, 82–0694
firefighters vs. nonfirefighters, 82–0715
graduated external filter use during smoking, effect, 82–0861
respiratory function tests vs. smoking history in predicting postsurgical pulmonary complications, 82–0711
simple pneumoconiosis, smoking effect, 82–0710
smoke inhalation experiment effects in rats, 82–0695

LUNG NEOPLASMS
alcohol and smoking effect, 82–0682
asbestos dose and smoking effects on exposure-death interval, 82–0679
asbestos exposure and smoking as risk factors, 82–0687
East Germany, incidence and smoking relationship, 82–0671
epidermoid, alveolar, undifferentiated, and adenocarcinoma vs. smoking habit, 82–0677
lifetime cigarette smoke-exposed rats, occurrence, 82–0666
morbidity rates, smoking vs. nonsmoking asbestos insulation workers, 82–0686
Mormons vs. non-Mormons, prevalence, 82–0651
morbidity and mortality estimates for 1982, United States, 82–0652
mortality, Norway vs. United Kingdom, 82–0903
mortality rates among black men, 82–0683
mortality vs. smoking in Great Britain, 82–0658
multifactor etiology, 82–0685
nonsmoking wives of smokers, risk, 82–0670, 82–0674, 82–0678, 82–0913
prevention counseling recommended in New Zealand, 82–0794
smoking-associated mortality, United States, 1960-77, 82–0650
squamous cell, oat cell, and adenocarcinoma, smoking relationships, 82–0681
welders, occurrence in, 82–0661
women, Kreyberg I vs. Kreyberg II tumor groups, smoking relationship, 82–0689

LUNGS
contaminants retained in stainless steel welders, magnetic measurement, 82–0703
enzyme induction with benzo(*a*)pyrene and cigarette smoke condensate in rat, 82–0645
lysozyme concentration in rats after cigarette smoke inhalation, 82–0702
smoke particulates, effect and chronic obstructive diseases, 82–0701
smoking role in respiratory disease, conference proceedings, 82–0648

LYMPHOCYTES
seasonal variation in aryl hydrocarbon hydroxylase inducibility, 82–0640
serum angiotensin-converting enzyme activity correlation in sarcoidosis patients, 82–0712

MARIHUANA SMOKING
health risks, tobacco smoking comparison, 82–0909
therapeutic uses and harmful effects compared to tobacco smoking, 82–0912

MASS MEDIA
advertising of cigarettes on radio and television, prohibition, 82–0904
cessation of smoking influence in Canada, 82–0836
role in smoking cessation, conference proceedings, 82–0648
televised antismoking and cancer screening campaigns, effect, 82–0779
television smoking cessation program, effectiveness, 82–0824

MATERNAL-FETAL EXCHANGE
blood-gas exchange, maternal smoking effect, 82–0762
vascular disruption in smokers, 82–0767

MATERNAL SMOKING
adolescent sons, influence of personality and maternal determinants on smoking, 82–0781
assays of aryl hydrocarbon hydroxylase for human placentas, 82–0760
birth weight effect, 82–0764
childhood rhabdomyosarcoma, negative risk factor, 82–0673
fetal and neonatal effects, 82–0757
fetal growth retardation in hypertensives, relationship, 82–0761
fetal hypoxia relationship, 82–0765
fetal infections relationship, 82–0763
lung function impairment in children, relationship, 82–0717
Nevada birth survey, reduced infant birth weight, 82–0766
nicotine effects on fetus, 82–0762
possible health hazards, 82–0755
pregnancy, effects on offspring, 82–0758
vascular disruption resulting in birth defects, 82–0767

MATHEMATICAL MODELS
diagnosis of coronary artery disease prior to valve replacement, smoking index, 82–0745

discriminant function analysis, abstainers and nonabstainers, 82–0808

MENTHOL
- aromatization of tobacco smoke, 82–0868
- method for adding to cigarette filters, 82–0875

MISSOURI
- morbidity, Mormons vs. non-Mormons, 82–0651

MORBIDITY
- cancer, avoidability and trends, 82–0668
- dust exposed Australian waterside workers, smoking factor, 82–0709
- health hazards of smoking, 82–0649, 82–0816
- medical graduates of University of Pretoria, smokers vs. nonsmokers, 82–0784
- Missouri Mormons vs. non-Mormons and Utah Mormons, 82–0651
- prospective medicine and risk appraisal, 82–0653
- prostate neoplasms, smoking factor, 82–0659
- United States, cancer statistics, 82–0652, 82–0692
- Uruguay, smoking-related, 82–0837

MORTALITY
- alcohol abstainers vs. moderate drinkers, smoking effect, 82–0682
- cancer, avoidability and trends, 82–0668
- cancer rates among black men, 82–0683
- cardiovascular death rate decline, smoking as factor, 82–0731
- cardiovascular vs. noncardiovascular diseases, alcohol consumption rate and smoking status effect, 82–0737
- cigarette smoke-exposed rats vs. untreated vs. sham-exposed controls, 82–0666
- coronary heart disease death rate, smokers vs. ex-smokers, 82–0750
- health hazard appraisal questionnaire, smoking responses, 82–0811
- international studies on smoking-related disease trends, 82–0649
- lung cancer, asbestos dosage and smoking effects on interval from exposure to death, 82–0679
- lung cancer in welders, 82–0661
- lung cancer, Norway vs. United Kingdom, 82–0903
- lung cancer, smoking vs. nonsmoking asbestos insulation workers, 82–0686
- lung cancer vs. smoking in Great Britain, 82–0658
- male vs. female all-causes mortality, smoking factor, 82–0656
- Missouri Mormons vs. non-Mormons and Utah Mormons, 82–0651
- myocardial infarct, smoking relationship in 1960s and 1970s, 82–0753
- prospective medicine and risk appraisal, 82–0653
- prostate neoplasms, smoking factor, 82–0659
- pulmonary embolism, smoking as factor, 82–0747
- smoking role in avoidable cancer deaths, 82–0667
- United States, cancer statistics, 82–0652, 82–0692
- United States, smoking-related, 1960-77, 82–0650
- Uruguay, smoking-related, 82–0837

MOTIVATION
- cessation of smoking, commitment-enhancement interviews effect, 82–0842
- cessation of smoking methods, topical bibliography, 82–0865, 82–0866
- cessation of smoking, predictor of success, 82–0850
- cessation of smoking, psychosocial factors, 82–0806
- cessation of smoking to improve employee productivity, 82–0848
- education level vs. smoking habits, 82–0809
- ex-smokers, relapse factors, 82–0815
- hospital patients, smoking against physician's advice, 82–0814
- initiation of cigarette smoking, social factors, 82–0795
- personality factors of smokers vs. nonsmokers vs. ex-smokers, 82–0818
- public responses to smoking hazards, 82–0915
- smokers' reluctance to receive health risk information, 82–0819
- social and behavioral science concepts, 82–0831
- televised smoking cessation program, subject variables, 82–0824
- women, factors in smoking, 82–0797

MOUTH DISEASES
- *Candida albicans* prevalence, smoking relationship, 82–0770

MOUTH MUCOSA
- *Candida albicans* prevalence, smoking relationship, 82–0770
- leukoedema vs. tobacco use in Sweden, 82–0769

MOUTH NEOPLASMS
- smoking-associated mortality, United States, 1960-77, 82–0650
- Sri Lanka, incidence and betel quid chewing relationship, 82–0690
- Thailand, incidence and tobacco use relationship, 82–0805

MUSCLES
- cervicobrachial disorders in cigarette factory workers, 82–0775

MUTAGENESIS
- nitrosamines, formation in curing and metabolism, 82–0614

MUTAGENS
- 2-aminoanthracene and cigarette smoke, 82–0623
- cigarette smoke condensate, decreased by human saliva, 82–0638

MYOCARDIAL INFARCT
- arrhythmia during rehabilitation, smoking relationship, 82–0734
- prognosis comparison between 1960s and 1970s patients, 82–0753
- reliability of self-reported smoking cessation in intervention studies, rapport factor, 82–0841
- siblings, incidence and possible risk factors, 82–0748
- smoking relationship, literature review, 82–0739

NEONATE
- maternal smoking effects, 82–0757
- Nevada, reduced birth weight vs. maternal smoking, 82–0766
- nicotine absorption through breastfeeding, effects, 82–0756

NEOPLASMS
- childhood rhabdomyosarcoma, relative risk from paternal smoking, 82–0673
- dermis in lifetime cigarette smoke-exposed rats, 82–0666
- differing smoking habits as factors in carcinogenesis, 82–0688
- less hazardous cigarettes, risk reduction, the Surgeon General's report, 82–0655
- maternal smoking effect on children, 82–0758
- morbidity and mortality statistics, 82–0652
- morbidity and mortality statistics for the United States, 82–0692
- public disbelief in smoking association in Poland, 82–0798
- radon daughter products and tobacco smoke, combined relationship in rats, 82–0664
- smoking-associated mortality, United States, 1960-77, 82–0650
- smoking role, conference proceedings, 82–0648
- smoking role in avoidable cancer, 82–0667
- tobacco and other risk factors, 82–0668
- tumors among smoke-exposed mice, 82–0676

NERVOUS SYSTEM DISEASES
- benzodiazepine use in anxiety treatment, coffee consumption and smoking effects, 82–0622
- nicotinic stimulation effect on responses of fowl rectum, 82–0634

NEURONS
- nicotine and 4-aminopyridine effects on adrenergic rabbit nerve terminals, 82–0628

NEUROTICISM
- college students, smoking habit relationship, 82–0800
- monozygotic vs. dizygotic twins, smoking relationship, 82–0801
- smoking status relationship, 82–0818

NEVADA
- birth survey, maternal smoking effects, 82–0766

NEW MEXICO
- Hispanics vs. Anglos, respiratory disease and smoking rates, 82–0713

NEW YORK STATE
- nurses, attitudes toward smoking and nonsmoking patients, 82–0908
- teachers, attitude and knowledge about cancer, survey, 82–0833

NEW ZEALAND
- cigarette smoking, 1976 and 1981 census reports, 82–0794

NICKEL
- urinary levels in smoking vs. nonsmoking stainless steel welders, 82–0703

NICOTINE
- basal and stimulated prostaglandin biosynthesis, 82–0632
- behavioral effects in squirrel monkeys, 82–0790
- binding and disposition, rat brain, 82–0646
- blood pressure, body weight, and serum lipids in rats, 82–0639
- chewing gum for smoking cessation, effectiveness, 82–0857
- coronary heart disease relationship, 82–0738
- fate during cigarette smoking, role in U.S. sales, 82–0881
- inhalation levels with carbon monoxide and cholesterol, effect on plasma lipoprotein composition, 82–0728
- intracranial self-stimulation, effect in rats, 82–0620
- lung neoplasm risk factor, 82–0681
- maternal blood, effect on fetus, 82–0767
- maternal-fetal effects via catecholamines, placental blood flow, carboxyhemoglobin levels, 82–0762
- prostaglandins, interaction, 82–0754
- psychobiological models of smoking behavior, nicotine role, 82–0807
- reduced intake, behavioral responses, 82–0786
- reduced leucine incorporation in liver, 82–0627
- sheesha smokers vs. cigarette smokers, health hazard risk, 82–0812
- smoking bout interval or puffs per bout, influence on, 82–0793
- snuff vs. nicotine chewing gum, health warnings and prescriptive control, 82–0911
- ulcer pathogenesis, acid secretion effect, 82–0768
- withdrawal effects, smoking as avoidance mechanism, 82–0813

NICOTINE CONTENT
- early topping effects on tobacco leaf content, 82–0607

plasma concentration in smokers vs. nonsmokers, extraction method for gas chromatography, 82–0635
plasma levels in nonsmokers, smokers before and after cigarette, microextraction method, 82–0636

NICOTINE DERIVATIVES
1-methylhistamine binding sites in brains of rats, 82–0704
N′-nitrosonornicotine, urinary metabolites, 82–0624

NICOTINE METABOLISM
breastfeeding, possible effects on neonate, 82–0756

NICOTINE PHARMACOLOGY
antagonistic effects of d-tubocurarine, mecamylamine in rat brain tissue, 82–0704
aortic endothelial bovine cells, effect, 82–0721
binding and disposition, rat brain, 82–0646
coronary blood flow and thrombosis in dog circumflex artery, effects, 82–0726
lipid and carbohydrate metabolism, possible effects, 82–0644
nicotine and 4-aminopyridine effects on adrenergic rabbit nerve terminals, 82–0628
nicotinic stimulation effect on responses of fowl rectum, 82–0634
reduced conceptus cell number, decreased oviductal and uterine blood flow in rats, 82–0759
reduced leucine incorporation in liver, 82–0626
stressed rats, plasma corticosterone and brain 5-hydroxyindole levels, 82–0619

NITRATES
content in tobacco, human metabolism and pharmacokinetics, 82–0637
tobacco content in relation to smoke yields of aromatic amines, 82–0616

NITROSAMINE CONTENT
snuff products, 82–0675
tobacco, human metabolism and pharmacokinetics, 82–0637

NITROSAMINES
formation during curing, carcinogenesis, 82–0614
human metabolism and pharmacokinetics, 82–0637
levels in saliva of snuff dippers, 82–0675
organ-specific tumor induction in the rat, 82–0663

NONSMOKERS
adults in Canada, 82–0783
American Lung Association's Rights Program, legislative recommendations, 82–0902
angina pectoris patients, prognosis compared with smokers, 82–0729
atherogenesis of coronary arteries compared with smokers, 82–0719
chronic bronchitis incidence vs. passive smoking in the home, 82–0714
ischemic heart disease risk factors compared with smokers and ex-smokers, 82–0742
lung cancer risk among wives of smokers, 82–0670, 82–0674, 82–0678, 82–0913
monozygote twins, comparison with smoking twins, 82–0788
personality factory compared with smokers and ex-smokers, 82–0818
rights, Japanese attitudes, 82–0885

NORNICOTINE
N′-nitrosonornicotine, urinary metabolites, 82–0624
organ-specific tumor induction in the rat, 82–0663

NORWAY
antismoking legislation and restrictive measures, 82–0903
national antismoking campaign vs. U.S. efforts, 82–0825
Oslo study, cholesterol and smoking, 82–0751

NOSE
nitrosonornicotine-induced tumors in rat nasal cavity, 82–0663

NOSE NEOPLASMS
lifetime cigarette smoke-exposed rats, occurrence, 82–0666

OBESITY
hypertension correlation, smoking effect, 82–0722
self-treatment of smoking and obesity, conference proceedings, 82–0648

OBSTRUCTIVE AIRWAY DISEASES
cotton dust exposure, 82–0694, 82–0708
electrocardiographic effects of hypoxemia in patients, smoking relationship, 82–0749
risk reduction via less hazardous cigarettes, Surgeon General's report, 82–0655
smoking as a risk factor, 82–0698
smoking-associated mortality, United States, 1960-77, 82–0650

OCCUPATIONAL DISEASES
adenocarcinoma risk, possible synergistic effect with smoking, 82–0681
asbestos workers, lung neoplasms among smokers vs. nonsmokers, 82–0686
bladder cancer risk, smoking relationship, 82–0672
cervicobrachial disorders in cigarette factory workers, 82–0775

OCCUPATIONS
asbestos insulation workers, 82–0686
asbestos workers, 82–0679, 82–0687
chemical industry workers, 82–0672

cigarette assembly line workers, 82–0775
coalworkers, 82–0710
cotton textile workers, 82–0694, 82–0708
dye workers, 82–0672
firefighters, 82–0715
hairdressers, 82–0672, 82–0772
leather workers, 82–0672
nurses, 82–0908
physicians, 82–0784, 82–0823
printers, 82–0672
scientists, 82–0782
stainless steel welders, 82–0703
waterside workers, 82–0709
welders, 82–0661

ORAL HYGIENE
leukoedema vs. tobacco use in Sweden, 82–0769
tobacco use effects, education programs, 82–0777

OXYGEN TRANSPORT
myocardium, smoking effect, 82–0724

PANCREATIC NEOPLASMS
coffee consumption vs. smoking in etiology of, 82–0662
risk factors, 82–0680
smoking-associated mortality, United States, 1960-77, 82–0650

PARENTAL SMOKING
adolescent smoking relationship, 82–0826
influence on students' smoking habit in Canada, 82–0804
respiratory symptoms in children, relationship, 82–0706

PASSIVE SMOKING
alpha emitters in cigarette smoke, 82–0691
chronic bronchitis incidence in Czechoslovakia, relationship, 82–0714
exposure equivalency to low-tar cigarettes, 82–0771
health hazards, 82–0657, 82–0776, 82–0913
lung cancer risk in nonsmoking wives of smokers, 82–0670, 82–0674, 82–0678
morbidity in nonsmokers, 82–0649
Nonsmokers' Rights Program, American Lung Association, 82–0902
respiratory symptoms in children, relationship to parental smoking, 82–0706
workplace, benefits of elimination, 82–0849

PATENTS
aromatization of tobacco smoke, method, 82–0868
catalysts for oxidizing carbon monoxide to carbon dioxide, 82–0882
cigarette with removable smoke-modifying cartridges, 82–0876
flavor and/or aroma enhancement substance, 82–0879
flavorant additive, heterocyclic-hydroxy-substituted carboxylate compound, 82–0871
hexenol derivatives as aroma or taste enhancers, 82–0874
2-oxabicyclooctane derivatives for flavor and aroma, 82–0877
oxabicyclooctane derivatives, tobacco flavor and aroma enhancers, 82–0878
sealed smoking article to help control consumption, 82–0884
tobacco flavorant additive and process, 82–0872
tobacco flavorant composition, 82–0870
tobacco flavorants, oxobicyclo-4, 4, 0-decane derivatives, 82–0880
tobacco substitute with improved taste and odor, 82–0883

PATERNAL SMOKING
childhood rhabdomyosarcoma, positive risk factor, 82–0673

PEER GROUPS
adolescent smoking relationship, 82–0826
smoking status effect in youth, 82–0840

PEPTIC ULCER
antacid or carbenoxolone treatment, smoking effect, 82–0778
prevalence in smokers vs. nonsmokers, 82–0768
smoking-associated mortality, United States, 1960-77, 82–0650

PERIODONTAL DISEASES
leukoedema vs. tobacco use in Sweden, 82–0769

PERSONALITY
college students, smoking habit relationship, 82–0800
flexibility (Fx) scale of California Psychological Inventory, relation to smoking cessation, 82–0842
male adolescent smokers and maternal determinants, relationship, 82–0781
monozygotic vs. dizygotic twins, smoking and alcohol use relationship, 82–0801
risk-taking and health practice attitudes in German smoking vs. nonsmoking students, 82–0789
smokers vs. ex-smokers vs. nonsmokers, statistical analysis, 82–0818
traits of smokers vs. ex-smokers, 82–0791

PESTICIDES
recommendations for use on tobacco, 82–0609

PHARYNGEAL NEOPLASMS
smoking-associated mortality, United States, 1960-77, 82–0650

PHYSICAL ACTIVITY
aerobic exercises in cessation of smoking, 82–0860

  - coronary heart disease risk reduction, smoking effect, 82–0723

- PHYSICAL FITNESS
  - adults in Canada, smoking status, 82–0783

- PIPE SMOKING
  - leukoedema incidence in Sweden, 82–0769
  - nicotine, cotinine, and carboxyhemoglobin levels, 82–0738

- PIPE TOBACCO
  - Sweden, sales in 1980, 82–0898

- PLACENTA
  - assays of aryl hydrocarbon hydroxylase in humans, 82–0760
  - blood flow, smoking effect, 82–0767
  - maternal smoking effect, 82–0763

- PNEUMOCONIOSIS
  - smoking effect, 82–0710

- PNEUMONIA
  - incidence among smoke-exposed mice, 82–0676
  - Mormons vs. non-Mormons, prevalence, 82–0651

- POLAND
  - ischemic heart disease risk factors, 82–0732, 82–0733
  - public disbelief in association of cancer and smoking, 82–0798

- POLYNESIA
  - Tonga Islands, kava, alcohol and tobacco consumption, 82–0787

- PREGNANCY
  - antismoking counseling recommended in New Zealand, 82–0794
  - birth defects in offspring of smokers, 82–0767
  - family physician's opportunity to counsel against smoking, 82–0821
  - hypertension and smoking, risk factors, 82–0761
  - maternal smoking effect on lung function of children, 82–0717
  - maternal smoking effects on offspring, 82–0758
  - maternal smoking effects, Surgeon General's 1980 report, 82–0654
  - maternal smoking, possible health hazards, 82–0755
  - maternal smoking relationship to fetal hypoxia, 82–0765
  - reduced conceptions and decreased oviductal and uterine blood flow in rats, nicotine effect, 82–0759
  - smoking cessation assistance for expectant mothers, 82–0846
  - smoking hazard risk reduction via less hazardous cigarettes, Surgeon General's report, 82–0655
  - smoking role in human reproduction, conference proceedings, 82–0648

- PREMATURITY
  - maternal smoking effects, 82–0757

- PREVENTION OF SMOKING
  - adolescents, survey indications of possible intervention areas, 82–0826
  - Canada, health education programs, 82–0847
  - Chile, proposed campaign, 82–0657
  - cigarette excise tax increase effect, 82–0889
  - intervention methods, suggestions, 82–0852
  - occupational program guide, 82–0835
  - physicians' role, 82–0823
  - role-playing and discussion among ninth grade students, 82–0828
  - tobacco use and oral health education, 82–0777
  - Washington State, smoking and health teaching guide for elementary and secondary schools, 82–0845
  - Wisconsin Athletes Against Cancer program, 82–0832

- PROGRAMS AND POLICIES
  - North Karelia Project, 82–0831
  - U.S. action on smoking and health, 82–0904

- PROSTATE NEOPLASMS
  - smoking factor, 82–0659

- PROTEIN CONTENT
  - tobacco content in relation to smoke yields of aromatic amines, 82–0616
  - tobacco substitute with improved taste and odor, 82–0883

- PROTEINS
  - nicotine administration in liver, reduced leucine incorporation, 82–0627

- PSYCHIATRIC DISORDERS
  - smoking status relationship, 82–0818

- PULMONARY HEART DISEASE
  - embolism deaths, smoking as factor, 82–0748

- PYRIDINES
  - nicotine and 4-aminopyridine effects on adrenergic rabbit nerve terminals, 82–0628

- RADIATION
  - bronchial epithelium exposure to alpha emitters in cigarette smoke, 82–0691
  - carcinogenic effect in rats of radon daughter products, with tobacco smoke, 82–0664

- RECIDIVISM
  - abstinence versus reduction as goal of smoking control program, effect on relapse, 82–0862
  - aerobic exercises and self-management cessation strategies, success at followup, 82–0860
  - coping behavior for maintenance of smoking abstinence, relapse factors, 82–0864

- nicotine chewing gum use in smoking cessation, relapse rate, 82–0857
- self-efficacy measurement, 82–0856
- situational analysis, 82–0815

REDUCTION OF SMOKING
- aerobic exercises and self-management strategies, 82–0860
- arterial patients, verification by serum thiocyanate levels, 82–0829
- behavioral responses, 82–0786
- biofeedback training, 82–0858
- cigarette excise tax increase effect, 82–0889
- coronary heart disease, risk reduction, 82–0751
- government health campaigns and media influence in Canada, 82–0836
- nondirective and noncontrolling techniques, 82–0855
- scaled article to help control consumption, 82–0884
- smoking control program goal, recidivism effects, 82–0862

RESPIRATORY FUNCTION TESTS
- bacterial clearance and/or detoxification, smoking effect, 82–0699
- carbon monoxide absorption, location, 82–0697
- coalworkers with simple pneumoconiosis, smoking effect, 82–0710
- decreases in Australian waterside workers, 82–0709
- evaluation of tobacco-smoke bioassay model, 82–0700
- firefighters vs. nonfirefighters, 82–0715
- graduated external filter use during smoking, effect, 82–0861
- predicting postsurgical pulmonary complications, smoking history relationship, 82–0711

RESPIRATORY MUCOSA
- epithelial cell population and morphology, smoking effects, 82–0693

RESPIRATORY SYMPTOMS
- occurrence in firefighters, 82–0715

RESPIRATORY SYSTEM
- glycol monoacetin toxicity in animals, 82–0626
- lysozyme concentration in rats after cigarette smoke inhalation, 82–0702
- site-specific absorption of carbon monoxide, 82–0697

RESPIRATORY TRACT DISEASES
- cigarette particulates, effect in smokers vs. nonsmokers, 82–0701
- Hispanics vs. Anglos in New Mexico, disease and smoking rates, 82–0713
- obstructive airway illness, smoking as a risk factor, 82–0698
- occurrence in firefighters, 82–0715

RESPIRATORY TRACT INFECTIONS
- Mormons, lifestyle effects, 82–0651
- Wales vs. western England children, household smoking as factor, 82–0716

RESPIRATORY TRACT NEOPLASMS
- host and environmental factors, 82–0685
- Thailand, incidence and tobacco use relationship, 82–0805

SALIVA
- *N*-nitrosamine levels in snuff dippers, 82–0675
- mutagenicity of carcinogens, effect on, 82–0638

SAUDI ARABIA
- sheesha smoking, health risks, 82–0812

SELENIUM
- kidney level in deceased cancer patients, 82–0641

SEX RATIO
- Canadian students' smoking habit 1960-1980, 82–0804
- cessation of smoking factor, 82–0850
- laryngeal morbidity in smoking vs. nonsmoking patients, 82–0696
- male vs. female mortality rates, smoking factor, 82–0656
- New Zealand smoking habits, 1976 and 1981, 82–0794
- nurses' perceived behavior of smoking and nonsmoking patients, 82–0908
- pancreatic cancer risk factors in men vs. women, 82–0680
- passive smoking effects, males vs. females, 82–0714
- smoking and alcohol consumption in India, societal constraints as factor, 82–0817

SKIN
- glycol monoacetin toxicity in animals, 82–0626

SMOKE INHALATION, ANIMAL
- aortic endothelium and myocardial capillaries permeability in rats, 82–0720
- lung lysozyme concentration in rats, 82–0702
- ontogenic variation in rat liver, lung, and kidney monoxygenase induction, 82–0645
- pathological changes from exposure among mice, 82–0676
- plasma lipoprotein levels in stumptailed macaques, effect, 82–0746

SMOKE STREAMS
- additives for flavor, 82–0871
- analysis of aromatic amines, 82–0616
- flavor constituents, 82–0881
- isolation and identification of two C_{20} seco-cembranoids, 82–0612
- leaf composition correlation, 82–0613

oxabicyclooctane derivatives, aroma and flavor enhancement, 82–0878

SMOKING
role in avoidable cancer mortality, 82–0667

SMOKING ARTICLES
built-in cigarette smoke modification cartridge, 82–0876
ventilated cigarette holders, effect on rate and total number of puffs, 82–0792
water pipes for sheesha smoking, 82–0812

SMOKING CHARACTERISTICS
compensatory smoking when brand switching, 82–0786
measurement of natural smoking behavior in humans, 82–0792
puffs per bout and smoking bout interval, influence of nicotine dose, 82–0793
sheesha smokers vs. cigarette smokers, 82–0812

SMOKING CONTROL PROGRAMS
Canada, health education, 82–0847
cessation among ninth grade students, 82–0828
counselor smoking status, program impact, 82–0830
East Germany, 82–0671
Finland, North Karelia Project, 82–0831
government's role, 82–0915
ischemic heart disease primary prevention programs, 82–0851
measures in 15 countries, review, 82–0905
no smoking policy for business, 82–0849
nonsmokers' rights, American Lung Association, 82–0902
Norway, legislation and restrictive measures, 82–0903
occupational program guide, 82–0835
passive smoking health hazard reduction, 82–0776
restrictions and actions by employers, recommendations, 82–0848
Scottish antismoking exercise with pharmacists, 82–0844
tobacco use and oral health education, 82–0777
United States, educational efforts, 82–0825
United States, governmental actions, 82–0904
Uruguay, proposal, 82–0837

SMOKING HABIT
adolescent son's personality and maternal determinants, relationship, 82–0781
adolescents, saliva thiocyanate as chemical measure, 82–0633
adolescents, survey of immediate and long-term patterns, 82–0826
adults in Canada, 82–0783
blood carboxyhemoglobin concentration relationship, 82–0631
cancer patients, 82–0688
college students in the Soviet Union, attitudes toward smoking relationship, 82–0802
compensation for less hazardous cigarettes, Surgeon General's report, 82–0655
consumption patterns and changes in the United States, 82–0915
consumption patterns in Australia, 82–0901
consumption patterns in New Zealand, 1976 and 1981 census reports, 82–0794
consumption rates in Norway vs. United Kingdom, 82–0903
coronary rehabilitative program participation, as factor with socioeconomic status, 82–0741
cultural determinants in forms of tobacco use, 82–0799
female lung cancer patients, 82–0689
hospital patients and medical staff, 82–0814
light vs. heavy smokers in cell types of lung neoplasms, 82–0677
medical students in India, 82–0810
men vs. women, consumption in East Germany, 82–0671
monozygote twins, 82–0788
nicotine as factor in maintenance, experimental evidence, 82–0790
nicotine withdrawal effects, smoking as avoidance mechanism, 82–0813
oral and respiratory neoplasm incidence relationship in Thailand, 82–0805
predicting postsurgical pulmonary complications with lung function tests, 82–0711
prostate cancer, effect, 82–0659
psychobiological models, and implications for modification, 82–0807
psychological and physiological factors, 82–0816
public and private self-awareness effect, 82–0820
reduction in hospitals, effect of positively vs. negatively worded "no smoking" signs, 82–0785
rural and urban Tonga Island natives, 82–0787
school and college students in India, 82–0817
scientists, publishing relationship, 82–0782
secondary school students and apprentices in Switzerland, 82–0780
students in Canada, 1960-1980, 82–0804
University of Pretoria medical graduates, 82–0784
women, social factors in cigarette consumption, 82–0797
women, Surgeon General's 1980 report, 82–0654

SMOKING MACHINES
Battelle Mk III inhalation machine, 82–0695
instrumental inhaled-smoke dosimeter model for quantitative characterization of whole cigarette smoke, 82–0700

SMOKING SURVEYS
academic biologists, chemists, mathematicians, and psychologists, 82–0782

Canada Health Survey information, 82–0783
Canada, Winnipeg students, 82–0804
cancer patients, 82–0688
hospital patients and medical staff, 82–0814
India, West Bengal medical students, 82–0810
Ontario, Canada, youth, smoking status predictors, 82–0840
South Africa, University of Pretoria medical graduates, 82–0784
Soviet Union, college students, 82–0802
Switzerland, secondary school students and apprentices, 82–0780
United States adolescents, immediate and long-term patterns, 82–0826

SNUFF
N-nitrosamine content, 82–0675
factors favorable to development of smokeless products, 82–0896
nicotine chewing gum regulation comparison, 82–0911
statistical data from selected countries, production and import/exports, 82–0899
Sweden, consumption in 1980, 82–0898

SNUFF-DIPPING
leukoedema incidence in Sweden, 82–0769
N-nitrosamine content, 82–0675

SOCIOECONOMIC STATUS
adults in Canada, smoking habits relationship, 82–0783
cancer mortality rates among black men, 82–0683
college students in the Soviet Union, relationship to smoking habit, 82–0802
coronary rehabilitative program participation, as factor with smoking, 82–0741
cultural determinants of tobacco use, conference proceedings, 82–0648
determining factors in form of tobacco use, 82–0799
HDL cholesterol relationship, 82–0727
pulmonary embolism deaths, smoking as factor, 82–0747

SOUTH AFRICA
University of Pretoria medical graduates, smokers vs. nonsmokers, 82–0784

SOUTH AMERICA
tobacco consumption increase, possible growth area for U.S. companies, 82–0886

SPUTUM
asthma severity and cigarette smoking relationship to production, 82–0707
cell population, smoking effects, 82–0693

SRI LANKA
oral cancer, betel quid chewing relationship, 82–0690

STATISTICAL ANALYSIS
alcohol consumption and mortality, smoking relationship, 82–0737
cancer morbidity and mortality, 82–0652
diseases associated with smoking in the United States, 1960-77, 82–0650
HDL cholesterol and educational level relationship, 82–0727
lung cancer mortality vs. smoking in Great Britain, 82–0658
personality, smokers vs. ex-smokers vs. nonsmokers, 82–0818
tobacco production and import/export data from selected countries, 82–0899
tobacco production, sales, and consumption in Japan, 82–0885

STRESS
nicotine treatment in rats, plasma corticosterone and brain 5-hydroxyindole levels, 82–0619
situational stress study in Turkey, smoking as suppressor variable, 82–0803
smoking cessation relationship, 82–0806
smoking habit relationship, nicotine replacement and withdrawal factors, 82–0813

STUDENTS
Canada, smoking habit 1960-1980, 82–0804
college, Diffusion of Innovation Theory in smoking cessation program, 82–0827
college, psychosocial factors associated with smoking habit, 82–0800
college, risk-taking and health practice attitudes in West German smokers vs. nonsmokers, 82–0789
college, smoking attitudes in the Soviet Union, effect on habit, 82–0802
college, survey of smokers' vs. nonsmokers' receptivity to smoking health risk data, 82–0819
drug abuse survey in India, 82–0817
medical students in India, smoking habits, 82–0810
ninth graders, smoking intervention program, 82–0828
prevention of smoking, Wisconsin Athletes Against Cancer program, 82–0832
smoking awareness curriculum impact, 82–0840
Switzerland, smoking survey, 82–0780

SWEDEN
carboxyhemoglobin levels in urban middle-aged men, 82–0631
leukoedema incidence vs. tobacco use, 82–0769
tobacco consumption in 1980, 82–0898

SWITZERLAND
smoking prevalence among students and apprentices, 82–0780

TAR CONTENT
role in flavor, U.S. cigarette sales, 82–0881

TARS, CIGARETTE
condensate and 2-aminoanthracene as comutagens, 82–0623
flavone and butylatedhydroxyanisole vs. smoke condensate microsomal activation, 82–0647
lung compliance and residual volume effects in rats, 82–0695
lung neoplasm risk factor, 82–0681
mutagenicity decreased by human saliva, 82–0638
particulates, effects on lungs and chronic obstructive diseases, 82–0701

TARS, TOBACCO
early topping effects on tobacco leaf content, 82–0607
sheesha smokers vs. cigarette smokers, health hazard risk, 82–0812

TASTE
electric gustatory threshold in smoking vs. nonsmoking diabetics, 82–0774

TAXATION
arguments against possible increase of Federal excise on cigarettes, 82–0890
cigarette sales effect in Australia, 82–0901
Federal, State, and local tobacco data, 82–0900
reduction of smoking, potential, 82–0889
State and local income from tobacco taxes, 82–0892
Treasury revenues from cigarettes in 1979, 82–0896
West Germany, expected effects on cigarette consumption, 82–0888

THAILAND
tobacco and alcohol use in Chiang Mai province, 82–0805

THIOCYANATES
saliva chemical measure of smoking among adolescents, 82–0633
serum level assessment in arterial patients to measure reduction of smoking, 82–0829

THROMBOSIS
nicotine effects in dogs, 82–0726

TOBACCO
statistical data from selected countries, production and import/exports, 82–0899

TOBACCO ADDITIVES
flavor and aroma enhancer, 82–0872
flavor and/or aroma enhancement substance, 82–0879
flavorants, oxobicyclo-4, 4, 0-decane derivatives, 82–0880
flavoring to enhance taste in weak tobaccos, 82–0869
flavorings, possible health hazards, 82–0873
menthol added to cigarette filters, 82–0875
new dangers in less hazardous cigarettes, Surgeon General's report, 82–0655

TOBACCO AROMA
aromatization of smoke, 82–0868
enhancement and improvement process, 82–0872
enhancement substance, 82–0879
enhancer, oxobicyclo-4, 4, 0-decane derivatives, 82–0880
essential oil components, 82–0615
hexenol derivatives as enhancers, 82–0874
menthol added to cigarette filters, 82–0875
2-oxabicyclooctane derivatives, 82–0877
oxabicyclooctane derivatives, enhancer, 82–0878
tobacco substitute incorporating proteins and protein hydrolysates, 82–0883

TOBACCO CONTAMINANTS
maleic hydrazide, 82–0609
maleic hydrazide residues, reduction by combination with a *N*-benzyldinitroaniline, 82–0608

TOBACCO EXTRACTS
aromatization of tobacco smoke, 82–0868
essential oil components, 82–0615
flavone and butylatedhydroxyanisole vs. smoke condensate microsomal activation, 82–0647

TOBACCO FLAVOR
additive cartridge on cigarette, 82–0876
additive, heterocyclic-hydroxy-substituted carboxylate compound, 82–0871
additive to enhance taste in weak tobaccos, 82–0869
additives, possible health hazards, 82–0873
enhancement and improvement process, 82–0872
enhancement substance, 82–0879
enhancer, oxobicyclo-4, 4, 0-decane derivatives, 82–0880
genetic, agricultural, chemical factors, 82–0881
hexenol derivatives as enhancers, 82–0874
menthol added to cigarette filters, 82–0875
2-oxabicyclooctane derivatives, 82–0877
oxabicyclooctane derivatives, enhancer, 82–0878
synthesized inorganic compound using ion-exchanger, 82–0870
tobacco substitute incorporating proteins and protein hydrolysates, 82–0883

TOBACCO INDUSTRY
Australia, diversification, 82–0901
Brazil, cigarette sales decrease, 82–0893
Britain, sports sponsorship by tobacco companies, 82–0907, 82–0914
cigarette sales up for 1981, 82–0892
continued growth in cigarette production, 82–0890
economic status, 82–0896
expected trends for 1982, 82–0887
Federal, State, and local tax data, 82–0900

high 1981 production, expected trends for 1982, 82–0895
programs and regulating institutions in United States and Canada, 82–0894
proposed legislative controls, 82–0852
smoking control measures in 15 countries, review, 82–0905
Sweden, tobacco consumption in 1980, 82–0898
Third World markets, growth area for U.S. companies, 82–0886
West Germany, tax increase effects on consumption, 82–0888
world manufacturing, consumption, and sales, 1978-1980, 82–0897

TOBACCO LEAF COMPONENTS
burning capacity a function of chemical composition, 82–0610
chemical composition analysis via gas chromatography and mass spectrometry, 82–0611
essential oil, 82–0615
smoke composition correlation, 82–0613

TOBACCO PYROLYSIS PRODUCTS
leaf composition correlation, 82–0613

TOBACCO SMOKE
analysis of aromatic amines, 82–0616
aromatization method, 82–0868
carcinogenic effect in rats, with radon daughter products, 82–0664
carcinogenic substances, correlation of combustion temperature, 82–0610
indoor pollutant, 82–0913
leaf composition correlation, 82–0613
lipid and carbohydrate metabolism, possible effects, 82–0644
promoting effect in carcinogenesis, 82–0669

TOBACCO SUBSTITUTES
control of nicotine chewing gum vs. snuff, 82–0911
lettuce leaves, dried, compatibility with oxabicyclooctane derivatives, 82–0878
nicotine chewing gum, relapse rate, 82–0857
protein and protein hydrolysates smoking composition, 82–0883

TRACHEAL NEOPLASMS
smoking-associated mortality, United States, 1960-77, 82–0650

TRACHEOBRONCHIAL EPITHELIUM
activation of aromatic hydrocarbons, 82–0669
bacterial clearance and/or detoxification, smoking effect, 82–0699

TRIGLYCERIDES
exercise and smoking effect, 82–0723
serum and lipoprotein levels relationship, smoking association, 82–0743

TURKEY
situational stress study, smoking as suppressor variable, 82–0803

TWINS
discordant monozygotes, smoking characteristics, 82–0788
smoking and alcohol use, personality factors relationship, 82–0801

UNION OF SOVIET SOCIALIST REPUBLICS
college students' smoking attitudes, effect on consumption, 82–0802
smoking factor in hypertension and coronary disease, 82–0742

UNITED KINGDOM
advertising ban, cigarette consumption relationship, 82–0906
alcohol consumption and smoking influence on blood lead concentrations in middle-aged men, 82–0642
lung cancer mortality rate and cigarette consumption compared with Norway, 82–0903
lung cancer mortality vs. smoking, 82–0658
respiratory morbidity and lung function in children, South Wales vs. Western England, 82–0716
Scotland, antismoking campaigns, 82–0844
sports sponsorship by tobacco companies, 82–0907, 82–0914

UNITED STATES
adolescent smoking habits, immediate and long-term patterns, 82–0826
cancer morbidity and mortality, 1982 estimates, 82–0652
cancer morbidity and mortality statistics, 82–0692
consumption decline, effects on tobacco industry, 82–0886
legislative history relating to smoking and health, 82–0904
lung carcinoma morbidity in men and women, 82–0681
1980 Maxwell Report on cigar sales and imports, 82–0891
nationwide smoking reduction campaign, 82–0825
tobacco production, pricing and marketing system, 82–0894

URINE
2-aminoanthracene and cigarette smoke as comutagens, 82–0623
catecholamines and their metabolites presence, smoking effect, 82–0625
catecholamines content after acupuncture therapy for smoking, 82–0863

chromium and nickel determinations in smoking vs. nonsmoking stainless steel welders, 82–0703
metabolites of nitrosonornicotine in rat, 82–0624
pH and nicotine excretion, implications in smoking habit, 82–0813

UROGENITAL NEOPLASMS
carcinoma of the cervix, smoking relationship, 82–0665

URUGUAY
mortality, morbidity, and smoking control programs, 82–0837

UTAH
Mormon vs. Missouri Mormon morbidity, 82–0651

UTERUS
reduced blood flow in rats, nicotine effect, 82–0759

VASCULAR DISEASES
nicotine effect on prostacyclin as reduced protection of endothelium, 82–0632

VITAMIN C
ascorbic acid requirements in smokers, 82–0910
possible protectant against acetaldehyde toxicity, 82–0643

WASHINGTON STATE
smoking and health teaching guide for elementary and secondary schools, 82–0845

WISCONSIN
Milwaukee, health hazard questionnaire, smoking responses, 82–0811
prevention of smoking program, Wisconsin Athletes Against Cancer, 82–0832

WITHDRAWAL CLINICS
nicotine chewing gum, effectiveness, 82–0857
pregnant women, in antenatal clinics, 82–0846

WITHDRAWAL SYMPTOMS
negative reinforcement factor in smoking habit, 82–0807
properties of addiction, recidivism effects, 82–0862
smoking to avoid nicotine withdrawal, urine pH factor, 82–0813

WOMEN
cessation of smoking, success factors, 82–0855
educational achievement and smoking relationship, 82–0727
health consequences of smoking, Surgeon General's 1980 report, 82–0654
health hazards of smoking, 82–0816
lower mortality rates, smoking and behavioral factors, 82–0656
lung carcinoma, Kreyberg I vs. Kreyberg II groups, smoking relationship, 82–0689
lung neoplasms in nonsmoking wives of smokers, 82–0913
morbidity and mortality, smoking-related diseases, 82–0649
nurses, perception of smoking and nonsmoking patients, 82–0908
smoking cessation assistance during pregnancy, 82–0846
smoking rates among female scientists, 82–0782
social factors in smoking habit, and guide to quitting, 82–0797

SMOKING AND HEALTH LITERATURE SEARCH REQUEST

Individual requesting search	Title or position
Individual other than requester completing form	Telephone number
Organization	Date of request
Address including zip code	**Purpose of search** ☐ Research ☐ Paper presentation ☐ Writing article ☐ Lecture ☐ Other ______

SEARCH REQUIREMENTS Give below a detailed statement of information requirements together with any necessary background data to meet your needs. Be as specific as possible to help formulate search strategy.

If topic has been reviewed or cited in past issues of the ***Smoking and Health Bulletin*** or ***Bibliography on Smoking and Health*** give pertinent accession number(s) or citation of articles.

Give below other articles which may serve as a guide on subject area (list full bibliographic citation if known)

List relevant key words or index terms appropriate to search.

If a search has been performed on subject utilizing other data sources, please list sources, results of search and search strategy if known.

SEARCH RESTRICTIONS Check appropriate search out-put desired.

☐ Full range (no restrictions)

☐ Year(s) ______________ only

☐ Citation only (no abstract)

☐ English language only

☐ Other language ______________________

____

AGENCY USE ONLY. Do Not Write Below This Line.

Search statement or criteria	Date search received Date search released
	Number of documents retrieved

NOTICE

If you no longer need this publication, check here____, return this sheet to the address below, and your name will be dropped from the mailing list.

If you have a change in name or address, please write the change on this sheet and return it to the address below.

____

____

____

WHEN REQUESTING A CHANGE PLEASE BE SURE YOUR FORMER LISTING ALSO APPEARS ON THIS SHEET.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health
Rockville, Maryland 20857

U.S. MAIL

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS
BOOK

982/4

NEWARK PUBLIC LIBRARY

OCT 5 1982

U. S. GOVT. PUBS.
DEPOSITORY 376

July-August 1982
Abstracts 82-0916-82-1212

Smoking and Health Bulletin

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

HE 20. 7211:

INTRODUCTION

All items appearing in the Smoking and Health Bulletin become part of the permanent document collection of the Technical Information Center (TIC) of the Office on Smoking and Health and can be referred to by their accession number.

In the past, at the end of each calender year, it has been customary to publish the Bibliography on Smoking and Health which contained a cumulative author and subject index based on the individual Bulletins issued that year. However, this year, due to fiscal and other constraints, it may not be possible to issue the index in the form of the Bibliography. All readers are therefore advised to keep the individual Bulletin issues for both 1981 and 1982 until further notice.

The TIC offers bibliographic search and retrieval services for information on smoking, tobacco, and tobacco use through its Automated Search and Retrieval System. Readers are encouraged to submit searches on the search request form found at the end of each Bulletin issue. Multiple copies of this form can be ordered from the TIC. Currently the automated file contains approximately 25,000 records, the majority of which were published and processed into the TIC collection since 1970. The TIC collection totals more than 35,000 items.

Bibliographic and reference services continue to be offered. Readers can take advantage of these services by writing or calling the TIC at the number listed below. Staff limitations currently exists for photocopying services, therefore your cooperation is sought when requesting such services. Until further notice no more than three items per Bulletin per request can be provided. Requests for services above this three document limit cannot be honored. In order that we may continue to provide photocopying services to the largest number of readers, we ask before requesting such items from the TIC collection, to first check your local resources regarding their availability. Visitors may use the TIC library during regular office hours, 8:30 a.m. to 5:00 p.m. weekdays. Advance arrrangements of any planned visits are suggested.

We welcome suggestions or comments from our readership regarding our publications and services.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Telephone: 301-443-1690

ISSN: 0081-0363
July-August 1982
Abstracts 82-0916-82-1212

Smoking and Health Bulletin

CONTENTS

AGRICULTURE 219
CHEMISTRY 220
PHARMACOLOGY AND TOXICOLOGY 222
MORTALITY AND MORBIDITY 227
NEOPLASTIC DISEASES 229
NON-NEOPLASTIC RESPIRATORY DISEASES 232
CARDIOVASCULAR DISEASES 241
PREGNANCY AND INFANT HEALTH 247
OTHER DISEASES AND CONDITIONS 249
BEHAVIORAL AND PSYCHOLOGICAL ASPECTS 254
SMOKING PREVENTION AND INTERVENTION 261
SMOKING CESSATION METHODS 266
TOBACCO PRODUCT ADDITIVES 267
TOBACCO MANUFACTURING AND PROCESSING 271
TOBACCO ECONOMICS 275
LEGISLATION 277
GENERAL 278
AUTHOR AND ORGANIZATIONAL INDEX i
SUBJECT INDEX vii

82–0916. Chaplin, J. F.; Spurr, H. W., Jr. **Altering Condensate Levels in Tobacco Smoke by Genetic Techniques.** *Beitraege zur Tabakforschung International* 11(3): 151-160, January 1982.

Experiments conducted to evaluate the potential for genetically controlling condensate delivery in cigarette smoke are reported. In general, when comparing the condensate delivery potential for different classes of tobacco, Burley delivered higher condensate values followed by dark fire-cured, dark air-cured, flue-cured, and Maryland cultivars. In selecting genotypes for lowering condensate deliveries, the condensate/g of tobacco smoked is a better index than condensate/cigarette. In evaluating low-alkaloid tobaccos, the condensate delivery did not decrease appreciably as the total alkaloids were decreased. The decrease averaged 4 mg of condensate/cigarette when compared with normal-alkaloid types, but the condensate:nicotine ratio increased greatly. The data showed that condensate deliveries from tobaccos vary with the environment, cultural practices, and the weather conditions under which the tobacco is produced. To make progress in breeding for lower condensate delivery in tobacco, it is necessary to assay large numbers of samples; methods for doing this are discussed. To breed low-condensate delivery lines with good smoking flavor, it will be necessary, simultaneously, to select for certain flavor compounds. More research is needed on the flavor chemistry of tobacco and its relationship to condensate. (Auth. Abs. Mod.)

82–0917. Kostowska, B.; Piasecka-Grzeszek, A.; Sadowski, J. **Pozostalosci Herbicydow w Lisciach Tytoniu. [Herbicide Residues in Tobacco Leaves.]** *Pamietnik Pulawski* 72: 183-194, 1980, Polish.

Patoran, Fl-251, Aresin, Afalon, Balan, and Paarlan residues were estimated in tobacco leaves during 1976 and 1977. Field experiments with tobacco were conducted at four locations, by the Central Laboratory of Tobacco Industry (Cracow). The herbicides were applied to the soil 7 days before planting. Leaves were harvested successively at the stage of technological maturity. Herbicide residues were determined in fermented and nonfermented material. The results showed that metobromuron residues were the highest: 0.108-1.746 mg/kg, 0.097-2,895 mg/kg, and 0.675-3.797 mg/kg for rates of 2, 3, and 4 kg/ha, respectively. Linuron and monolinuron residues were lower and reached 0.009-0.605 mg/kg. The lowest amounts of herbicides (about 0.2 mg/kg) were found in leaves from plots treated with Balan and Paarlan. Fermentation of tobacco leaves influenced the herbicide residues in an irregular way. Herbicide residues, except linuron, were lower in the 1977 experimental season than in 1976. (Auth. Abs.)

82–0918. Legg, P. D.; Chaplin, J. F. **Influence of Chlorophyll Content on Smoke Constituents in Burley and Flue-Cured Tobacco.** *Crop Science* 21(5): 692-695, September-October 1981.

Comparisons among standard tobacco cultivars indicated that Burley cultivars were generally higher in tar/puff and nicotine/cigarette puff, and lower in number of puffs/cigarette than flue-cured cultivars. Differences among Burley cultivars were significant for total particulate matter/cigarette, tar/puff, tar/gram of tobacco smoked, and the tar—nicotine ratio. The chlorophyll content influenced the level of 9 of the 10 cigarette characteristics and accounted for the difference between Burley and flue-cured cultivars for number of puffs/cigarette. However, diversity between the two classes of cultivars for tar/puff and nicotine/puff was not totally due to chlorophyll differences. (Auth. Abs. Mod.)

82–0919. Madeley, J. **Kenyan Farmers Risk Their Lives for Smokers.** *New Scientist* 94(1300): 67, April 8, 1982.

The British American Tobacco Company is criticized for supplying the pesticide Aldrin, which is banned by most Western governments, to approximately 8,500 Kenyan farmers who cannot use it safely. A leaflet of instructions given with the chemical states that the pesticide is safe if used with certain precautions, such as washing with soap and water after use, keeping it away from children, avoiding contamination of the water supply, and consulting a doctor if symptoms develop. Although the leaflets are printed in English and Swahili, the variety of languages (15) and the illiteracy in Kenya mean the instructions may not be understood by the farmer. In addition, soap is often unavailable, as are doctors, and the hilly tobacco farming regions make prevention of contaminating streams and rivers impossible. It is claimed that because the pesticide is used, thousands of farm workers are endangered and killed each year.

82–0920. Saunders, J. W.; Pudliner, H. J.; Bush, L. P. **Nicotine Accumulation in Callus and Small Plants of Tobacco (*Nicotiana Tabacum* L.) Grown in Media Supplemented With Nicotine.** *Plant Science Letters* 23(3): 315-319, December 1981.

A study of the possibility of altering tissue levels of nicotine in callus and small plants by growth in the presence of relatively high levels of nicotine is reported. Callus of *Nicotiana tabacum* L. cv. Burley 21 was grown on Murashige-Skoog medium with 0.03 mg/l kinetin and 2.0 mg/l β-indoleacetic acid and nicotine levels of 0-32 mM. With increased nicotine concentrations in the medium, callus nicotine accumulation increased from nearly zero as fresh weight decreased. Nicotine accumulation was as great as 6.2 percent with 60 percent of the dry weight accumulation compared with the control to which no nicotine was added. Burley 21 callus accumulated only nornicotine if nornicotine was the sole supplemental alkaloid in the medium. Callus of Robinson Medium Broadleaf accumulated only nicotine when grown on a medium supplemented only with nicotine, although both green and cured leaves of this cultivar demethylate nicotine to nornicotine. Burley 21 plants at the 6- to 8-leaf stage grown hydroponically with 8 mM nicotine accumulated 2.4 percent nicotine in 10 days compared with 0.3 percent in the controls. (Auth. Abs. Mod.)

CHEMISTRY

82-0921. Chamberlain, W. J.; Arrendale, R. F. **Rapid Method for the Analysis of Volatile N-Nitrosamines in Cigarette Smoke by Glass Capillary Chromatography.** *Journal of Chromatography* 234(2): 478-481, January 15, 1982.

An inexpensive method employs glass capillary gas chromatography and a nitrogen-phosphorus thermionic detector (NPD) to determine differences in volatile nitrosamine contents of various cigarettes. This method enables dimethylnitrosamine (DMNA), diethylnitrosamine (DENA), and nitrosopyrollidine (NPYR) to be monitored. Since cigarette smoke condensate (CSC) is such a complex mixture, almost any separation will still yield an impure and heterogeneous mixture. Therefore, it was felt that an NPD would eliminate any interferences of nonnitrogen containing compounds in the quantitation of the volatile N-nitrosamines. Also, the increased sensitivity of the nitrogen phosphate detector over the flame-ionization detector was helpful, as the nitrosamines are present in the ng/cigarette range. The resulting CSC chromatograms are complex but the volatile N-nitrosamines, DMNA, DENA, and NPYR, can be detected.

82-0922. Dymond, H.F.D.; Yallup, A. E. **Gas Detection.** United States Patent No. 4,257,777, March 24, 1981, 5 pp.

A method and an apparatus for assessing the concentration of nitric oxide in a gas mixture, particularly in tobacco smoke, are described. The apparatus isolates a predetermined amount of the gas mixture and passes it to a reaction chamber, and then supplies to that chamber a gas chemiluminescently reactive with the nitric oxide, thus detecting the oxide through the luminescence emitted in the chamber. The apparatus also has a timing mechanism which operates in response to signals from a source of the gas mixture, such as a cigarette-smoking machine, and can sample smoke at predetermined times. A carrier gas may be used for transferring the predetermined amount of gas mixture from the sampling means to the reaction chamber. (Auth. Abs. Mod.)

82-0923. Federal Trade Commission. **Report of "Tar," Nicotine and Carbon Monoxide of the Smoke of 200 Varieties of Cigarettes.** Federal Trade Commission, Washington, D.C., December 1981, 23 pp.

The Federal Trade Commission's laboratory test results of 200 varieties of cigarettes show an increase in the number of cigarettes with low "tar" (dry particulate matter), nicotine (total alkaloid), and carbon monoxide levels. The laboratory used the Cambridge filter method to test samples which were representative of the 200 varieties of cigarettes available in 50 geographical locations throughout the country during March and April 1981. It is noted that the Commission is investigating a complaint from a group of cigarette companies that the current test method does not accurately assess the "tar" and nicotine levels in Barclay cigarettes relative to the levels of other cigarettes. The cigarette varieties tested appear alphabetically in tabular form with the "tar" and carbon monoxide content reported to the nearest 1/10 mg and nicotine to the nearest 1/100 mg, each with appropriate statistical values. Weight in gram per cigarette and butt length range to the nearest 1/10 mm are provided. Three other tables respectively list varieties in increasing order of "tar" values, nicotine values, and carbon monoxide values.

82-0924. Jarvik, M. E. **Biological Influences on Cigarette Smoking.** In: Krasnegor, N. A. (Editor). National Institute on Drug Abuse Research Monograph 26, U.S. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (ADM) 79-882, August 1979, pp. 7-45.

The biological, biochemical, and physiological correlates of the smoking habit over the three development stages of establishment, maintenance, and cessation are reviewed. Among the topics covered are the chemistry and biochemistry of tobacco smoke, including the known characteristics of carbon monoxide, tar, and nicotine. The metabolism and fate of tobacco in the body is briefly discussed, and genetic and endocrinological factors are covered for possible predisposition effects. The acute effects of tobacco and its components on the central nervous system and the cardiovascular system are reviewed. Also, maintenance of the smoking habit is examined in terms of tolerance, metabolism, and dependence. The physiological effects of tobacco components on smoking maintenance are discussed for the central nervous system, the cardiovascular system, and the endocrinological system. The early and long-term effects of smoking cessation as well as the characteristics of dependence and ways to measure tobacco consumption are addressed. Tabular data on the gas phase and particulate phase constituents of cigarette smoke are included.

82–0925. Lippmann, M.; Cohen, B. **Characteristics of Cigarette Smoke and Its Deposition Pattern Within the Lungs.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). Tampa, Florida, October 20-22, 1976, U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 297-314.

The physical characteristics and behavior of the cigarette smoke aerosol, its particle concentrations, size distributions, deposition pattern within the airways, and the activity of α-emitting radionuclides are discussed. The physical and chemical characteristics of the mainstream smoke vary considerably at any given moment and are determined by (1) the composition and structure of the tobacco leaf; (2) chemicals added during manufacture; (3) packing characteristics and moisture content of the cigarette; (4) the temporal, volume, and flow rate characteristics of the combustion process during each puff; (5) the number of puffs; and (6) the fractional removals of various components in filters at the downstream end. In the case of particles, deposition is strongly influenced by size, size distribution, mass concentration of the aerosol, and effects of size on deposition patterns. Major factors influencing smoke composition include cigarette composition; combustion temperature; puff rate, volume, and number; and age of the smoker. The distribution of aerosol deposition within the respiratory tract is probably more important than the amount deposited, as insoluble or slowly soluble particles on nonciliated alveolar spaces have a longer residence time and are closer to basal cells. It has been suggested that polonium-210, an alpha-emitter in tobacco, is the causative agent in bronchogenic carcinoma. Cigarette concentrations average about 0.4 pCi per unit. Approximately half is transferred to the smoke with 20 to 50 percent in the mainstream. Average concentrations of 4.5 pCi/g have been located at the bifurcations of the segmental bronchi of smokers and levels of three orders of magnitude lower in the bronchial wall and submucosa. See also 81-1114.

82–0926. Merli, F.; Wiesler, D.; Maskarinec, M. P.; Novotny, M.; Vassilaros, D. L.; Lee, M. L. **Characterization of the Basic Fraction of Marijuana Smoke by Capillary Gas Chromatography/Mass Spectrometry.** *Analytical Chemistry* 53(12): 1929-1935, October 1981.

The basic fraction of Mexican marihuana smoke condensate was characterized by combined capillary gas chromatography/mass spectrometry. Enrichment of some trace components was accomplished with high-performance liquid chromatography, permitting tentative identification of nearly 300 nitrogen-containing sample components. A comparison with the basic fraction of tobacco smoke condensate, characterized by the same methodology, revealed that there are both qualitative and quantitative differences between the two condensate fractions. Although many observed differences are primarily quantitative, the amounts can be strikingly different and may have toxicological significance. For instance, nicotine exists only as a trace component in marihuana smoke or plant extract, but accounts for the majority of the tobacco fraction. The total profiles of marihuana and tobacco smoke bases are also very different: the presence of a large array of structures would be more logical for tobacco than marihuana due to the possible pyrolytical decomposition of nicotine and the related bases. A reasonable assumption can be made that the content of proteins and nucleic acids in both plants should be roughly comparable. (Auth. Abs. Mod.)

82–0927. Muramatsu, M.; Umemura, S.; Okada, T. **A Mathematical Model of Evaporation—Pyrolysis Processes Inside a Naturally Smoldering Cigarette.** *Combustion and Flame* 36: 245-262, 1979.

A one-dimensional mathematical model for heat and mass balance has been proposed to elucidate the changes occurring in the temperature and density of the evaporation—pyrolysis zone in a naturally smoldering cigarette. The model considers: (1) pyrolysis of tobacco obeying Arrhenius kinetics, (2) evaporation of water from tobacco following a mass-transfer and rate-determined process, (3) weight loss of tobacco due to pyrolysis and evaporation, (4) internal heat transfer characterized by effective thermal conductivity, (5) heat loss attributable to free convection and radiation from the outer surface of the cigarette and endothermicity of the evaporation process, and (6) smoldering speed. These processes are expressed in a set of simultaneous ordinary differential equations that can be solved numerically by the Runge-Kutta-Gill method. The calculated distribution profiles of temperature and density in the evaporation—pyrolysis zone approximate the experimental results measured using thermocouples and a β-ray attenuation technique. On the basis of the model, the influences of several physical parameters on the shapes of the temperature and density profiles are predicted. (Auth. Abs.)

82–0928. Reist, P. C. **Particle Size and Its Role in Physical and Chemical Interactions of Tobacco Smoke Aerosol.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). Tampa, Florida, October 20-22, 1976, U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 195-217.

A number of factors which influence the size distribution and hence the aerodynamic properties of cigarette smoke are discussed. When the obvious ones are eliminated, those dealing with the aerosol physics remain. These are gravitational settling, wall deposition, coagulation, and chemical speciation. The first three effects can be minimized by keeping the time from smoke formation to exposure as short as possible. However, this approach results in a chemically heterogeneous aerosol. For a more homogeneous aerosol, "aging" is necessary and the other factors become important. It then becomes the responsibility of the experimenter to choose his own set of optimum conditions. (Auth. Abs.) See also 81-1114.

CHEMISTRY

82-0929. Schlotzhauer, W. S.; Martin, R. M.; Snook, M. E.; Williamson, R. E. **Pyrolytic Studies on the Contribution of Tobacco Leaf Constituents to the Formation of Smoke Catechols.** *Journal of Agricultural and Food Chemistry* 30(2): 372-374, March-April 1982.

Pyrolyses of different tobacco leaf varieties, under conditions designed to simulate cigarette smoke formation, demonstrated a correlation between leaf polyphenol levels and pyrolyzate catechol. Bright tobacco varieties contained significantly higher levels of polyphenols as compared to identically cured Burley tobacco varieties and produced correspondingly higher pyrolyzate catechol yields. Leaf constituents, including polyphenols, lignin, cellulose, and sugars, were pyrolyzed to determine potential conversion to catechol and alkylcatechols. Chlorogenic acid, the polyphenol generally present in highest amounts in tobacco leaf, produced the highest pyrolytic conversions to catechol and 4-ethylcatechol. Flavanoids (rutin and quercetin) produced lesser amounts of catechol and 4-alkylcatechols. The polymeric phenolic leaf constituent, lignin, produced significant yields of catechol. (Auth. Abs.)

82-0930. Tso, T. C.; Chaplin, J. F.; Adams, J. D.; Hoffmann, D. **Simple Correlation and Multiple Regression Among Leaf and Smoke Characteristics of Burley Tobaccos.** *Beitraege zur Tabakforschung International* 11(3): 141-150, January 1982.

Leaves from eight stalk positions from four Burley tobaccos, the cultivars Burley 21, Ky 14, and Ky 12 and one breeding line of low-alkaloid Burley 21 (LA Burley 21), were tested. A total of 116 variables, including leaf and smoke characteristics, were determined using established methods. Simple correlations and multiple regressions among selected variables were determined to examine the relationship among these variables. Generally, Burley tobaccos do not differ from bright tobaccos, in that the nitrogenous fractions and combustibility appear to be the major factors affecting nicotine, dry TPM (total particulate matter) delivery, and other major smoke constituents. Stalk position and varieties also demonstrated significant differences in nicotine and dry TPM delivery. Benzo(*a*)pyrene content, however, did not show significant differences among these experimental Burley tobaccos or stalk positions. These correlations can serve as indicators for the chemical nature of the leaf precursors of smoke constituents and can suggest model studies. However, absolute correlations can be best established by studying the fate of ^{14}C-labeled precursors. (Auth. Abs. Mod.)

82-0931. United Kingdom Laboratory of the Government Chemist. **Smoking and Health.** In: *Report of the Government Chemist 1980*, London, Her Majesty's Stationery Office, 1981, pp. 67-85.

Data on the tar, nicotine, and carbon monoxide (CO) levels of 147 brands currently marketed (November 1979 to April 1980) in the United Kingdom, as well as a large number of cigarettes manufactured between 1920 and 1972, are presented. There is a poorer correlation between CO and tar than between tar and nicotine content. For any given tar yield, the CO is directly related to the total length of the cigarette. The filtration and ventilation properties of the cigarette have a greater influence on smoke yields of these components than the composition of the tobacco. The relationship of butt length to smoke properties, however, is variable, and depends on the composition and design of each brand of cigarette. Although tar and nicotine levels have fallen steadily since the 1960's, there has been little change in CO yields.

PHARMACOLOGY AND TOXICOLOGY

82-0932. Abood, L. G.; Reynolds, D. T.; Booth, H.; Bidlack, J. M. **Sites and Mechanisms for Nicotine's Action in the Brain.** *Neuroscience and Biobehavioral Reviews* 5(4): 479-486, Winter 1981.

A variety of pharmacologic, behavioral, and receptor-binding studies were performed to determine the mechanism and site of action of nicotine on the rat brain. When nicotine was given into the lateral or fourth ventricles or directly into the lateral vestibular nuclei of rats, it produced a characteristic prostration often accompanied by tonic seizures and body rotation along a longitudinal axis. Of a variety of brain areas studied, the prostration response could only be elicited from the lateral and, to a lesser extent, medial vestibular nuclei. The response could not be produced by a variety of cholinergic agonists or antagonized with nicotinic cholinergic antagonists, with the possible exception of mecamylamine. A good correlation was observed between the ability of nicotine analogues to antagonize the nicotine-induced prostration and their ability to compete with ^{3}H-nicotine binding to rat brain membranes. ^{3}H-nicotine binding had a high affinity, was stereoselective, and was concentrated in nerve endings and such brain regions as the thalamus, cerebrum, and hippocampus. When nicotine was administered intraventricularly to rats, it significantly elevated the threshold to an aversive shock. It was concluded that many of the central actions of nicotine could not be explained on the basis of traditional nicotinic cholinergic mechanisms. (Auth. Abs.)

82-0933. Andersen, P.; Pedersen, O. F.; Bach, B.; Bonde, G. J. **Serum Antibodies and Immunoglobulins in Smokers and Nonsmokers.** *Clinical and Experimental Immunology* 47(2): 467-473, February 1982.

A study of smoking and nonsmoking cigar plant workers suggested that smoking may suppress the humoral immune response to inhaled antigens, though not to antigens which are supposed to be absorbed through membranes other than those of the bronchopulmonary system. Antibodies to antigens in humidifier water were detected by double immunodiffision in 30 of 63 (47.6 percent) persons who were exposed to aerosols from a water humidification unit in a cigar plant, whereas no antibodies could be detected in 49 unexposed blood donors. The presence of antibodies could not be related to fever or pulmonary symptoms (cough, expectoration, dyspnea). Antibodies were found in 14 (93.3 percent) of 15 nonsmokers and in only 13 (31.7 percent) of 41 smokers, and the titers were highest in nonsmokers. Serum IgG and IgA levels were higher in nonsmokers than in smokers, and the variances within the groups were significantly different. The mean serum IgM values were not significantly different in the two groups. Antibodies to *Candida albicans* and *Escherichia coli* were detected with equal prevalences and titers in smokers and nonsmokers. These findings may partly explain the reported increased incidence of allergic alveolitis in nonsmokers. (Auth. Abs. Mod.)

82-0934. Caton, J. R.; Guerin, M. R.; Dalbey, W. E.; Nettesheim, P. **Tobacco Smoke Inhalation Exposure Dosimetry.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 339-366.

Experimental means of measuring smoke inhalation are discussed, from both a direct and an indirect dosimetric approach. A list of standards for tracer compounds, some compounds currently available, and their properties are given. Also, preparation of the experimental cigarettes and the problems involved in quality control are examined. The relationship of tracer dosimetry to smoke particulates is calculated for expressing dosimetry results. In addition, the parameters for total exposure to smoke, such as puff number and duration and smoke concentration per puff, are plotted against particle deposition in the lungs. Finally, the distribution of smoke particulate matter throughout the animal and its effect on dosage (true dose varies according to internal exposure, external exposure, and preening activities) are reviewed, with recommendations for appropriate containment methods. See also 81-1114.

82-0935. Feyerabend, C.; Higenbottam, T.; Russell, M.A.H. **Nicotine Concentrations in Urine and Saliva of Smokers and Non-smokers.** *British Medical Journal* 284(6321): 1002-1004, April 3, 1982.

Nicotine concentrations were measured in saliva and urine samples collected from 82 smokers and 56 nonsmokers after a morning at work. Each subject answered a series of questions related to their recent intentional or passive exposure to tobacco smoke. All nonsmokers had measurable amounts of nicotine in both saliva and urine. Those nonsmokers who reported recent exposure to tobacco smoke had significantly higher nicotine concentrations ($p < 0.001$) than those who had not been exposed; their concentrations overlapped those of smokers who had smoked up to three cigarettes before sampling. Among the cigarette smokers, the number of cigarettes smoked before sampling had the greatest influence on nicotine concentrations ($r = 0.62$ for saliva and $r = 0.51$ for urine). Neither the nicotine yield of cigarettes nor the self-reported degree of inhalation had any significant effect on nicotine concentrations. (Auth. Abs.)

82-0936. Guerin, M. R.; Stokely, J. R., Jr. **Proband-Machine-Animal Interactions in Inhalation Bioassays.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). Tampa, Florida, October 20-22, 1976, U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 315-338.

The possible effects of interactions between the proband (the agent tested), machine, and animals on inhalation bioassays are discussed. Bioassays are carried out to determine the effect of the proband on the exposed animal. Interactions between the machine used to generate the proband, the proband itself, and the animals can act as perturbations on the bioassay. At the extreme, this perturbation can invalidate the bioassay. Its method of generation and physical/chemical properties make tobacco smoke particularly susceptible to this extreme perturbation. Summarized examples are given of interactions between proband (smoke), animal, and machine that affect tobacco smoke inhalation exposure experiments. (Auth. Abs. Mod.)

82-0937. Guetat, M.; Pignal, C.; Berger, P.; Losten, F.; Sevenier, L. **A Propos de l'Administration du Tiapride en Pre-, Per- et Post-Operatoire, Chez des Malades Comportant un Terrain Ethylique et Tabagique. Etude de Trente Observations. [Administration of Tiapride to Patients With History of Alcohol and Tobacco Consumption, Before, During, and After an Operation. Study of Thirty Cases.]** *Annales de Chirurgie* 35(2): 121-123, February 1981, French.

Tiapride, alone or in association with other tranquilizers, narcotics, and analgesics, was administered to 30 male patients, aged 40 to 80 years (average age 55), with a history of alcoholism and smoking. All were surgical candidates for cancer of the otorhinolaryngologic region. In a preoperative period of 1 to 13 days, administration of 200 to 300 mg/day of tiapride produced good or mediocre results compared to those obtained with an associated sedative for agitation, anxiety, and tremor. Administration of 100 to 200 mg of tiapride with alimemazine and atropine on the day of surgery produced a state of relaxation in all patients and neurovegetative equilibrium. The parenteral application of 200 mg of tiapride in preoperative perfusion allowed a reduction in dosage of narcotics and analgesics, e.g., phenoperidine doses were reduced to 4 mg or less in 25 cases. Postoperative administration of 200 to 300 mg tiapride/day for 5 to 15 days was sufficient in 23 cases to produce analgesia, and to prevent occurrence of predelirium or delirium tremens. In no case were higher than indicated doses of tiapride necessary for the patients with a preoperative history of smoking.

82–0938. Hallak, A. B. **Proton-Induced X-Ray Emission Analysis of Jordanian Cigarettes.** *Journal of Radioanalytical Chemistry* 67(2): 459-465, 1981.

Four Jordanian and one foreign (American) cigarette brands were analyzed by proton-induced X-ray emission spectroscopy to determine the concentration of trace and other elements in the tobacco, wrapping paper, and smoke collected on a filter. The results, tabulated in micrograms or nanograms/cigarette, give a convenient measure of the elements in one cigarette and the expected absolute amount of each element that may enter the body of the smoker. The concentration of elements in Jordanian tobacco is variable but is comparable to the elemental concentration of the American brand except for bromine and strontium which are appreciably higher in the Jordanian cigarettes. This may be attributed to the use of bromine-containing agricultural chemicals and pesticides such as methyl bromide. The presence of poisonous elements such as arsenic might be below the limit of detection by this method because of sample thickness. In the case of wrapping paper, a wide variation in titanium content is noted in Jordanian brands. With cigarette smoke analysis, the presence of arsenic and bromide is noted in all samples. A person who smokes 20 cigarettes/day may accumulate as much as 0.3 to 0.6 µg/day. Since arsenic may cause myocardial necrosis, this accumulation may be hazardous. Jordanian cigarette consumers may also inhale 32-76 µg of bromine daily. The World Health Organization's maximum permissible intake is 1 mg/kg body weight/day; higher intake may damage cardiac tissue.

82–0939. James, W. H.; Moreau, T.; Lellouch, J.; Orssaud, G.; Claude, J. R.; Juguet, B.; Festy, B. **Effects of Alcohol and Smoking on Blood Lead in Middle-Aged British Men. (Letters).** *British Medical Journal* 284(6321): 1046-1047, April 3, 1982.

The conclusion of a previous study (*British Medical Journal* 284(6312): 299-301, January 30, 1982), which attributed elevated blood lead levels in smokers and drinkers to hepatic dysfunction from alcohol consumption, are commented on. It is noted that blood lead levels in dull children are higher than in brighter ones. Therefore, an alternative hypothesis that could be plausible is that extroverted smokers, drinkers, and dull children spend more time on the streets, and are thus exposed to high lead concentrations. Another alternative to the liver dysfunction hypothesis is the increase in mean corpuscular volume (MCV) in smokers and drinkers, which would result in more lead, bound to the surface of red blood cells. However, a study of 442 men showed that an increase in lead intake in wine drinkers is independent of blood lead concentrations. It appears that the elevated blood lead of smokers and drinkers is also independent of MCV since an increase in MCV was accompanied by a decrease in red cell count. See also 82-0642.

82–0940. Kozlowski, L. T.; Frecker, R. C.; Lei, H. **Nicotine Yields of Cigarettes, Plasma Nicotine in Smokers, and Public Health.** *Preventive Medicine* 11(2): 240-244, March 1982.

A major study (*British Medical Journal* 280(6219): 972-976, April 5, 1980) concluded that standard nicotine yields of cigarettes are very poor indicators of plasma nicotine concentrations in smokers and therefore the presumed health advantage of lower yield cigarettes was probably not due to reduced "tar" intake by smokers of these cigarettes. The laboratory data, however, were not analyzed in a way that permits direct generalization to the epidemiological findings. A reanalysis of the data shows that the odds of deriving high yields increases substantially as the standard nicotine yield increases: lower risks associated with lower yield cigarettes may be attributable to reduced nicotine (tar) yields to smokers. Implications for public health and the special risks of ventilated-filter cigarettes are discussed. (Auth. Abs. Mod.) See also 81-0087.

82–0941. Lai, D. Y.; Arcos, J. C.; Argus, M. F. **Interaction of the Tobacco-Specific Nitrosamines, Methylethylnitrosamine and N-Nitrosonornicotine, With DNA and Guanosine.** *Research Communications in Chemical Pathology and Pharmacology* 28(1): 87-103, April 1980.

In vitro binding of the tobacco-specific nitrosamines, methylethylnitrosamine (MEN) and nitrosonornicotine (NNN), to exogenous DNA and guanosine was studied in a rat liver microsome-catalyzed system. MEN (N-(ethyl-1-^{14}C)) binds covalently to calf thymus DNA whereas NNN (N′-(pyrrolidine-2-^{14}C)) binds only to guanosine but not to DNA. Pretreatment of the rats with either phenobarbital (PB) or 3-methylcholanthrene (MC) greatly diminishes the binding of ^{14}C-MEN to DNA. Both MEN-demethylase and -deethylase activities are stimulated by PB pretreatment and inhibited by MC pretreatment, but the degree of stimulation and inhibition of the two dealkylases are not the same. Addition of cytosol to the incubation system does not enhance but suppresses the binding of ^{14}C-MEN to DNA. Inclusion of mitochondria in the system has no effect on the binding. Addition of benzylamine (250 µM), which is a potent inhibitor of dimethylnitrosamine-demethylase, however, totally abolishes the binding of ^{14}C-MEN catalyzed by microsomes. The data suggest that ethylcarbonium ion may be the metabolically activated intermediate of MEN that binds to DNA. (Auth. Abs.)

82–0942. MacDonald, J. C.; Jonsson, A. **Ethyl Nitrite. The *in vivo* Synthesis of a Mutagenic Substance From Tobacco Smoke and Ethanol.** *Acta Chemica Scandinavica* B35(7): 485-487, 1981.

A study among volunteer subjects showed that tobacco smoking and ethanol drinking together could generate the mutagen ethyl nitrite. Subjects' breath samples were examined by gas chromatography. Ethyl nitrite was found among individuals who both smoked and drank, but not among those who only smoked or only drank. The in vivo synthesis of mutagenic ethyl nitrite may help explain this synergistic effect.

82–0943. McCoy, E. C.; Rosenkranz, H. S. **Cigarette Smoking May Yield Nitroarenes.** *Cancer Letters* 15(1): 9-13, January 1982.

A *Salmonella* mutagenicity assay was carried out to investigate the possibility that nitroarenes are formed during the smoking process. Cigarettes were smoked on an automatic smoking machine under standard conditions: a single puff of 2 seconds duration every minute and a puff volume of 35 ml. The particulate matter from three to four cigarettes was collected, assayed, and nitrated. Findings revealed that cigarette smoke condensates can be nitrated to mutagenic substances, presumably nitroarenes. Since nitration can also be achieved by exposure to oxides of nitrogen in the presence of traces of acid, it is possible that it can occur in situ (inhaled smoke) as well as in ambient environment (exhaled smoke). Because nitrates may be present in tobacco, they could lead to the further formation of mutagenic nitroarenes.

82–0944. Norman, T. R.; Fulton, A.; Burrows, G. D.; Maguire, K. P. **Pharmacokinetics of N-Desmethyldiazepam After a Single Oral Dose of Clorazepate: The Effect of Smoking.** *European Journal of Clinical Pharmacology* 21(3): 229-233, 1981.

Smoking apparently affected the pharmacokinetics of N-desmethyldiazepam after oral administration of 20 mg clorazepate to 12 healthy male volunteers (6 smokers; 6 nonsmokers), aged 23-29 years. Plasma levels of desmethyldiazepam were measured by gas liquid chromatography. The half-life of elimination was significantly longer in the nonsmoking volunteers than in the smokers: 54.7±17.7 versus 29.8±9.9 h. Peak plasma concentrations were higher in nonsmokers than in smokers, 413±106 μg/l and 245±50 μg/l, respectively. The sedative effect of clorazepate was less severe in smokers than in nonsmokers. (Auth. Abs. Mod.)

82–0945. Ochs, H. R.; Otten, H. **Einfluss von Alter, Geschlecht und Rauchgewohnheiten auf die Kinetik von Oxazepam. [Effects of Age, Sex, and Smoking Habit on Oxazepam Kinetics.]** *Verhandlungen der Deutschen Gesellschaft fuer Innere Medizin* 87: 1205-1208, April 26-30, 1981, German.

Results from a study show a relationship between smoking and oxazepam clearance. A single dose of 30 mg oxazepam was administered to 22 men, average age 50.7 years, and 9 women, average age 43.7 years. The male subjects smoked an average of 5.9 and the female subjects an average of 9.2 cigarettes/day. Plasma oxazepam concentrations were determined over a 36-hour period. Regression coefficients for the independent variables of age, sex, and smoking habits were calculated in relation to distribution volume, elimination to half-value, and clearance of oxazepam. The latter characteristic is shown to be greatly affected by cigarette smoking in both male and female subjects. It is concluded that nicotine abuse significantly increases oxazepam clearance. The oxazepam kinetics remained unaffected by age or sex.

82–0946. Oreland, L.; Fowler, C. J.; Schalling, D. **Low Platelet Monoamine Oxidase Activity in Cigarette Smokers.** *Life Sciences* 29(24): 2511-2518, December 14, 1981.

The activity of platelet monoamine oxidase (MAO) was found to be significantly lower in normal female subjects who smoked at least five cigarettes per day than in nonsmokers. The platelet MAO activity of individuals who had given up smoking was not significantly different from the activity for nonsmokers. The difference in activities between smokers and nonsmokers, due entirely to a V_{max} rather than a K_m change, was not due to a direct effect of nicotine upon the platelet MAO. In addition, platelet-poor plasma from smokers activated platelet MAO in an identical manner to that from nonsmokers. Personality characteristics such as impulsivity and sensation seeking may be related to both smoking and to low MAO activity. (Auth. Abs.)

82–0947. Pleban, P. A.; Kerkay, J.; Pearson, K. H. **Cadmium, Copper, Lead, Manganese, and Selenium Levels, and Glutathione Peroxidase Activity in Human Kidney Cortex.** *Analytical Letters* 14(B13): 1089-1109, 1981.

Cadmium, copper, lead, manganese, and selenium concentrations in human kidney cortex specimens obtained at autopsy were determined by Zeeman-effect atomic absorption spectroscopy, while glutathione peroxidase activity was assayed in tissue collected at a site adjacent to that selected for the trace metal determinations. Glutathione peroxidase activities were determined with two substrates, hydrogen peroxide and t-butylhydroperoxide. The effects of age, smoking history, sex, race, and blood pressure on kidney cortex trace metal levels and glutathione peroxidase activity were investigated. Mean kidney cortex cadmium concentration was found to be significantly higher in smokers than in nonsmokers ($p=0.010$). In addition, the mean kidney cortex lead concentration of hypertensive individuals was found to be significantly higher than that of nonhypertensive individuals ($p=0.017$). Glutathione peroxidase activities utilizing hydrogen peroxide substrate were inversely correlated to kidney cortex manganese levels ($p=0.004$). (Auth. Abs. Mod.)

82–0948. Radford, E. P.; Drizd, T. A. **Blood Carbon Monoxide Levels in Persons 3-74 Years of Age: United States, 1976-80.** U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, *Advance Data*

From Vital and Health Statistics, No. 76, DHHS Publication No. (PHS) 82-1250, March 17, 1982, 24 pp.

National estimates of the distribution of carbon monoxide (CO) levels in the blood of persons ages 3 to 74 years in the United States by age, smoking status, race, urbanization status of residence, annual family income, and season of the year are presented. In this report, the relative contributions of the four principal sources of carbon monoxide (smoking, ambient or outdoor exposures, occupational exposures, and indoor exposures) to carboxyhemoglobin (COHb) levels are examined. Smoking constitutes the greatest source of exposure to carbon monoxide; the mean COHb level for smokers was more than four times the level for never-smokers, and the smoking effect completely overwhelmed the much more subtle contributions of indoor and ambient sources. In addition, adults who reported a history as ex-smokers may include some current smokers, and some adult never-smokers may have COHb levels of more than 2 percent as a result of occupational exposures to CO; thus only a modest smoking effect is revealed by the data. Smokers' COHb levels were insensitive to increments in environmental CO and showed great variability, making it necessary to limit analysis of environmental factors to never-smokers.

82-0949. Rosecrans, J. A.; Meltzer, L. T. **Central Sites and Mechanisms of Action of Nicotine.** *Neuroscience and Biobehavioral Reviews* 5(4): 497-501, Winter 1981.

Ten years of laboratory research to determine possible central nervous system (CNS) sites and mechanisms by which nicotine produces its pharmacological effects are described. A drug discrimination paradigm was utilized in which rats were trained to detect nicotine using a 2-lever operant procedure. Nicotine acted as a discriminative stimulus (DS) to correct lever responding. Rats had to be able to differentiate nicotine's effects from saline to obtain a food reinforcement. The ability of nicotine to exert its DS effects appeared to be dependent upon a stimulation of central nicotinic—cholinergic receptors which are stereospecific to (-)-nicotine. The nicotine DS could not be mimicked or potentiated by elevating brain acetylcholine levels centrally, suggesting that the receptor action was noncholinergic. Additional studies indicated that nicotine was acting at both reticular formation and hippocampus sites. The hippocampal site of nicotine action also appeared to be dependent on a dopamine neuron connection as well. (Auth. Abs. Mod.)

82-0950. Stetter, J. R.; Rutt, D. R. **Instrumental Carbon Monoxide Dosimetry.** *American Industrial Hygiene Association Journal* 41(10): 704-712, October 1980.

Results of a carbon monoxide exposure study found that smoking could be an important source of exposure in a working environment. A portable electrochemical instrument capable of measuring personal exposures to carbon monoxide was used in the study. The study of the carbon monoxide exposure in a light manufacturing facility illustrated that effective dosimetry could be performed with expectations of accuracy typically better than ± 15 percent, and that voluntary carbon monoxide exposures such as smoking were a significant contribution to the individual's exposure.

82-0951. Stokely, J. R., Jr.; Guerin, M. R. **Tobacco Smoke Inhalation Exposure Systems.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). Tampa, Florida, October 20-22, 1976, U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 219-239.

The basic methodology for delivering tobacco smoke to animals in inhalation studies is discussed, with emphasis on new developments in the field. Two basic methods are described: (1) active exposures, in which the animal generates smoke by drawing on the tobacco product and puffing in the way a human smokes a cigarette; and (2) passive exposures, in which smoke is generated by mechanical means and provided to animals to be inhaled during normal respiration. The two systems for performing mechanical (passive) smoking, advantageous because the smoke produced can be controlled for maximum reproducibility and adherence to accepted parameters, are (1) the static system, in which the puff is taken mechanically and the smoke delivered to an exposure chamber where it is allowed to stand for a selected time period; and (2) the dynamic system, in which the smoke is replenished by a continuous smoke stream generating 2-second, 35 cc puffs on 30 cigarettes. Examples of various mechanical systems are illustrated and schematically diagrammed. Four basic requirements for a viable inhalation system are (1) exposure sufficient to induce the biological response of interest must be provided at the maximum tolerated dose; (2) the exposure must be defined; (3) the chemical and physical properties of the smoke offered must be relevant; and (4) the system must produce minimal extraneous stress on the animal. See also 81-1114.

82-0952. Warren, C.P.W.; Holford-Strevens, V.; Wong, C.; Manfreda, J. **The Relationship Between Smoking and Total Immunoglobulin E Levels.** *Journal of Allergy and Clinical Immunology* 69(4): 370-375, April 1982.

Results of skin testing to common allergens, total serum immunoglobulin E (IgE) levels, and the responses to a respiratory questionnaire found smoking to be associated with increased IgE levels. Data were obtained for 1,768 individuals participating in a survey of a rural population. The geometric mean total IgE levels in a group of subjects without skin-test reactions and with no histories of asthma or hay fever were 14.8 U/ml for men and 11.9 U/ml for women. When individuals were classified according to skin reactivity and smoking history, there was a significant difference in IgE levels among nonsmokers, ex-smokers, and smokers, with smokers having the highest levels. The percentage of subjects with elevated total IgE levels was higher in smokers than in nonsmokers regardless of skin reactivity to common allergens. Among smokers, there was no relationship between intensity and duration of smoking and IgE levels. Among ex-smokers, IgE levels tended to be lower in those who had stopped smoking earlier. (Auth. Abs. Mod.)

82–0953. Zahran, F. **Sheesha Risks. (Letter).** *New Scientist* 93(1298): 804, March 25, 1982.

The conclusions of a previous article (*New Scientist* 93(1293): 459-460, February 18, 1982) that there is less chance for sheesha smokers to develop lung cancer than there is for cigarette smokers are reiterated. Carboxyhemoglobin levels were higher in sheesha smokers compared to those in cigarette smokers, and this caused acute carbon monoxide poisoning, though the effects were transient. Nicotine is trapped in the water at the base of sheesha and no ill effects from nicotine are observed in sheesha smokers. (Auth. Abs. Mod.) See also 82-0812.

See also, **82–0996, 82–1144**

MORTALITY AND MORBIDITY

82–0954. Holme, I.; Helgeland, A.; Hjermann, I.; Leren, P. **The Oslo Study: Social Indicators, Risk Factors and Mortality.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Stockholm, Almqvist and Wiksell International, 1981, pp. 165-181.

A variety of findings and analyses are presented involving the Oslo Study, a combined epidemiologic and preventive study of atherosclerotic heart disease in middle-aged Oslo men. It was confirmed that cigarette smoking is a strong risk factor for coronary heart disease (CHD) and total cancer mortality. Also, mortality from accidents and homicides proved to be almost as strongly correlated to cigarette smoking as CHD mortality. A direct mechanism between accidents and cigarette smoking was hard to detect, but cigarette smoking is greatly dependent on social class/occupation, and accidents have been shown to be highly associated with social class.

82–0955. Junge, B.; Hoffmeister, H. **"Civilization-Associated" Diseases in Europe and Industrial Countries Outside of Europe: Regional Differences and Trends in Mortality.** *Preventive Medicine* 11(2): 117-130, March 1982.

The causes of early death among men and women 55 to 64 years old in Europe, the United States, Japan, and Australia were compared for the period 1958 to 1976. Evaluation of the death rates from heart disease, cerebrovascular disease, lung carcinoma, and other conditions showed that death rates for men were one and one-half to three times those for women; for lung carcinoma the sex ratio was 7 to 1, males to females. For women, the relative reduction of total mortality was five times that for men and for cerebrovascular diseases, double. The increase in lung carcinoma in women was twice as high as that in men. There was an average 11 percent increase in heart disease among men but there was an average reduction in women of 16 percent. There may be physiological reasons for the lower mortality among women; however, it appears that other factors may also play a role. There still exist large sex-related differences in tobacco and alcohol consumption as well as in professional and family life roles. Studies of subpopulations, i.e., Seventh Day Adventists and Mormons, have shown that specific health practices, including tobacco abstinence, have a noteworthy influence on overall mortality. In conclusion, intervention to improve the health status of various populations is most likely to be successful if "unhealthy" norms are altered. (Auth. Abs. Mod.)

82–0956. Omenn, G. S. **Predictive Identification of Hypersusceptible Individuals.** *Journal of Occupational Medicine* 24(5): 369-374, May 1982.

Occupational and environmental health hazards, including susceptibility due to genetic predisposition and factors such as cigarette smoking, which may interact with occupational exposures, are described. Six possible types of interaction are: toxic substances at work which are also present in tobacco smoke, raising the exposure level; chemicals at work which can be pyrolyzed into more harmful substances; tobacco products possibly contaminated by workplace substances; smoking effects that are additive with workplace substances; smoking effects which might be synergistic; and increases in the rate of accidents due to smoking. Smoking may also show important environmental/genetic interactions. Many companies are attempting to help workers to quit smoking.

82–0957. Prior, I.A.M.; Salmond, C. E.; Clements, J.; Beaglehole, R.; Stanhope, J.; Rees, R.; Tuia, H. **Mortality in Polynesians in Isolated Communities and New Zealand.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Stockholm, Almqvist and Wiksell International, 1981, pp. 143-164.

In comparing coronary heart disease risks between undeveloped and developed nations, mortality from all causes was studied in Tokelau, a small, traditional, nonindustrial Polynesian society in the Pacific. Two groups were examined: a sample population on the native islands, and a population which had migrated to New Zealand. Systolic blood pressure, serum cholesterol, body mass index, and smoking were recorded between 1968 and 1977 on males and females above age 15. Few of the native women were smokers. The data provided no evidence that smoking is a mortality risk factor among the island adults. However, mortality was higher among the New Zealand adults, contributed to by higher rates of obesity, diabetes, and smoking.

82–0958. Ware, J. H.; Thibodeau, L. A.; Speizer, F. E.; Colome, S.; Ferris, B. G., Jr. **Assessment of the Health Effects of Atmospheric Sulfur Oxides and Particulate Matter: Evidence From Observational Studies.** *Environmental Health Perspectives* 41: 255-276, October 1981.

The currently available epidemiological evidence from population studies is examined. The separate and multiplicative hazards of sulfur dioxide and particulate matter are discussed. Problems which must be considered in designing studies of chronic exposure include other risk factors such as smoking, occupational status, and confounding factors such as socioeconomic status. The sensitivity of asthmatics to exogenous allergens, dust, smoking, animals, and other factors must be accounted for in attributing changes in symptom prevalence to changes in air pollution concentration. Also, in comparing urban and rural areas, characteristics such as smoking patterns, family size, age distribution, occupation, nutrition, and physical activity often differ. In one study conducted in Cracow, Poland (see 77-1000), it was concluded that smoking, and to a lesser extent, occupational exposure and age, had the greatest effect on respiratory illness prevalence, while air pollution was listed as one of several factors having a smaller effect.

82–0959. Wilhelmsson, C.; Vedin, A.; Ulvenstam, G.; Aberg, A.; Johansson, S.; Wilhelmssen, L. **Tobacco Smoking and Mortality From Various Causes.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Stockholm, Almqvist and Wiksell International, 1981, pp. 328-344.

Cigarette smoking is the single most important risk factor for premature mortality in Western Europe and the United States. Smoking is the major cause of mortality for lung cancer and bronchopulmonary diseases, and a major factor in ischemic heart disease and myocardial infarction. Total mortality is high among those who inhale compared to those who do not, and there is a continuous increase in mortality with increasing daily consumption. The tar and nicotine content of the cigarettes, as well as the duration of the habit, is also related to increasing mortality, particularly in those who began smoking at a young age. Mortality in women shows the same relationship as in men, but to a somewhat smaller degree. Pipe and cigar smokers have higher total mortality than nonsmokers, but less than cigarette smokers.

82–0960. Winkelstein, W., Jr.; Levin, L. I.; Johnson, K. **Re: "Health Effects of Particulate Pollution: Reappraising the Evidence." (Letter).** *American Journal of Epidemiology* 115(3): 471-475, March 1982.

Arguments are presented in criticism of a study (*American Journal of Epidemiology* 110(5): 525-659, November 1979) that found the present U.S. air quality standards for total suspended particulates (75 µg/m3) to be too stringent. It had been concluded that the inability to precisely measure both the compponents of air pollution and their biologic effects, as well as the problem of isolating confounding factors, justified a relaxation of particulate air quality standards to a level closer to that at which obviously harmful responses are consistently observed. Failure to give adequate attention to the following are listed as weaknesses of the report: (1) the relationship between dose—response of exposure and disease effects, (2) disease effects other than those affecting the respiratory system, (3) implications of the consistency of results of studies conducted in different places at different times, (4) multifactorial disease causation and possible synergism, and (5) nature and consequences of error. With respect to smoking and lung cancer, there is a recognized possibility of complex interactions among pollutants in the atmosphere. It is concluded that if a "cleanest possible air" strategy is adopted on an erroneous basis, excessive cost and effort will be expended, while if an "air that is clean enough" strategy is adopted on an erroneous basis, excess adverse health effects will result. See also 81-1757.

82–0961. World Health Organization. **Health Effects of Combined Exposures in the Work Environment.** Technical Report Series 662, World Health Organization, Geneva, 1981, 76 pp.

The issue of the health effects of combined exposures at work, including the possible influence of factors such as smoking, is discussed. Included are theoretical considerations, response mechanisms to combined exposure, a review of relevant research, personal characteristics (such as smoking) which can affect response to combined occupational exposures, combined exposures and occupational cancer, the epidemiological approach, practical implications involving combined exposure, summary and conclusions, and recommendations. Tobacco products used at work may become contaminated by substances occurring in the workplace, while cigarette pyrolysis may change chemical substances into more harmful materials. Many constituents present in tobacco may also occur in the working environment, thus raising the exposure to these substances. Tobacco smoking may potentiate or add to the other effects of occupational exposures.

NEOPLASTIC DISEASES

82–0962. Ames, R. G. **Case—Control Study.** In: *Gastric Cancer Among Coal Miners.* Morgantown, West Virginia, Appalachian Laboratory for Occupational Safety and Health, February 1982, pp. 91-103.

In a case—control study examining gastric cancer incidence among coal miners, it was found that the elevated gastric cancer risk for coal mine dust exposure is limited to coal miners who smoke cigarettes. Neither cigarette smoking in the absence of high exposure to coal mine dust nor high coal mine dust exposure in the absence of cigarette smoking is a gastric cancer risk. This conjunctive effect of cigarette smoking and coal mine dust exposure is a true interactive effect. The mechanism producing the interactive effect requires further study. Two previously identified mechanisms, the lung clearance function and the nitrosation process, appear to be important in the etiology of gastric neoplasms. (Auth. Abs. Mod.)

82–0963. Cross, F. T.; Palmer, R. F.; Filipy, R. E.; Dagle, G. E.; Stuart, B. O. **Carcinogenic Effects of Radon Daughters, Uranium Ore Dust and Cigarette Smoke in Beagle Dogs.** *Health Physics* 42(1): 33-52, January 1982.

The development of pulmonary lesions in beagle dogs was studied following chronic inhalation exposures to radon (at 105±20 nCi/l), radon daughters (at 605±169 working level), uranium ore dust (at 12.9±6.7 mg/m^3), and cigarette smoke. Chronic exposures to mixtures of these agents caused significant lifespan shortening when compared with controls. Survival times of controls and smoke-exposed dogs were equivalent during the 4- to 5-year mean survival time of the dogs exposed to radon-daughter and ore-dust mixtures (with or without added cigarette smoke). Animals with tumors of the respiratory tract generally had cumulative radon-daughter exposures exceeding 13,000 working level months, and their survival time was longer than the survival time of nontumor-bearing animals. Under the conditions of the experiment, exposure to cigarette smoke was found to have a mitigating effect on radon daughter-induced tumors. It is uncertain whether this would be a general finding applicable to other levels of exposure to radon daughters, uranium ore dust, and cigarette smoke. Exposures to smoke from 10 cigarettes/day, 7 days/week produced no significant respiratory tract lesions. However, exposure to 20 cigarettes/day, 7 days/week resulted in pulmonary emphysema, fibrosis, and chronic bronchitis and bronchiolitis. Emphysema and fibrosis were much more prevalent and severe in the dogs exposed to mixtures which included radon daughters and uranium ore dust. (Auth. Abs.)

82–0964. Flanders, W. D.; Rothman, K. J. **Interaction of Alcohol and Tobacco in Laryngeal Cancer.** *American Journal of Epidemiology* 115(3): 371-379, March 1982.

Both alcohol and tobacco use are accepted risk factors for laryngeal carcinoma. Case—control data from previous studies were used to estimate the value of a previously proposed index of interaction between these two risk factors. In addition to the weighting procedure over exposure categories that was previously proposed for estimating a summary index, maximum-likelihood techniques to facilitate the estimation were applied. Overall, moderate synergy was found between alcohol and tobacco in increasing the risk of laryngeal carcinoma, in that exposure to both factors increased the risk about 50 percent more than the increase predicted if the effects of tobacco and alcohol were simply additive. The public health implication of a greater-than-additive effect of joint exposure is the improved prospect of intervention. An antismoking campaign would prevent more laryngeal cancer if alcohol drinkers were persuaded to stop smoking than if an equivalent number of nondrinkers were persuaded to stop smoking. (Auth. Abs. Mod.)

82–0965. Friedlander, E.; Morrison, A. S. **Urinary Tryptophan Metabolites and Cancer of the Bladder in Humans.** *Journal of the National Cancer Institute* 67(2): 347-351, August 1981.

Urinary excretion of six aromatic amine metabolites of tryptophan was compared between a group of 30 patients with bladder cancer and a group of 33 controls selected from the general population of the study area. Measurements were based on a single overnight urine specimen collected at home by each subject, without a "loading" dose of L-tryptophan. For each metabolite, both the distribution and the mean amount excreted were nearly identical for cases and controls. The small case—control differences in mean values were virtually unchanged by adjustment for age, sex, and smoking status. Generally, excretion levels were higher for females than for males. Excretion levels also tended to be higher for older than for younger subjects and for smokers compared to nonsmokers. However, most of these differences were also small. (Auth. Abs.)

82–0966. Gottlieb, L. S.; Husen, L. A. **Lung Cancer Among Navajo Uranium Miners.** *Chest* 81(4): 449-452, April 1982.

Data related to the controversy of cigarettes vs. radiation in the causation of lung cancer was provided by a study of 17 Navajo men, since they represent a singular group who smoked few cigarettes and had a very low rate of lung cancer before the advent of uranium mining. This study centers on Navajo men with lung cancer who were admitted to the hospital from February 1965 to May 1979. Of a total of 17 patients with lung cancer, 16 were uranium miners, and one was a nonminer. The mean value of cumulative radon exposure for this group was 1,139.5 working level months (WLMs). The predominant cancer type was the small cell undifferentiated category (62.5 percent). The low frequency of cigarette smoking in this group supports the view that radiation is the primary cause of lung cancer among uranium miners and that cigarette smoking acts as a promoting agent. (Auth. Abs. Mod.)

82–0967. Hernandez-Llamas, H.; Kimball, A. W. **Critique of a Study of Cancer Incidence and Alcohol/Cigarette Consumption in Hawaii.** *British Journal of Cancer* 45(1): 150-154, January 1982.

The statistical methods used in a previous study (*British Journal of Cancer* 41(6): 929-940, June 1980) are criticized. Shortcomings in the analysis of the data are identified as inappropriate use of regression methods for adjustment, unweighted linear regression for heteroscedastic variables with nonlinear relationships to consumption, omission of important interactions, and improper standardization of incidence rates. Although some of the statistical problems could have been avoided by using, for example, nonparametric techniques or more robust estimation methods on age-specific incidence rates and consumption indicators, the characteristics of the data set used are inappropriate to epidemiological analysis. (Auth. Abs. Mod.) See also 81-0412.

82–0968. Hinds, M. W.; Yang, H.-Y.; Stemmermann, G. N.; Lee, J.; Kolonel, L. N. **Smoking History and Lung Cancer Survival in Women.** *Journal of the National Cancer Institute* 68(3): 395-399, March 1982.

In a study of 223 women with lung cancer, smoking appeared to adversely affect lung cancer survival. The 12-month survival rate was examined according to the patients' smoking status as given in the hospital record. A multivariate regression model was used to adjust simultaneously for age, tumor stage at diagnosis, tumor histology, and treatment. With adjustment for all these covariates, the 136 ever-smokers demonstrated a significantly greater probability of dying during the 12 months after diagnosis than the 87 never-smokers. (Auth. Abs. Mod.)

82–0969. Journal of the American Dental Association. **Smoking and Oral Cancer.** *Journal of the American Dental Association* 104: 16C, 1982.

Smoking can increase the risk of oral cancer, and smokers are strongly advised to quit the habit. Oral cancer causes about 9,000 deaths per year, with smokers greatly outnumbering nonsmokers. Several warning signs of oral cancer are given and it is noted that oral cancer cannot be avoided by changing from cigarette smoking to pipe or cigar smoking. In addition to the risk of oral cancer, smoking can also worsen oral health by staining the teeth, impairing taste and smell, promoting bad breath, and by increasing the susceptibility to other diseases. Smokers should quit smoking and nonsmokers should not start.

82–0970. Keller, C.; de Alleluia, I. B. **Plutonium und Andere Schwere Radioelemente in der Natur. [Plutonium and Other Heavy Radioactive Elements in Nature.]** *Chemiker-Zeitung* 103(4): 139-153, 1979, German.

The origin and concentration of the polonium-210 (^{210}Po) isotope in the tobacco plant; the exposure of smokers' lungs to radiation from the inhalation of ^{210}Po; and the possible effect of inhaled ^{210}Po on cancer incidence are discussed in the framework of a general review involving the naturally occurring heavy radioactive elements. The synergetic effect of carcinogenic polycyclic hydrocarbons, particularly benzo(*a*)pyrene, in the tar products of smoke is considered in discussing the effect of the inhaled ^{210}Po.

82–0971. Kramer, I.R.H. **Oral Leukoplakia.** *Journal of the Royal Society of Medicine* 73(11): 765-767, November 1980.

The means of predicting malignant change in areas of oral leukoplakia is discussed. It has been shown that approximately 4 percent of patients presenting this condition will ultimately develop squamous cell carcinoma, this progression being on a very long time scale. If the leukoplakia is associated with a chronic candidal infection, then the risk of carcinoma may increase. Pathologically, it appears that the homogeneous variety is a lesser hazard than the speckled or erosive lesion and that the site of involvement may also have a marked influence on the risk of malignant change: those occurring on the floor of the mouth and the ventral surface of the tongue seem to carry a very high risk with one in every two possibly becoming malignant. An explanation for this is the tendency for saliva and potential carcinogens to pool in the lower part of the mouth. The tobacco habit seems to be associated with the appearance of the lesion though cigar, pipe, and cheroot smokers have infrequent malignant changes. In conclusion, the principle prognostic features can be expressed as SHTD, where S indicates the site, H the habits (especially tobacco), T represents the clinical type, and D the degree of epithelial dysplasia.

82–0972. McLemore, T. L.; Martin, R. R.; Wray, N. P.; Cantrell, E. T.; Busbee, D. L. **Reassessment of the Relationship Between Aryl Hydrocarbon Hydroxylase and Lung Cancer.** *Cancer* 48(6): 1438-1443, September 15, 1981.

Analysis of aryl hydrocarbon hydroxylase (AHH) levels in pulmonary alveolar macrophages (PAMs) that were freshly lavaged from lung cancer patients showed no increase in enzyme activity over that observed in the age-matched control patient group. AHH was measured in cultured human lymphocytes induced with benzanthracene and in PAMs induced in situ in cigarette smokers. Considered separately, neither lymphocyte AHH nor macrophage AHH levels were distinctly different in either noncancer or lung cancer patients. Considered simultaneously, lymphocyte and macrophage AHH levels were quite different in noncancer and lung cancer patients. The lung cancer patient group was seen to contain a significantly higher percentage of persons with high levels of AHH than did the group of noncancer patients, when more than one tissue was assayed to determine the individual's enzyme levels. (Auth. Abs. Mod.)

82–0973. Miller, D. G.; Somboonchamoen, S. **Cancer in Thailand: Lessons for Cancer Control.** *Preventive Medicine* 11(2): 232-239, March 1982.

Cancer patterns observed in Thailand are not the same as those found in the United States, and appear to be affected by a variety of factors and habits. Using a broad viewpoint of cancer control, the Thai experience is examined, and lessons which are considered relevant to cancer control in the United States are reported. An example of this is that young Thai urban individuals have ceased chewing betel nuts and the incidence of head and neck cancer is declining. The outcome is apparently due to toothpaste advertising promoting the attractiveness of white teeth, which betel nut use would stain. Marketing methods, however, have been utilized to make cigarette use common, which has apparently resulted in higher lung cancer among Thailand's population. The effect has not been equalled by antismoking programs.

82–0974. Muir, C. S.; Choi, N. W.; Schifflers, E. **Time Trends in Cancer Mortality in Some Countries: Their Possible Causes and Significance.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Skandia International Symposium, September 23-25, 1980, Stockholm, Almqvist and Wiksell International, 1981, pp. 269-313.

Time trends in mortality for 15 cancer sites for the 1955-56 to 1975-76 period in 23 countries are reviewed. The tobacco/alcohol relationship in larynx and esophageal cancer is discussed, as well as the etiological role of tobacco in lung and bladder cancer. It is commented that differences in smoking habits between social classes may account for the variables in cancer mortality rates between the classes. No change, or less than a 5 percent increase in 5-year survival, is reported for leukemia, esophageal, stomach, rectal, pancreatic, lung, cervix uteri, corpus uteri, or ovarian cancers. A 5 to 9 percent increase is given for colon, breast, and bladder cancers, and a 10 to 19 percent increase is listed for laryngeal, malignant melanoma, and prostate cancers. A substantial fall in overall cancer incidence and mortality would be noticed if lung cancer were excluded. Thus the real epidemic of tobacco-induced cancer has been overlooked.

82–0975. Nomura, A.; Yamakawa, H.; Ishidate, T.; Kamiyama, S.; Masuda, H.; Stemmermann, G. N.; Heilbrun, L. K.; Hankin, J. H. **Intestinal Metaplasia in Japan: Association With Diet.** *Journal of the National Cancer Institute* 68(3): 401-405, March 1982.

An investigation of intestinal metaplasia (a precursor lesion of gastric carcinoma) among a study sample in Japan, revealed no clear association between cigarette smoking and the extent of intestinal metaplasia. Of the 1,749 volunteers, 206 men and 181 women received a systematic gastroscopic examination and responded to a dietary questionnaire estimating their quantitative intake of 33 food items during the past week. Subjects were also asked questions relating to other factors, including cigarette smoking, education, occupation, and alcohol intake. Cigarette smoking did not appear to be clearly associated with the degree of intestinal metaplasia. (Auth. Abs. Mod.)

82–0976. Perera, F. **Carcinogenicity of Airborne Fine Particulate Benzo(*a*)pyrene: An Appraisal of the Evidence and the Need for Control.** *Environmental Health Perspectives* 42: 163-185, December 1981.

Benzo(*a*)pyrene (BaP), originating from fossil fuel and other organic combustion processes, is largely adsorbed on fine particulate and hence is a widespread atmospheric pollutant, and is the major initiating carcinogen in cigarette smoke. One cigarette contains 10 to 50 nanograms of BaP, so that even passive smoking is a concentrated source of the pollutant. Because of the particulate size, BaP is substantially deposited in the lower lung and readily eluted into surrounding tissue. After elution in the lung, BaP is metabolically activated to its electrophilic, carcinogenic form by a complex enzyme system whose activity is increased by prior exposure to air pollutants, cigarette smoke, and certain drugs. The effect of small, divided doses of BaP has been shown to be greater than that of a single high dose, but no threshold has been established. Epidemiological studies show that mixtures containing BaP (such as urban air, industrial emissions, and cigarette smoke) are carcinogenic and may interact synergistically. In the current absence of health and environmental standards for BaP, there is little incentive to control BaP emissions. Although BaP meets the criteria for regulation under the Clean Air Act, no such standards have yet been proposed. (Auth. Abs. Mod.)

82–0977. Peto, J.; Seidman, H.; Selikoff, I. J. **Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment.** *British Journal of Cancer* 45(1): 124-135, January 1982.

Duration of asbestos exposure, length of time since exposure, and smoking are a better basis for risk assessment than age at first exposure. Mesothelioma death rates in asbestos workers appear to be proportional to the 3rd or 4th power of time from first exposure for both pleural and peritoneal tumors, although the peritoneal—pleural ratio depends on fiber dimension and type. Smoking is unrelated to mesothelioma incidence. The ratio of asbestos-induced lung cancer to mesothelioma was 3.9 among smokers exposed to asbestos at age 25 or over, 1.8 among smokers exposed prior to age 25, and 0.2 among nonsmokers. In contrast to duration, age at first exposure has little or no influence on incidence, which supports the "multistage" model of carcinogenesis.

82-0978. Rigel, D. S.; Friedman, R. J.; Levine, J.; Kopf, A. W.; Levenstein, M. **Cigarette Smoking and Malignant Melanoma. Prognostic Implications.** *Journal of Dermatologic Surgery and Oncology* 7(11): 889-891, November 1981.

In a prospective study of 178 patients with malignant melanoma, a subset of 33 patients (18.5 percent) was identified to be at significantly higher risk for developing metastatic disease based on history of cigarette smoking. Patients in this high-risk group (current smokers with a >15 pack-years of smoking history) had 2-year disease-free survival rates of 74.2 percent vs. 92.3 percent for the remaining patients (p=0.008). A possible explanation of this phenomenon is that chronic smoking diminishes host defense mechanisms and results in an adverse effect on the biologic behavior of established malignant melanomas. (Auth. Abs.)

82-0979. Stock, S. L. **Passive Smoking and Lung Cancer. (Letter).** *Lancet* 1(8279): 1014-1015, May 1, 1982.

Objections are raised to Lee's criticism (*Lancet* 1:(8275): 791, April 3, 1982) of Hirayama's study of lung cancer in passively smoking women (*British Medical Journal* 282: 183-185, January 17, 1981). It is pointed out that since the composition of tar and dimethylnitrosamine differs between sidestream smoke and mainstream smoke, passive exposure of certain carcinogens can be as great as from direct smoking. Also, a woman with a smoking husband probably associates with other smokers, so the number of cigarettes consumed by the husband can be better regarded as an index, rather than as a measure of exposure. Another objection states that noninhaling pipe and cigar smokers who passively inhale their own smoke do have an increased risk of lung cancer, though contrary statements have been made in criticizing Hirayama's study. The filtering ability of the nose is claimed to be overestimated, and the different chemical nature of stale ambient smoke combined with varying breathing patterns between smokers and nonsmokers may negate the efficacy of such filtration. However, the chief objection raised against public smoking is an esthetic one. See also 82-1072 and 81-1088.

See also, **82-0925, 82-0953, 82-1025**

82-0980. Trell, E.; Bjorlin, G.; Andreasson, L.; Korsgaard, R.; Mattiasson, I. **Carcinoma of the Oral Cavity in Relation to Aryl Hydrocarbon Hydroxylase Inducibility, Smoking and Dental Status.** *International Journal of Oral Surgery* 10(2): 93-99, April 1981.

Evidence is presented to support the conclusion that smoking is a prominent exogenous factor associated with carcinoma of the oral cavity. The smoking patterns of 20 consecutive patients (16 men, 4 women) with oral cancer were investigated, and the aryl hydrocarbon hydroxylase (AHH) inducibility distribution and dental status in this variety of squamous cell carcinoma were assessed. The patients, 18 smokers and 2 nonsmokers, were referred to the General Hospital, Malmo, Sweden. The control group used consisted of 118 healthy employees of the University Hospital, Lund, Sweden, and various research workers; 41 were smokers. The AHH level proved high in eight patients, intermediate in seven, and low in five. High AHH inducibility was more common (p= >0.001) and low AHH inducibility less common (p= <0.001) than in the controls. Dental status was poor in only two patients indicating that poor dental status was of minor importance.

82-0981. Vineis, P.; Segnan, N.; Costa, G.; Terracini, B. **Evidence of a Multiplicative Effect Between Cigarette Smoking and Occupational Exposures in the Aetiology of Bladder Cancer.** *Cancer Letters* 14(3): 285-290, December 1981.

New findings from an on-going case-control study of urinary tract cancer in men in the Province of Torino, Italy, appear to confirm the possibility of a more than additive effect between occupation and tobacco smoke in the etiology of bladder cancer. The analysis of tobacco smoke and occupational histories of the first 225 case-control pairs (average age = 59 years) is described. The overall relative risks for tobacco smoke and for occupations known to entail a risk for bladder cancer were respectively 4.8 (95 percent confidence limits, 2.4-9.3) and 1.7 (1.0-2.7). There was a clearcut dose—response relationship between different categories of cigarette consumption and risk for urinary tract cancer. For each level of cigarette consumption, relative risks were compared between men reporting and nonreporting occupations at risk. This analysis suggested a multiplicative effect between the two factors. (Auth. Abs. Mod.)

NON—NEOPLASTIC RESPIRATORY DISEASES

82-0982. Agnew, J. E.; Little, F.; Pavia, D.; Clarke, S. W. **Mucus Clearance From the Airways in Chronic Bronchitis—Smokers and Ex-Smokers.** *Clinical Respiratory Physiology* 18(3): 473-484, May-June 1982.

Factors such as smoking, which might influence mucus clearance among subjects with chronic bronchitis, were examined. For some patients with chronic obstructive bronchitis, it was found that giving up smoking may lead to poorer mucus clearance because of decreased transport by productive cough.

The study assessed lung clearance of inhaled radioactive particles, predominantly deposited in the smaller bronchi, in 60 tests on patients with chronic bronchitis. When tests were matched for sputum production, clearance in ex-smokers was not significantly faster than in patients still currently smoking. Clearance was significantly faster in the patients who coughed frequently and produced high volume of sputum. (Auth. Abs. Mod.)

82-0983. Battigelli, M. C. **Smoke Inhalation: Respiratory Physiologic Monitoring in Man.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 37-45.

A review of the effect of smoking on respiratory function test parameters is presented. The acute consequences of smoking include modification of ciliary activity, airways constrictive responses, macrophagic activation, metabolic effects at the cellular and tissue level, bronchial and bronchiolar changes, secretory effects, and hypertrophic and metaplastic changes. These eventually merge into chronic symptoms. The measurements which reveal disturbed function in smokers include spirometry, isovolume flow, closing volume, nitrogen plateau, resistance of airways, frequency-dependent compliance, gas exchange function, and ciliary activity. The technological limitations of studying ciliary activity are discussed. See also 81-1114.

82-0984. Bayes, J. **Asymptomatic Smokers: ASA I or II? (Letter).** *Anesthesiology* 56(1): 76, January 1982.

An argument for Physical Status Classification of ASA II as opposed to ASA I for asymptomatic smokers is presented. ASA II is defined as a "mild to moderate systemic disturbance." To support this, it is noted that: (1) asymptomatic smokers have shown statistically significant increases in closing volumes consistent with small airways disease and mucociliatory transport abnormalities; (2) patients smoking > 10 cigarettes/day have increased postoperative morbidity; and (3) nonbronchitic smokers have been shown to have a 53 percent incidence of chest complications vs. an incidence of 23 percent in nonsmokers. It is concluded that the evidence suggests both quantifiable disease and increased perioperative risk in asymptomatic smokers.

82-0985. Beck, G. J.; Doyle, C. A.; Schachter, E. N. **A Longitudinal Study of Respiratory Health in a Rural Community.** *American Review of Respiratory Disease* 125(4): 375-381, April 1982.

Smoking appeared to adversely affect lung function and respiratory symptoms in a rural study population. White residents of Lebanon, Connecticut, a rural community, were studied cross-sectionally in 1972 and in a followup survey in 1978. The study examined prospectively the growth and decay of lung function and the prevalence, attack, and remission of respiratory symptoms in 1,262 persons. These longitudinal findings were related to sex, age, and smoking habits. Most of the prevalence results were consistent with a harmful effect on symptoms from smoking. Smokers usually had larger attack rates and smaller remission rates than nonsmokers. The longitudinal data showed that lung growth continued into early adulthood (25 to 34 years of age) with a leveling off and decline beginning at 35 years and older. The rates of growth in both males and females were affected by their smoking status, with smokers having less growth and greater decline than nonsmokers. In particular, lung function growth was impaired in young smokers as early as 15 to 24 years of age, and an actual decline was seen in the females of this age group. (Auth. Abs. Mod.)

82-0986. Bellon, G.; Hornebeck, W.; Lagrue, G.; Martin, J. P.; Robert, L. **Methode Simple de Detection des Deficiences Fonctionnelles et Genetiques en Alpha-1-Antitrypsine du Serum Humain. [A Simple Method for Detection of Functional and Genetic Alpha-1-Antitrypsin Deficiencies of Human Serum.]** *Annales de Biologie Clinique* 39(4): 185-188, 1981, French.

Insoluble elastin immobilized in Agar plates was used as substrate to quantitate the elastase inhibitory capacity of human sera. This technique was found valuable to evaluate the genetic deficiency of alpha-1-antitrypsin (Z phenotypes) and also the functional loss of the elastase inhibitory capacity in smokers. A significant decrease of elastase inhibitory capacity was demonstrated in the sera of smokers between 40 and 80 years of age, as compared to nonsmokers of the same age. This method may be useful for the evaluation of patients suffering from elastic tissue diseases (emphysema, arteriosclerosis). (Auth. Abs.)

82-0987. Bilimoria, M. H.; Ecobichon, D. J. **The Induction of Rat Lung Aryl Hydrocarbon Hydroxylase by Diluted Smoke From Commercial Cigarettes.** *Toxicology* 22(3): 205-210, 1981.

Using the British-American Tobacco Co. (B.A.T.)-Mason inhalation system and cigarettes delivering tar equal to 1, 4, and 20 mg/cigarette, it was shown that induction of pulmonary aryl hydrocarbon hydroxylase (AHH) in male Sprague-Dawley rat lung is an extremely sensitive system to inhaled cigarette smoke. AHH induction was observed at a 200-s exposure to 40 puffs of a 1:5 dilution of smoke from an ultramild cigarette delivering 1 mg tar. At this low concentration, it is not even possible to accurately weigh the total particulate matter from diluted smoke in the exposure chamber. This extremely sensitive rat pulmonary enzyme system may prove valuable in the biological testing of modern low-tar cigarettes.

82-0988. Carp, H.; Miller, F.; Hoidal, J. R.; Janoff, A. **Potential Mechanism of Emphysema: α_1-Proteinase Inhibitor**

Recovered From Lungs of Cigarette Smokers Contains Oxidized Methionine and Has Decreased Elastase Inhibitory Capacity. *Proceedings of the National Academy of Sciences* 79(6): 2041-2045, March 1982.

The elastase inhibitory capacity per mg of α_1-proteinase inhibitor (α_1PI) was measured in the bronchoalveolar lavage (BAL) fluid from 26 healthy smokers and 24 nonsmokers. Activity was decreased by 40 percent in smokers' BAL fluid compared to nonsmokers. This effect was demonstrable by using human neutrophil elastase as well as porcine pancreatic elastase as test enzyme, and was reproducible when selected individuals in each group underwent lavage on repeated occasions. In contrast, the functional activity of α_1-antichymotrypsin was not decreased in smokers' BAL fluid. Crossed antigen—antibody electrophoresis confirmed that inactivation of α_1PI was responsible for the decrease in the elastase inhibitory capacity of smokers' BAL fluid. The α_1PI purified from smokers' BAL fluids contained methionine sulfoxide (4 mol/mol of inactive α_1PI), whereas α_1PI from nonsmokers' BAL fluid did not. Smokers' α_1PI was indistinguishable from nonsmokers' α_1PI on the basis of electrophoretic mobility, molecular weight, and immunoreactivity. Thus, oxidation of methionine residues in lung α_1PI is associated with cigarette smoking. Because chemical oxidation of α_1PI in vitro causes loss of its elastase inhibitory activity, the present observations suggest that methionine oxidation may also be the cause of decreased functional activity of lung α_1PI in smokers. It is concluded that oxidative inactivation of α_1PI in the lungs of cigarette smokers may play a role in the development of pulmonary emphysema in this group. (Auth. Abs.)

82-0989. Chowdhury, P. **Correlation of Cigarette Smoking With Human Serum Antitrypsin Activity.** *Indian Journal of Medical Research* 74: 763-766, November 1981.

Investigation of the influence of cigarette smoke on the serum antitrypsin activity of smokers as compared to nonsmokers revealed that cigarette smoke produces a significant depression in serum antitrypsin activity, although serum antitrypsin content was not affected by smoking. These results are consistent with the reported findings of decreased α_1-antitrypsin activity in rat lung due to cigarette smoke. The decreased serum antitrypsin activity in the smokers' serum may explain the development of emphysema in heavy cigarette smokers which may be induced as a result of protease-antiprotease imbalance in smokers' serum. (Auth. Abs.)

82-0990. Clarke, J. R.; Jaeger, M. J.; Zumrick, J. L.; O'Bryan, R.; Spaur, W. H. **Respiratory Resistance From 1 to 46 ATA Measured With the Interrupter Technique.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 52(3): 549-555, March 1982.

Measurements of respiratory resistance by an interrupter technique, made in six subjects during a U.S. Navy saturation dive to 457 m, showed that smoking can affect resistance. Repeated measurements were made at five depths with complete pulmonary function testing after the dive. Increase in resistance was about linear with gas density (ρ). Three smokers exhibited a significantly steeper slope of the respiratory resistance versus ρ relationship than three nonsmokers. Individually plotted data at various depths show a different pattern for smokers than nonsmokers, especially for expiratory resistances.

82-0991. Cohen, D.; Crowther, T. S.; Gibbs, G. W.; Becklake, M. R. **Magnetic Lung Measurements in Relation to Occupational Exposure in Asbestos Miners and Millers of Quebec.** *Environmental Research* 26(2): 535-550, December 1981.

Fe_3O_4 particles (ferrimagnetic) are usually attached to asbestos fibers (nonferrimagnetic) in the chrysotile asbestos mining and milling industries; therefore, a magnetic measurement of FE_3O_4 in the lungs of workers in these industries could help determine the amount of asbestos which has been inhaled and retained in their lungs. As a first assessment of this method, magnetic measurements were made of Fe_3O_4 in the lungs of 115 miners and millers (ages 26 to 50 years, smokers and nonsmokers) in Quebec. These measurements at an industrial site were found to be feasible and practical; however, the amount of Fe_3O_4 seen in the lungs of those with welding exposure was large enough to mask the Fe_3O_4 contributed by asbestos, and this subgroup was considered separately. For the remainder (nonwelders), the amount of Fe_3O_4 was plotted against a total dust exposure index (asbestos and other dust) estimated for each worker. Although the correlation between these quantities was not high, it was statistically significant at the 1 percent level. Because retained asbestos is likely to increase with increasing exposure to total dust, this correlation suggests that a magnetic lung measurement of a chrysotile miner or a miller does reflect, to some extent, the amount of asbestos in his lungs. There was considerable scatter in the data, partly due to individual variations in deposition and clearance, to which this method is sensitive. When the data of only the nonsmokers were plotted, the amount of Fe_3O_4 was greater than for the total group of nonwelders. This is consistent with previous findings that less dust is deeply deposited in the lungs of smokers, due to constriction of small airways. (Auth. Abs.)

82-0992. Cordeiro, R. **Tabac et Macrophage Alveolaire. [Smoking and Alveolar Macrophage.]** *Broncho-Pneumologie* 30(5): 394-403, September-October 1980, French.

Comparative clinical, radiological, respiratory function, serum biochemical, and alveolar macrophage (AM) studies were carried out in selected population subgroups of 16 smoking and 16 nonsmoking uranium mine workers. Among the smokers, the median consumption was 11.75 cigarette packs/year. Respiratory symptoms were found to be more frequent in smokers, with association of three symptoms present in seven smokers, but there was no association for nonsmokers. The laboratory studies of AM in the bronchoalveolar lavage liquids

(BALL) showed significant differences between the smokers and nonsmokers with respect to: the AM count, which was higher in BALL of the smokers; certain morphologic and tinctorial characteristics of AM; a greater adherence of AM of the smokers to nonsilicone material, at least in a few protocols; and AM migration capability in capillaries after only 2 days. Comparative biochemical studies of the BALL and peripheral blood of both population subgroups revealed that the only significant difference was in the lymphocyte B count. The count was found to be in BALL consistently one-half that in peripheral blood within each subgroup.

82–0993. Efthimiou, J.; Higenbottam, T.; Holt, D.; Cochrane, G. M. **Plasma Concentrations of Lignocaine During Fibreoptic Bronchoscopy.** *Thorax* 37(1): 68-71, January 1982.

Cigarette smoking and other factors likely to influence mucosal absorption of lignocaine were examined. Use of lignocaine as an anesthesia for fibreoptic bronchoscopy, though accepted as safe, may produce significant plasma concentrations as it is readily absorbed from mucosal surfaces. Results showed that peak concentrations were affected only by dose per unit weight; no changes were observed with respect to sputum production, airflow obstruction, or cigarette smoking. A major proportion of the total dose of lignocaine was required to anesthetise the nose, pharynx, and larynx, only a small proportion being needed for the bronchial tree. Lignocaine gel (2 percent of dose per unit body weight (w/v)) was preferred by patients, and in a study of 10 volunteers, produced lower plasma concentrations when used as a topical anesthetic than did lignocaine aerosol (10 percent w/v) or lignocaine solution (4 percent w/v). (Auth. Abs. Mod.)

82–0994. Elwood, P. C.; Thomas, H. F.; Sweetnam, P. M.; Elwood, J. H. **Mortality of Flax Workers.** *British Journal of Industrial Medicine* 39(1): 18-22, February 1982.

A study of flax workers suggested that smoking may be associated with excess mortality among those with byssinosis. A total of 2,528 workers in flax mills in Northern Ireland were followed up for 16 years (followup was 97 percent complete). Deaths were identified and date and cause ascertained. Expected deaths were calculated on the basis of age and sex specific rates for Northern Ireland, but could not be standardized for smoking habit. There was a small excess in the mortality of workers who had had byssinosis at the time of the original survey, but there was no evidence that the more severe grades of byssinosis were associated with higher mortality than the less severe grades. Workers who smoke are known to have an increased risk of developing byssinosis. The lack of a relationship between more severe grades of byssinosis and increased mortality suggests that cigarette smoking may be responsible for most of the excess deaths. (Auth. Abs. Mod.)

82–0995. Eschmann, G.; Schulz, V. **Die Dichteabhaengigkeit der Fluss-Volumen-Kurve in der Frueherfassung Bronchialer Erkrankungen. [The Flow-Volume Curve in Early Detection of Bronchial Disorders.]** *Praxis und Klinik der Pneumologie* 35: 770-771, November 1981, German.

The standard spirometric and body plethysmographic parameters of small airways function were measured and the flow-volume curves during breathing of a He/O$_2$ mixture were plotted for 27 male smokers and an equal number of nonsmokers, the average age being 24.7 years. The smokers had smoked an average of 21 cigarettes/day over an average period of 7.4 years. A four-step discriminatory analysis of the cited experimental data produced "discriminant scores" which were found to represent highly specific evidence of small airways disease. Thus, discriminant scores of the smokers were from -0.207 to -2.189 and those of nonsmokers were in the +0.451 to +1.558 range. This analytical procedure constitutes a reliable and highly sensitive diagnostic tool for small airways disease, in contrast to a single parameter that tends to fluctuate considerably from one individual to another.

82–0996. Fleischmann, R.; Heinrich, R.; Malchow, H.; Bozler, G. **Einfluss von Rifampicin und Zigarettenrauch auf die Theophyllinclearance. [Effects of Rifampin and Cigarette Smoke on Theophylline Clearance.]** *Verhandlungen der Deutschen Gesellschaft fur Innere Medizin* 87: 1202-1205, April 26-30, 1981, German.

Theophylline was administered by intravenous infusion to five smoking and five nonsmoking patients with pulmonary tuberculosis, aged 30 to 56 years. Theophylline clearance was determined before tuberculosis therapy with rifampin and on the 10th day of treatment. The total theophylline clearance in smokers, which increased from 4.8 l/hr before the treatment to 7.3 l/hr under treatment, remained nearly constant in nonsmokers. The cause of the accelerated elimination of theophylline in smokers under treatment for tuberculosis remains undetermined because of the relatively small sample of patients. In contrast to other study data, no difference was found in theophylline elimination from plasma between smokers and nonsmokers before tuberculosis therapy started. A significant difference was determined between 17 smokers and 35 nonsmokers in another group with respect to the metabolizing activity of two enzymes in the microsomal fraction of the liver in vitro, in the absence of an induction effect from drugs.

82–0997. Giesler, G. J., Jr. **Byssinosis.** *Journal of the Medical Association of Georgia* 71: 109-111, February 1982.

A position paper on the definition of and compensation for byssinosis, prepared as an official statement of the Georgia Thoracic Society, is presented. Information about the disease includes: risk factors, such as smoking; data on the background of the disease; its clinical features; pulmonary function abnormalities; pathology; and possible etiology. A grading system for byssinosis is described, but smoking can bring in extraneous influences. If the byssinosis patient is a smoker, it can be hard to separate the effects of smoking and the effects of cotton dust exposure. The Society recommends that the Georgia Lung Association continue current attempts to assist employers and employees to set up sound smoking cessation and education programs.

82-0998. Haines, A. P.; Imeson, J. D.; Meade, T. W. **ABH Secretor Status and Pulmonary Function.** *American Journal of Epidemiology* 115(3): 367-370, March 1982.

Among current cigarette smokers in the Northwick Park Heart Study in N.W. London, England, secretors of ABH antigen had a higher mean peak expiratory flow rate than did nonsecretors, suggesting that secretion of ABH antigen into the respiratory tract may have a protective effect against pulmonary damage by noxious agents. The relationship was independent of other factors known to affect peak expiratory flow rate. Among current cigarette smokers, the unadjusted peak expiratory flow rate was less in nonsecretors only in those age 40 and over. This suggested that any protective effect by ABH secretion may become apparent only after long-term exposure to cigarette smoke. No significant differences in peak expiratory flow rate by secretor status were detected in nonsmokers, or in pipe and cigar smokers, perhaps because pipe and cigar smoking caused less respiratory function impairment than cigarettes. A smaller difference was seen among secretors and nonsecretors who were ex-cigarette smokers than among those who were current cigarette smokers, which is consistent with the theory of ABH's possible protective effect appearing after long-term cigarette smoke exposure. (Auth. Abs. Mod.)

82-0999. Harf, R.; Pacheco, Y.; Biot, N.; Perrin-Fayolle, M. **Status Immunitaire du Poumon et Tabagisme. [Lung Immunity and Smoking. (Letter).]** *Nouvelle Presse Medicale* 10(23): 1942, May 23, 1981, French.

Results of experimental studies are cited which support the findings of a previous report (*Nouvelle Presse Medicale* 9(42): 3171-3175, November 8, 1980), which concluded that smoking plays a major role in modifying the immune response of the lungs by enhancing immunosuppressive activity. It is further noted that the reduced incidence of extrinsic allergic alveolitis and sarcoidosis in smokers is explained by the immunosuppressive activity of tobacco, as opposed to the exacerbated immune responses characteristic of the two pathologic conditions. See also 81-0384.

82-1000. Hiller, F. C.; McCusker, K. T.; Mazumder, M. K.; Wilson, J. D.; Bone, R. C. **Deposition of Sidestream Cigarette Smoke in the Human Respiratory Tract.** *American Review of Respiratory Disease* 125(4): 406-408, April 1982.

Measurement of deposition of sidestream cigarette smoke in the human respiratory tract is important for assessing the health effects of sidestream cigarette smoke. The deposition fraction of sidestream cigarette smoke in five normal adult male volunteers (three nonsmokers, one ex-smoker, one occasional pipe smoker) was measured using sidestream smoke at a concentration similar to that encountered indoors with smokers present. The mean deposition was 11 percent. Using the data collected, it was possible to estimate the deposition of sidestream smoke over an 8-hour day in a normal person breathing 10 L/min in a room with 500 $\mu g/m^3$ of smoke. Assuming a deposition fraction of 0.11, the total deposition would be 0.26 mg. Deposition varies widely, depending upon such variables as duration of exposure, breathing pattern, and concentration of sidestream smoke. In contrast, smokers who inhale mainstream smoke would deposit from 0.7 mg to 14 mg from one cigarette containing, respectively, 1 mg and 20 mg of tar, assuming a 70 percent deposition. Although deposition from sidestream smoke is low compared with the mass deposited in the lungs of one-pack/day smokers, it cannot be assumed that sidestream smoke is innocuous. (Auth. Abs. Mod.)

82-1001. Hubert, H. B.; Fabsitz, R. R.; Feinleib, M.; Gwinn, C. **Genetic and Environmental Influences on Pulmonary Function in Adult Twins.** *American Review of Respiratory Disease* 125(4): 409-415, April 1982.

Pulmonary function tests were administered to 127 monozygotic and 141 dizygotic white male twin pairs 42 to 56 years of age. Initial twin analyses showed significant genetic variance ($p<0.001$) for both forced vital capacity (FVC) and forced expiratory volume (FEV_1), whether or not adjustments were made for differences in age and body size. After adjustment, heritability estimates were 0.91 and 0.77 for FVC and FEV_1, respectively. Further analyses indicated that the observed heritability of FVC resulted from the effects of pack-years of smoking as well as from genetic factors related to body size. These findings suggest that there were no other significant genetic determinants of FVC. In contrast, heritabilty of FEV_1 could not be explained by constitutional factors, such as height and weight, or by cigarette smoking or propensity for cardiopulmonary disease symptoms. Additional analyses based on frequency of twin contact, which served as an indirect measure of environmental similarity between cotwins, suggested a shared genetic and environmental variation in FEV_1 that argued in favor of early as well as adult environmental influences on pulmonary function. (Auth. Abs.)

82-1002. Hughes, J. A.; Hutchison, D.C.S.; Bellamy, D.; Dowd, D. E.; Ryan, K. C.; Hugh-Jones, P. **Annual Decline of Lung Function in Pulmonary Emphysema: Influence of Radiological Distribution.** *Thorax* 37(1): 32-37, January 1982.

In patients with emphysema, for the majority of lung function indices, the rate of decline is faster in smokers than in ex-smokers. The effects of the radiological distribution of emphysema on the annual rate of change of lung function indices were followed for a minimum period of 3 years (range 3 to 13 years) in 56 male patients with pulmonary emphysema and normal serum alpha$_1$-antitrypsin. The subjects were placed in three categories: upper zone (UZ), lower zone (LZ), and generalized (G). The influence of smoking was taken into account with subjects in each radiographic category being divided into ex-smokers (group EX-S) and continuing smokers (group S). The rate of decline of forced expiratory volume in 1 second (FEV_1), vital capacity (VC), transfer factor for carbon monoxide (Tco), and permeability coefficient (Kco) was greater in group S than EX-S in all three X-ray categories. In group S, FEV_1, VC, Tco, and Kco declined significantly faster in UZ emphysema than in G emphysema. In group EX-S, FEV_1, Tco, and Pao_2 (arterial oxygen tension) declined significantly faster in UZ than G emphysema but VC remained unaltered. Kco and $Paco_2$ (arterial dioxide tension) did not change significantly in any radiographic category in either group S or EX-S. Patients with G and LZ emphysema underwent very similar physiological changes over the course of time. Those with UZ emphysema, however, complained of exertional dyspnoea at an earlier age; a number of their lung function indices deteriorated at a faster rate than in patients from the other two categories, suggesting that UZ emphysema is a different disease entity. (Auth. Abs. Mod.)

82-1003. Kambe, M.; Tsubokura, T.; Kobayakawa, T.; Ogoshi, M.; Hiramoto, M.; Mittmap, C. **Study on Mechanical Properties of Lung Between Smokers and Nonsmokers.** *Hiroshima Journal of Medical Sciences* 30(4): 345-350, December 1981.

Smoking appeared to adversely affect lung functions in a study group of five smokers and five nonsmokers. The mechanical properties of the lungs were measured and detailed investigations were carried out on smokers during periods when they were smoking and when smoking was discontinued for 3 months. Results of the study showed that (1) the lung work of breathing for smokers tended to be lower than that of nonsmokers; (2) the lung resistance of smokers tended to be higher than that of nonsmokers; (3) the pressure-volume loops of smokers shifted farther left than that of nonsmokers; and (4) the disturbances in several mechanical properties observed during the period of smoking improved with the discontinuation of smoking. (Auth. Abs. Mod.)

82-1004. Lan, S.-P.; Shy, C. **Effect of Air Pollution on Chronic Respiratory Disease in the New York City Metropolitan Area, 1972.** *Environmental Health Perspectives* 42: 203-214, December 1981.

Smoking was found to be the most important factor in determining the level of severity of air pollution on chronic respiratory diseases (CRD) in the New York metropolitan area in 1972. Information was obtained by questionnaire from 5,416 white long-term residents of four communities, without occupational exposure to dust or fumes. Confounding factors, including smoking status, age, level of education of head-of-household, and crowding within the home, were examined. The effect of air pollution showed differential patterns among the smokers and nonsmokers. Among the smokers, no air pollution effect was observed. Among nonsmokers, however, a statistically significant difference was observed in females. Male nonsmokers demonstrated a similar pattern, but the effect was not statistically significant. Other possible factors that could contribute to the difference are discussed. (Auth. Abs. Mod.)

82-1005. Landser, F. J.; Clement, J.; Van de Woestijne, K. P. **Normal Values of Total Respiratory Resistance and Reactance Determined by Forced Oscillations. Influence of Smoking.** *Chest* 81(5): 586-591, May 1982.

A forced oscillation technique for measuring respiratory impedance apparently lacked sensitivity for detecting the influence of smoking. In 407 healthy male subjects, smokers and nonsmokers, the resistance and reactance of the respiratory system were determined between 4 and 24 Hertz, using a forced oscillation technique. The values were significantly correlated with age, weight, height, forced expiratory volume in first second, and vital capacity. After standardization of the data for the latter variables, there were no differences between smokers and nonsmokers. The technique thus lacks sensitivity for the detection of the effects of smoking. (Auth. Abs. Mod.)

82-1006. Lissner, H. G.; Trauth, H. A.; Staecker, K.-H. **Die Lungenfunktion Junger, Gesunder Maennlicher Raucher und Nichtraucher in Abhaengigkeit vom Auspraegungsgrad der Persoenlichkeitsdimension "Extraversion." [Lung Function of Young Healthy Male Smokers and Nonsmokers in Relation to the Score of the "Extraversion" Dimension of Personality.]** *Praxis und Klinik der Pneumologie* 35: 718-720, November 1981, German.

Extroversion was measured using the Eysenck Personality Inventory, conventional spirographic and body plethysmographic parameters were determined, and subjective pulmonary symptoms were surveyed by questionnaire in 24 smokers and 28 nonsmokers. The average age of all subjects was 27 years. The subjects who smoked had averaged 25 cigarettes/day for a period of 10 years. The tidal volume and minute respiratory volume after forced smoking were found to be significantly higher in smokers relative to nonsmokers, especially in introverted as compared with extroverted smokers. The questionnaire survey showed more frequent pulmonary symptoms in smokers than in nonsmokers. A state-of-health scale found a highly significant difference for a positive sense of health in nonsmokers. Introverted nonsmokers required significantly more repeated measurements than extroverted nonsmokers to attain their maximum vital capacity (VC). In the test before forced smoking, the VC of extroverted smokers was significantly higher than the VC of introverted smokers.

82-1007. Maehrlein, W.; Krause, M.; Kummer, G.; Roth, J. **Beitrag ueber Beziehungen des Closing-Volume zu Weiteren Lungenfunktionspruefungen. [Relationship Between Closing Volume and Other Lung Function Tests.]** *Zeitschrift fur die Gesamte Innere Medizin* 36(21): 835-837, November 1, 1981, German.

Closing volumes (CV) were determined in 82 asymptomatic and 38 symptomatic (elevated CV) males aged 20 to 59 years. These results indicated that the closing volume test may be suitable for early diagnosis of obstructive airways disease. However, in comparing the results of closing capacity tests, slope indices, and standard lung function parameters between 18 smokers and 24 nonsmokers, the CV test did not reveal deteriorating lung function among smokers whereas the other tests did. Thus the use of the CV test in early diagnosis of small airways obstruction is less promising among smokers.

82–1008. Mastrangelo, G.; Saia, B.; Marcer, G.; Piazza, G. **Epidemiological Study of Pneumoconiosis in the Italian Poly(vinyl chloride) Industry.** *Environmental Health Perspectives* 41: 153-157, October 1981.

An association between smoking and slight chest X-ray alterations was suggested in a study of industrial workers in the Italian poly(vinyl chloride) (PVC) industry. Among 1,216 workers, 20 cases of pneumoconiosis were found. None of these workers had had previous exposure to organic or inorganic dusts; 731 had been exposed to PVC dust, and 485 had been exposed to vinyl chloride monomer (VCM) alone. Chest X-rays were taken, and mild nonspecific alterations were found both in the group exposed to PVC dust and in the group exposed only to VCM. All 20 workers with PVC-induced pneumoconiosis had been exposed to high PVC dust pollution for at least 5 years; 16 subjects (80 percent) were either smokers or ex-smokers. Such changes (observed in 388 cases, 31.9 percent of the total study population) were related mainly to age and smoking habits, and the role of exposure was minor. (Auth. Abs. Mod.)

82–1009. McMillan, G.H.G.; Rossiter, C. E. **Development of Radiological and Clinical Evidence of Parenchymal Fibrosis in Men With Non-Malignant Asbestos-Related Pleural Lesions.** *British Journal of Industrial Medicine* 39(1): 54-59, February 1982.

A study of parenchymal fibrosis among dockworkers found no significant effects from smoking. After assessment of radiographs taken in 1966, 201 men employed at HM Dockyard, Devonport, were judged to have pleural abnormalities due to exposure to asbestos but to be free from small opacities, mesothelioma, or bronchial carcinoma. In 1976, data that was available from 175 subjects (155 survivors, 20 dead), 23 of whom had developed small opacities by 1976 or their death, appeared to indicate no consistent relationship between smoking and the development of small opacities. In 1976, the 155 survivors who participated were reexamined to determine the attack rates of parenchymal fibrosis or malignant disease, or both. The observed parenchymal fibrosis attack rates were substantially higher than those observed in a sample of men with no initial pleural abnormality but were unrelated to smoking habit, age, occupation, duration of exposure to asbestos, or type of pleural abnormality. The number of cases of malignant disease was too small to allow any reliable conclusions. (Auth. Abs. Mod.)

82–1010. National Research Council. **Byssinosis: Clinical and Research Issues.** Washington, D.C., National Academy Press, 1982, 151 pp.

Aspects of byssinosis are discussed including the clinical, epidemiologic, pathologic, and etiologic features of the disease; pulmonary function; characterization of cotton dust; recommendations for research; an appendix on the microbiologic characterization of cotton dust; and a minority report. Smoking and its possible relationships with byssinosis are mentioned. It is reported that in most studies, cigarette smoking has acted as a confounding variable in evaluating the dust dose—response relationship in byssinosis. Smoking has also been found to be an important factor affecting the prevalence of byssinosis. Active cigarette smoking is a host factor that may affect individual response to the work environment. Research suggests smoking may aggravate the symptoms of byssinosis.

82–1011. Noro, M.; Tanaka, U.; Imai, T.; Notoya, Y.; Yoshikawa, O.; Miyajima, I.; Kojima, T.; Itoh, K.; Hatsushiba, S.; Nagai, S.; Fukuda, J. **The 1st Report: Relationship of Cigarette Smoking to Some Laboratory Examination Data. (Lung Function Test, Peripheral Blood Counts, Levels of Serum-Cholesterol, B-Lipoprotein, and Triglyceride).** In: Yasaka, T. (Editor). *Progress in Health Monitoring (AMHTS),* Excerpta Medica, International Congress Series 539, 1981, pp. 410-415.

A tendency toward pulmonary function deterioration and polycythemia was observed among smokers in an examination of 3,467 healthy adults in Japan. This deterioration was in proportion to the number of cigarettes smoked and the number of years the subject had been smoking. The subjects (2,423 males, 1,045 females) were questioned as to number of cigarettes smoked/day and number of years they had been smoking, and tested for vital capacity, forced expiratory volume and maximum expiratory flow, peripheral red blood cells, hemoglobin, hematocrit values, white blood cells, serum cholesterol, triglyceride and B-lipoprotein. Subjects were classified by smoking habit (nonsmoking, 1-20 cigarettes/day, and 21 cigarettes/day) and subdivided by age. Results show that deterioration of pulmonary function was slow in developing. There was a tendency among some, but not all, groups and subdivisions of smokers to show high hematocrit values, high values for the number of red and white blood cells, and high hemoglobin values. Changes in values of serum cholesterol, triglyceride, and B-lipoprotein according to age category were noted; however, no sex differences were observed in either of the three groups of subjects.

82–1012. Peslin, R.; Jardin, P.; Bohadana, A.; Hannhart, B. **Contenu Harmonique du Signal de Debit Pendant L'Expiration Forcee Chez L'Homme Normal. [Harmonic**

Content of the Flow Signal During Forced Expiration in Normal Man.] *Clinical Respiratory Physiology* 18(3): 491-500, May-June, 1982, French.

To define the minimal frequency response requirements for spirometric measurements, the harmonic content of forced expiratory flow was assessed in 63 healthy subjects. The amplitude of the harmonics was found to decrease almost exponentially with increasing frequency. The average cumulated frequency content beyond 10 Hz represented less than 5 percent of the total frequency content in men, and less than 3 percent in women. No significant difference has been observed between male smokers and nonsmokers. The harmonic content per unit frequency was found to be correlated to maximal flows at middle and low lung volumes and to maximal mid-expiratory flow up to 4 Hz. For FEV_1, maximal flow at 75 percent of the vital capacity and, particularly, peak expiratory flow rate (PEFR), the correlations were still significant at quite higher frequencies. The data suggest that, except for PEFR, all these indices may be obtained with a good accuracy when the frequency response of the equipment is flat up to 5 to 7 Hz. (Auth. Abs.)

82-1013. Poukkula, A.; Huhti, E.; Makarainen, M. **Chronic Respiratory Disease Among Workers in a Pulp Mill. A Ten-Year Follow-Up Study.** *Chest* 81(3): 285-289, March 1982.

A 10-year followup study was carried out on 659 men (aged 18 to 64 years when first surveyed by questionnaire and simple spirometry in 1967), to determine the effect of smoking habits on respiratory symptoms and ventilatory function. Symptoms tended to increase during the followup period and were most marked among the men who continued to smoke. The remission rates of respiratory symptoms were high in all the smoking groups, however, and cough and phlegm actually diminished during the 10 years in the men who stopped smoking after the first survey. The followup survey in 1977 showed a prevalence of 6 to 8 percent for severe airways obstruction in the ex-smokers and smokers, but only 2 percent in the lifelong nonsmokers. The average decrease in 1 second forced expiratory volume (FEV_1) was 44 ml/year in the total series, 37 ml/year in the lifelong nonsmokers, and 49 ml/year in the continuous smokers ($p<0.001$). The men who had stopped smoking before the first survey resembled the nonsmokers, whereas those who had stopped during the followup period resembled the continuous smokers in respect to the decrease in FEV_1. (Auth. Abs.)

82-1014. Schwartz, H. R.; McDuffie, F. C.; Black, L. F.; Schroeter, A. L.; Conn, D. L. **Hypocomplementemic Urticarial Vasculitis. Association With Chronic Obstructive Pulmonary Disease.** *Mayo Clinic Proceedings* 57(4): 231-238, April 1982.

It is suggested that lung disease results from the interaction of two major risk factors—smoking and an immunologically mediated process not yet identified. Since 1973, 16 patients (10 smokers) with hypocomplementemic urticarial vasculitis have been identified and followup conducted. Preliminary diagnostic criteria are the presence of typical urticarial skin lesions and low levels of serum complement (all components), plus two of the following: dermal venulitis, arthritis, glomerulonephritis, episcleritis or uveitis, recurrent abdominal pain, and C1q precipitin in plasma. Exclusions are systemic lupus erythematosus, mixed cryoglobulinemia, elevated antinuclear antibody titer, hereditary deficiency of a complement component or of C1 esterase inhibitor, and presence of antinative DNA or hepatitis B antigen. The renal involvement is relatively benign, and generally the patients do well and respond to specific treatment when this is indicated. Eight of ten smokers had evidence of chronic obstructive pulmonary disease (COPD), one of whom died of this complication. In three patients, COPD developed at a young age after relatively low pack-year cigarette-smoking histories. (Auth. Abs. Mod.)

82-1015. Spanel, R.; Vorlaender, W.; Galle, J.; Haferkamp, O. **Ultrastruktur von Alveolarmakrophagen bei Rauchern und Emphysematikern. [Ultrastructure of Alveolar Macrophages in Smokers and Patients Affected With Emphysema.]** *Verhandlungen der Deutschen Gesellschaft fur Pathologie* 65: 496, 1981, German.

Ultrastructural indications of activation of alveolar macrophages in cigarette smokers compared with nonsmokers were detected by examining materials obtained from 25 patients by bronchial lavage or by lavage of the lung resection material. The so-called smoker's inclusions detected in alveolar macrophages were correlated with actual smoking habits. The emphysematous alterations in the lungs were correlated with long-term smoking habits. The smoker's inclusions were not directly related to the extent of emphysematous alterations. Besides their correlation with smoking habits, the smoker's inclusions are considered as an indication of an enormous phagocytotic efficiency of the cells. The diameter of alveolar macrophages increased to 45 m by exposure to smoke vs. a maximum 30 m diameter in nonsmokers. Small undeveloped monocyte-like alveolar macrophages (10 to 20 m diameter) were found in substantially greater numbers in smokers than in nonsmokers. This finding was taken as an indication of an increased new growth of alveolar macrophages due to exposure to smoke. Thus, smoking appears to increase the actual concentration of proteolytic enzymes in the lungs by activation and multiplication of macrophages.

82-1016. Sykut, E. **Wplyw Zanieczyszczen Chemicznych Powietrza w Srodowisku Pracy na Wystepowanie Przewleklego Niezytu Oskrzeli Oraz na Sprawnosc Wentylacyjna Pluc. [Effect of Chemical Contamination of the Atmosphere at Work on the Occurrence of Chronic Bronchitis and Ventilatory Lung Efficiency.]** *Przeglad Lekarski* 38(4): 399-402, 1981, Polish.

The effects of exposure to organic solvents and smoking on the occurrence of symptoms of chronic bronchitis, lung function (FEV_1), and absence from work due to sickness were studied in 110 male workers in the painting department of the

Manufacturer of Transportation Equipment in Mielec, Poland. A questionnaire was administered to determine the symptoms, such as coughing, choking, and dyspnea. Anthropometric and spirometric measurements were also made. The results were compared with those obtained from a group of 420 office workers at the same plant. Among the "painters," 66.4 were smokers, while 58.3 percent of the office workers were smokers. The incidence of chronic bronchitis (CB) symptoms was 11 percent in "painters" vs. 8 percent in office workers. The CB incidence was 16.4 percent in smoking "painters" vs. 2.7 percent in nonsmokers and ex-smokers; 9.8 percent in smoking office workers vs. 5.1 percent in nonsmokers and ex-smokers. It is concluded that cigarette smoking presents an additional risk of CB in workers exposed to organic solvents at work. The measured FEV_1 values were practically identical in smokers and nonsmokers.

82–1017. Talseth, T.; Boye, N. P.; Kongerud, J.; Bredesen, J. E. **Aging, Cigarette Smoking and Oral Theophylline Requirement.** *European Journal of Clinical Pharmacology* 21(1): 33-37, 1981.

In a prospective study in 73 patients with obstructive pulmonary disease, aged 63.5±13.5 years (SD), it was found that theophylline dose, cigarette smoking, and age were all significant determinants of the steady-state trough plasma theophylline level during oral administration of the drug. As the predictive efficiency of the three factors combined amounted only to 25 percent, firm dosage recommendations cannot be made. Even among elderly patients, tobacco smokers had significantly lower plasma concentrations of theophylline. It is suggested that to obtain trough plasma concentrations of 50 μmol/l (approximately 9 μg/ml), a nonsmoking 50-year-old patient would require 9.8 mg/kg/day of oral theophylline, the dose increasing to 14.2 mg/kg/day in smokers of the same age. These doses should probably be reduced by 15-20 percent in 75-year-old patients. (Auth. Abs.)

82–1018. Talseth, T.; Kornstad, S.; Boye, N. P.; Bredesen, J. E. **Individualization of Oral Theophylline Dosage in Elderly Patients.** *Acta Medica Scandinavica* 210(6): 489-492, 1981.

Smoking was found to be a factor associated with a higher than average theophylline requirement in a prospective evaluation of a dosing nomogram for oral theophylline in 69 middle-aged and elderly patients (mean age 58.5) with obstructive lung disease. Of these, 23 were classified as smokers (smoked daily for 1 year and had not refrained from smoking during the preceding 3 months). The main features of the nomogram were that the theophylline dose was increased in middle-aged as compared with old patients, and that smokers were always prescribed a higher dose than nonsmokers of a similar age. The target theophylline concentration is presently defined as a trough level at steady state of 50 μmol/l. Approximately 50 percent of the patients achieved theophylline levels within ±20 percent of the target concentration, and none had theophylline levels exceeding 110 μmol/l. More smokers (57 percent) than nonsmokers (46 percent) missed this range. Smokers were also overrepresented among the extreme outliers (patients having trough levels outside the 25-75 μmol/l range). It was concluded that smoking is a source of considerable variability in dose requirement. Therefore, standardized doses, 12.4-19.2 mg/kg/day, may be appropriate only for a smoking middle-aged person. A 75-year-old nonsmoker should probably receive about 7.5 mg/kg/day. (Auth. Abs. Mod.)

82–1019. Tennant, F. S., Jr. **Histopathologic and Clinical Abnormalities of the Respiratory System in Chronic Hashish Smokers.** *Substance and Alcohol Actions/Misuse* 1: 93-100, 1980.

Results of a respiratory study suggest that hashish smoking and cigarette smoking together were more harmful than either habit separately. The investigation involved 30 young males with respiratory symptoms who were chronic and heavy smokers of hashish; 23 of them also smoked cigarettes. There were also six young male controls who did not use hashish, of whom three were cigarette smokers. A variety of respiratory evaluations were conducted. The abnormal histopathologic lesions found in hashish smokers were identical to those associated with later development of emphysema and carcinoma of the lung.

82–1020. Terr, A. I. **Respiratory Symptoms in Children Whose Parents Smoke.** *Western Journal of Medicine* 135(1): 47-48, July 1981.

In the diagnosis of chronic coughing in infants and persistent wheezing in children, parental smoking must be considered to avoid possible misdiagnosis. Survey findings from the United States, Great Britain, and Israel indicate that respiratory symptoms occur most commonly if both parents smoke, less commonly if one parent smokes, and least often if there is no smoking in the household. In families with one smoker, only smoking by the mother produced a significant effect on the child. Symptoms were directly correlated with the number of cigarettes smoked by the mother as well as with maternal respiratory symptoms, but not with birth weight, birth order, social class, or family size.

82–1021. Wallace, J. M.; Moser, K. M.; Hartman, M. T.; Ashburn, W. L. **Patterns of Pulmonary Perfusion Scans in Normal Subjects. II. The Prevalence of Abnormal Scans in Young Smokers.** *American Review of Respiratory Disease* 125(4): 465-467, April 1982.

Abnormal pulmonary perfusion (Q) scan defects, particularly lobar or segmental defects, are rarely present in normal young nonsmokers. To determine if normal smokers of the same age group have a higher prevalence of Q defects, six-view Q scans were performed in 40 subjects, 18 to 29 years of age, free of cardiopulmonary disease, and active smokers with a smoking history the equivalent of at least 20 cigarettes/day for 1 year.

None of the 40 subjects were found to have an abnormal Q scan defined as the presence of a lobar, segmental, or subsegmental defect on two views. When these data were compared with a previous study of nonsmokers, statistical analysis found no difference in the prevalence of abnormal Q scans in the two groups. It is concluded that among young normal persons, abnormal Q scans are extremely uncommon and the prevalence of significant Q defects is not influenced by smoking. (Auth. Abs. Mod.)

82–1022. Watson, C. B. **Predicting Pulmonary Risk.** *Ear, Nose, and Throat Journal* 61(3): 39-61, March 1982.

The effects of anesthesia and surgery on lung function, preoperative evaluation, and postoperative management procedures are discussed with regard to age, sex, length of surgery, special emergency procedures, specific anesthetic agents, active infection, smoking, heart disease, obesity, and other factors. How to deal with an otherwise healthy patient who is a heavy smoker remains controversial, as such patients frequently exhibit no impairment other than abnormalities of the small airways. Excessive secretions are well known to correlate with smoking, but such patients usually manage well. However, roentgenographic or bronchospirometric abnormality is more common in heavy smokers than in the general population and places the patient in a higher risk category. It is recommended that, prior to surgery, these tests be routinely performed and evaluated for this group. Patients should be made aware that cessation of smoking correlates with a better outcome.

See also, **82–0952, 82–0958, 82–0963, 82–1037, 82–1064**

CARDIOVASCULAR DISEASES

82–1023. Baird, R. N. **Diagnosis and Monitoring in Arterial Surgery: The Use of Ultrasound and Related Techniques.** *Annals of the Royal College of Surgeons of England* 64(2): 91-95, March 1982.

Ultrasound, plethysmographic, and related techniques have made a major contribution to the development of vascular surgery. In particular, their use has resulted in more precise assessment of the extent of arterial disease in smokers, i.e., the effect of smoking on the role of the distensible arterial wall in propagating pulsatile arterial flow for optimal perfusion of the extremities. Habitual smokers constitute almost half of the adult population of whom 2-4 percent will develop symptomatic ischemia. This subgroup seems to have an exaggerated vascular response to the inhalation of tobacco smoke. Recent advances in ultrasound techniques have identified arterial wall stiffening in the early stages of atherosclerosis in this group. Femoral artery Doppler signals can assess hemodynamic effects of incomplete stenosis in iliac arteries. After arterial reconstruction, these new techniques have improved the monitoring of the results of treatment. It has been shown with a Doppler probe that high-flow aorto-femoral grafts seldom give trouble unless the patient persists in smoking, but grafts with lower flow rates (axillofemoral, femoropopliteal, etc.) are at risk from late closure.

82–1024. Bornemisza, P.; Major, G.; Suciu, I.; Major, I. **Cigarette Smoking as a Risk Factor for Atherosclerosis. Study of the Biologic Changes Consecutive to Smoking in Normals, in Patients With Coronary Heart Disease and in Diabetics.** *Revue Roumaine de Medecine, Medecine Interne* 19(3): 251-258, 1981.

The atherogenic action of smoking was studied in 112 male subjects with a mean age of 48.6 years, including 40 healthy controls, 40 patients with compensated coronary heart disease, and 32 diabetics without ketoacidosis. Sixty-two were chronic smokers consuming 19.4 cigarettes/day; the remaining 50 did not smoke. The investigations performed in all the subjects were: erythrocyte count, hemoglobin and hematocrit determination, leukocyte absolute and differential counts, coagulation time (Howell), fibrinogen assay, platelet count and aggregability, total lipids, triglycerides, uric acid, creatininemia, and blood glucose determinations. In the group of smokers, these investigations were repeated 60 minutes after the smoking of two cigarettes. Comparison of the results obtained in smokers and nonsmokers, as well as before and after smoking, suggests that the influence of smoking upon the serum lipoprotein fractions is of little importance in atherogenesis, while its action upon the coagulation factors and the blood glucose level seems to have a real atherogenic role. (Auth. Abs.)

82–1025. Bottiger, L. E.; Carlson, L. A. **Mortality and Entry Characteristics ("Risk Factors") in the Stockholm Prospective Study.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Stockholm, Almqvist and Wiksell International, 1981, pp. 89-120.

In a study of the relationship between ischemic heart disease and triglycerides, smoking was also found to be a significant risk factor for myocardial infarction, occurring at a rate three times higher in smokers than in nonsmokers. Since 1961, the Stockholm Prospective Study has examined (yearly) approximately 6,500 individuals (55 percent men, 45 percent women) at a health survey center. While statistical analysis of deaths in men of all ages from neoplastic diseases shows the expected strong influence of smoking, a positive correlation remains even when cases of lung cancer are removed. Over 14 years, the deaths due to ischemic heart disease totaled 255/1,000 for men with four risk factors, while there were only 156/1,000 deaths due to neoplastic disease and 120/1,000 for all other causes. The rate of death from ischemic vascular disease increases in an almost linear way with the presence of an increasing number of risk factors; however, no such increase is apparent in the other two groups, indicating that the risk factors commonly discussed in connection with ischemic disease (age, smoking, systolic blood pressure, erythrocyte sedimentation rate, and hemoglobin), should in fact be considered as specific for that condition. The number of deaths among women in the study were much lower, although the age distribution was similar; i.e., 88 percent men compared to 12 percent women for deaths from myocardial infarction. The differences can be partly explained by the well known postponement of ischemic disease in women, although such a postponement does not seem to apply to other causes of death—neoplasia and "other," the time curves being identical in men and women. Risk factors for ischemic vascular disease in women, where hemoglobin and erythrocyte sedimentation rate are not significant, are narrowed to age, smoking, triglycerides, and systolic blood pressure. The less significant effect of smoking in women is attributed to their smoking much less than men (at the time of the study).

82–1026. Chesebro, J. H.; Fuster, V.; Elveback, L. R.; Frye, R. L. **Strong Family History and Cigarette Smoking as Risk Factors of Coronary Artery Disease in Young Adults.** *British Heart Journal* 47(1): 78-83, January 1982.

A study of 435 consecutive coronary patients age 50 or less, who had coronary arteriography, clearly demonstrated the association between strong family history, cigarette smoking, and serum cholesterol risk factors. There were 335 patients with coronary artery disease and 100 with normal coronary arteries. Risk factors reviewed were "packet-years" of cigarette smoking, family history of coronary disease in first-degree relatives 50 years of age or less, age- and sex-corrected serum cholesterol and triglycerides, hypertension, and diabetes. By univariate analysis, each risk factor except hypertension and diabetes was significantly more frequent in patients with coronary disease than in those without. By multivariate analysis of all risk factors in patients with and without coronary disease, the male or female patient with coronary disease could best be identified by the strong family history, cigarette smoking history, and age- and sex-corrected serum cholesterol. The percentage of patients with coronary disease when the three risk factors were present was 95 percent, two factors present 88 percent, one factor present 67 percent, none of the three 25 percent, strong family history alone 90 percent, smoking alone 66 percent, and serum cholesterol >80th centile alone 52 percent. (Auth. Abs. Mod.)

82–1027. Davis, J. W.; Davis, R. F.; Hassanein, K. M. **In Healthy Habitual Smokers Acetylsalicylic Acid Abolishes the Effects of Tobacco Smoke on the Platelet Aggregate Ratio.** *Canadian Medical Association Journal* 126(6): 637-639, March 15, 1982.

Acetylsalicylic acid (ASA) was found to reduce the effects of tobacco smoke on the platelet aggregate ratio in this study of 20 healthy habitual smokers. After abstinence for at least 8 hours, the subjects smoked two unfiltered cigarettes during each of two 20-minute periods separated by 48 hours. They had taken one 0.32-g tablet of ASA the night before the second period. The mean platelet aggregate ratios in venous blood taken immediately before and after each period of smoking were 0.79 and 0.70, respectively, when ASA had not been taken beforehand and 0.89 and 0.91 when it had. The mean after smoking was significantly higher when ASA had been taken beforehand. In conjunction with the previous finding that in nonsmokers ASA prevented a lowering of the platelet aggregate ratio by experimental smoking without affecting the ratio before smoking, the data from the present study suggest that ASA abolishes both acute and longer-lasting effects of tobacco smoke on the platelet aggregate ratio in healthy habitual smokers. (Auth. Abs. Mod.)

82–1028. Ducimetiere, P.; Richard, J. L.; Pequignot, G.; Warnet, J. M. **Varicose Veins: A Risk Factor for Atherosclerotic Disease in Middle-Aged Men?** *International Journal of Epidemiology* 10(4): 329-335, December 1981.

In a study of the factors associated with the presence of varicose veins, higher values of diastolic blood pressure, body mass index and number of cigarettes were found among men with varicosities. The problem of varicose veins was studied among 7,432 Paris policemen, aged 42-53 at entry (Paris Prospective Study). The prevalence of varicose veins was higher among lower social class subjects and was positively associated with cigarette consumption, a body-mass index, and diastolic blood pressure. The relationship found between higher cigarette consumption and varicosities may be of value because both cigarette smoking and the presence of varicose veins have been shown to be associated with decreased blood fibrinolytic activity. During followup for subsequent atherosclerotic disease (average 6.6 years), men with varicose veins were at a significantly higher risk for intermittent claudication and, at least in the lower social class, for hard coronary heart disease. No association was found with angina pectoris incidence. (Auth. Abs. Mod.)

82–1029. Hegarty, K. M.; Turgiss, L. E.; Mulligan, J. J.; Cluette, J. E.; Kew, R. R.; Stack, D. J.; Hojnacki, J. L. **Effect of Cigarette Smoking on High Density Lipoprotein Phospholipids.** *Biochemical and Biophysical Research Communications* 104(1): 212-219, January 15, 1982.

The effect of acute inhalation of cigarette smoke on high density lipoprotein (HDL) phospholipid composition in White

Carneau pigeons was examined. Four treatments included: (1) shelf control birds fed a chow diet and retained in their cages; (2) sham pigeons fed a cholesterol-saturated fat diet and exposed to fresh air by a smoking machine; (3) low nicotine—low carbon monoxide (LoLo) animals also fed the cholesterol diet and exposed to low concentrations of these cigarette smoke products; and (4) high nicotine—high carbon monoxide (HiHi) birds fed the cholesterol diet and subjected to high concentrations of these inhalants. The cholesterol—fat diet caused an increase in the concentration of most HDL phospholipid classes. Exposure to the HiHi regimen resulted in an increase in the HDL cholesterol/phospholipid ratio and a reduction in the concentration of HDL phosphatidyl ethanolamine, phosphatidyl serine/inositol, sphingomyelin and lysophosphatidyl choline. Cigarette smoking may thus attenuate HDL's anti-atherogenic properties by altering surface phospholipid components. (Auth. Abs.)

82–1030. Hodek-Demarin, V. **Doppler Velocity Measurements in Different Risk Factors in the Prevention of Stroke.** In: Kurjak, A.; Kratochwil, A. (Editors). *Recent Advances in Ultrasound Diagnosis 3.* Excerpta Medica International Congress Series 553, 1981, pp. 470-474.

In a study of stroke risk factors, heavy smokers showed a diminished cranial blood flow velocity (BFV) compared to nonsmoking controls of the same age. In examining the influence of various risk factors, and the validity of Doppler sonography in stroke detection and prevention, smokers consuming more than 20 cigarettes/day for at least 10 years were found to have a mean BFV value of 0.132, while the nonsmokers had a mean value of 0.169. Other risk factors discussed include age, hypertension, glucose tolerance, cardiac impairment, increased blood lipids, alcohol consumption, and oral contraceptive use.

82–1031. Kukita, H.; Imamura, Y.; Hamada, M.; Joh, T.; Kokubu, T. **Plasma Lipids and Lipoproteins in Japanese Male Patients With Coronary Artery Disease and in Their Relatives.** *Atherosclerosis* 42(1): 21-29, March 1982.

Plasma cholesterol (CH), triglyceride (TG), and high density lipoprotein cholesterol (HDL-C) were measured in 92 consecutive Japanese male subjects undergoing diagnostic coronary cineangiography. Interviews concerning alcohol and cigarette consumption were also conducted. Smoking history was defined as having smoked more than 10 cigarettes per day for 5 years. Sixty-nine of the patients were classified as having coronary artery disease (CAD), the remaining 23 subjects were classified as having normal coronary arteries (NCA). Clinical profile comparisons of CAD, NCA, and control groups showed a higher prevalence of smoking history and obesity in the CAD group than the NCA and the control groups, though there were no significant differences in the prevalences of alcohol intake, smoking history, and obesity between any of the age- and sex-matched groups. Obesity and cigarette smoking are associated with decreased HDL-C levels, and alcohol intake with increased HDL-C levels. The low HDL-C levels in CAD patients were due partly to these environmental factors. First-degree relatives of the CAD patients were also investigated. The male blood relatives of the CAD patients also had significantly lower HDL-C and higher TG levels than the nonblood male relatives and healthy control males. The female blood relatives, however, showed no significant differences from the nonblood female relatives and the healthy control females in plasma CH, TG, and HDL-C levels. These results suggest that low HDL-C and hypertriglyceridemia are the prevalent coronary risk factors, rather than hypercholesterolemia, in a population with a low fat intake such as the Japanese, and that these lipid abnormalities are related to sex and genetic factors. (Auth. Abs. Mod.)

82–1032. Levi, R.; Zavecz, J. H.; Burke, J. A.; Becker, C. G. **Cardiac and Pulmonary Anaphylaxis in Guinea Pigs and Rabbits Induced by Glycoprotein Isolated From Tobacco Leaves and Cigarette Smoke Condensate.** *American Journal of Pathology* 106(3): 318-325, March 1982.

The possibility that allergic reactions to tobacco constituents may initiate cardiac arrhythmia and sudden death in some smokers is suggested. Anaphylaxis was induced in the isolated hearts and lungs of rabbits and guinea pigs previously sensitized by immunizations with a tobacco glycoprotein antigen (TGP) and challenged with TGP isolated from either tobacco leaf or cigarette smoke condensate. Cardiac anaphylaxis was characterized by sinus tachycardia, decreased contractility, decreased coronary perfusion accompanied by hypoxic electrocardiographic changes, and a variety of rhythm disturbances, including idioventricular tachyarrhythmias. Allergic reactions may thus, in part, underlie the association between cigarette smoking and heart attack. (Auth. Abs. Mod.)

82–1033. Masarei, J.R.L.; Pyke, J. E.; Pyke, F. S. **Physical Fitness and Plasma HDL Cholesterol Concentrations in Male Business Executives.** *Atherosclerosis* 42(1): 77-83, March 1982.

Smokers had higher triglyceride levels than nonsmokers in this study of the relationships between several variables related to health and fitness, and the plasma lipid levels among middle-aged business executives. Endurance fitness was measured objectively (physical work capacity at pulse rate of 170/min, W_{170}), and related to a number of other physical characteristics and aspects of coronary risk status: smoking habit, FEV_1 (forced expiratory volume in first second), blood pressure, adiposity, alcohol consumption, plasma levels of total and non-high density lipoprotein cholesterol, triglyceride, and high density lipoprotein cholesterol (HDL-C). The primary question was whether HDL-C levels could be shown to be related to endurance fitness levels over the range encountered in a fairly homogeneous population and hence whether there could be value in terms of lipid coronary risk status in encouraging a moderate increase in physical activity. Even though the trend was toward an index of physical activity being able to separate the subjects in terms of HDL-C, this was not as clear-cut as the division in terms of endurance fitness. Alcohol and smoking were associated with higher triglyceride levels, but not with HDL-C. (Auth. Abs. Mod.)

82–1034. Meade, T. W. **Risk Associations in the Thrombotic Disorders.** *Clinics in Haematology* 10(2): 391-405, June 1981.

Smoking's effect on blood clotting factors, and the diseases associated with thrombosis are explored. Increasing age, white ethnic group, blood group other than O, cigarette smoking, oral contraceptive use, and diabetes all lead to elevated levels of one of the three factors. Diabetes and smoking are both characterized by an increased prevalence of vessel wall damage. Smoking appears to lower clotting factor VIII:C, has no effect on factor VII:C, and raises fibrinogen levels. Smoking was most strongly associated with ischemic heart disease at the youngest ages, and attenuated as age increased. Epidemiological evidence shows that smoking does not influence mortality from venous thrombosis, and possibly exerts a protective effect against venous thrombosis after myocardial infarction.

82–1035. Nakamura, S. **Influence of Exercise and Smoking on High Density Lipoprotein Cholesterol in University Students.** *Tohoku Journal of Experimental Medicine* 135(4): 443-444, December 1981.

Smoking habits and exercise were found to affect high density lipoprotein (HDL) cholesterol levels in a study of male university students with normal body weight and daily alcoholic intake of less than 22 g. Values of more than 60 mg/100 ml HDL cholesterol, measured by the precipitation method, were observed significantly more frequently in nonsmokers regardless of exercise habits, and in smokers who actively exercised than in smokers who did not exercise. Thus, the association of HDL cholesterol with exercise and smoking is already present in university students. (Auth. Abs. Mod.)

82–1036. Nash, J. D.; Farquhar, J. W. **Applications of Behavioral Medicine to Disease Prevention in a Total Community Setting: A Review of the Three Community Study.** In: Ferguson, J. M.; Taylor, C. B. (Editors). *The Comprehensive Handbook of Behavioral Medicine. Volume 3: Extended Applications and Issues.* Jamaica, New York, Spectrum Publications, Inc., 1980, pp. 313-335.

The Stanford Three Community Study for intervention in cardiovascular disease by lifestyle change is reviewed. Baseline studies were conducted in three semirural California communities; one then received both a mass media campaign and face-to-face intensive instruction and skills training, the second received only the media campaign, and the third community served as a control. Baseline values were uniform across the three communities. Results showed that after 2 years, both treatment communities showed significant positive effects on all variables except relative weight. At the end of the first year, the intensive instruction population showed greater overall reduction in cardiovascular disease risk, but by the end of the second year, the media-only community had caught up. Some variables, such as diet, were improved easily, but other changes, e.g., smoking habits, were achieved only through relatively cost- and effort-intensive means. The contribution of smoking to cardiovascular disease and the rationale behind the study are also discussed.

82–1037. Ohkubo, C. **Some Acute Cardiopulmonary Effects of Mainstream and Sidestream Cigarette Smoke in Man.** *Preventive Medicine* 11(2): 173-186, March 1982.

Acute physiological effects of alkaline and acid nicotine following transient inhalation of mainstream or sidestream cigarette smoke and nicotine aerosol were compared in two groups (smokers and nonsmokers) of seven healthy male students. Both groups were passively exposed for 2 sec to each preparation, and heart rate, blood pressure, finger pulse wave, forehead electromyogram, and respiratory movement were monitored. After inhalation of the preparations, decreases in the amplitude of finger pulse wave and increases in the amplitude of electromyogram were observed in both groups. The results showed that the irritation by sidestream smoke provokes the nasopharyngeal reflex more strongly and for longer periods than by mainstream smoke in an equivalent dose of nicotine for them and that this difference was mainly attributable to the percentage of nicotine presented as uncharged molecules according to pH. Although the equivalent dose of nicotine was applied, nicotine aerosols induced stronger and/or longer changes than cigarette smoke, which may be partly ascribed to the rather large diameter of nicotine aerosol particles. In addition, more irritant effects of smoke were observed on nonsmokers than on smokers. (Auth. Abs.)

82–1038. Olsson, J.-E. **Comparison Between Induced Platelet Aggregation and Circulating Platelet Aggregates as Platelet Function Tests in Patients With Transient Ischemic Attacks.** *Acta Neurologica Scandinavica* 65(2): 122-132, February 1982.

To help denote patients with transient ischemic attacks (TIA) who may be candidates for treatment with antiplatelet drugs to prevent ischemic stroke, the effects of smoking, sex, pregnancy, and oral contraceptive use on blood platelet function were studied. A total of 67 subjects (45 healthy controls, 10 nonstroke patients, and 12 TIA patients) were examined, before and after treatment with either anticoagulants (AC) or antiplatelet drugs (APD). Except for lower hemoglobin and hematocrit levels in women, sex, smoking, oral contraceptives, or pregnancy did not significantly influence the routine blood parameter. Smoking females taking oral contraceptives had an increased number of circulating platelet aggregates (CPA) and the most easily induced aggregation in vitro. Patients with TIA had no significant differences in blood or platelet function compared with the healthy controls (except smoking females on oral contraceptives) or the nonstroke patients, even though individual patients could have high numbers of CPA and an easily induced platelet aggregation in vitro. Treatment with AC did not influence platelet function, whereas APD therapy decreased the number of CPA and inhibited the secondary platelet aggregation in vitro. (Auth. Abs. Mod.)

82–1039. Plunkett, E. R. **Contraceptive Steroids, Age, and the Cardiovascular System.** *American Journal of Obstetrics and Gynecology* 142(6, part two): 747-751, March 15, 1982.

There is evidence that women who use oral contraceptives may be at slightly greater risk of cardiovascular complications as their age increases beyond 35 years. When combined with cigarette smoking, this relative risk more than triples. However, postmenopausal women taking natural estrogen alone or with minimal amounts of progestogen have not exhibited these increased risks when compared with untreated women. There is also evidence that relates progestogen dosage to morbidity rates from circulatory disease. Therefore the smallest dose of both estrogen and progestogen consistent with contraceptive efficacy should be prescribed, and patients should be advised to quit smoking. (Auth. Abs. Mod.)

82–1040. Pyorala, K.; Valkonen, T. **The High Ischaemic Heart Disease Mortality in Finland.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Stockholm, Almqvist and Wiksell International, 1981, pp. 37-61.

Ischemic heart disease (IHD) mortality in Finland is addressed under several headings, including comparisons with other nations, regional differences within Finland itself, mortality trends, and possible causes. Possible causes or risk factors for IHD include smoking, blood pressure, and dietary fats and serum cholesterol. There may also be genetic, environmental, socioeconomic, lifestyle, and health care risk factors. The high IHD mortality of Finns which particularly concerns working-age Finnish men reached its peak levels in the 1960s and started to decline in the 1970s. Decreasing trends in dairy fat consumption and cigarette smoking, and improving hypertension control commenced during the 1960s and seem to be continuing. These favorable changes may have a direct relationship to the decline in IHD mortality, but other factors may also be involved.

82–1041. Rafnsson, V.; Bengtsson, C. **Erythrocyte Sedimentation Rate and Cardiovascular Disease. Results from a Population Study of Women in Goteberg, Sweden.** *Atherosclerosis* 42(1): 97-108, March 1982.

In a population sample of women, the erythrocyte sedimentation rate (ESR) was studied in relation to the incidence of myocardial infarction, angina pectoris, stroke, and intermittent claudication. ESR could not be proved to be predictive of these manifestations of cardiovascular disease, and no association was found between smoking habits and ESR. Weak correlations were found between ESR and serum triglycerides, serum cholesterol, and serum uric acid, respectively. The ESR was higher in women with hyperlipidemia, in women with arterial hypertension, and in overweight women (women within the upper 10 centiles of weight index) compared with women of the same age in the population sample. No correlation between smoking and high hemoglobin values was found in this study, which could have masked an association between smoking and ESR. (Auth. Abs. Mod.)

82–1042. Salonen, J. T.; Puska, P.; Tuomilehto, J. **Physical Activity and Risk of Myocardial Infarction, Cerebral Stroke and Death.** *American Journal of Epidemiology* 115(4): 526-537, April 1982.

Serum cholesterol, tobacco consumption, and diastolic blood pressure had a notable association with the risk of both acute myocardial infarction and death among men in eastern Finland. Physical activity at work and in leisure time was studied by using a questionnaire in a random population sample from two counties of eastern Finland in 1972. Data on age, serum cholesterol, diastolic blood pressure, height, weight, and smoking were recorded in a field examination. The study population consisted of 3,978 men aged 30-59 years and 3,688 women aged 35-59 years. During the approximately 7-year followup, 210 men and 63 women had acute myocardial infarction (AMI), 89 men and 14 women died of acute ischemic heart disease, 71 men and 56 women had cerebral stroke, and 172 men and 75 women died of any disease. The subjects with a history of myocardial infarction, angina, or cerebral stroke in the 12 months preceding the baseline survey were excluded in the respective analysis. Low physical activity at work was associated with an increased risk of AMI, cerebral stroke, and death due to any disease in both men and women, even when controlling for age, serum total cholesterol, diastolic blood pressure, height, weight, and smoking in a multiple logistic model. The relative risk of AMI was 1.5 in men and 2.4 in women. The association of the lack of leisure time physical activity with the risk of acute myocardial infarction and cerebral stroke, based on the present data, was found to be mostly attributable to the confounding effect of smoking and other coronary risk factors. (Auth. Abs. Mod.)

82–1043. Shamarin, V. M.; Aleksandrov, A. A.; Glazunov, I. S.; Antonova, L. T. **Trekhletnyaya Dinamika Arterial'nogo Davleniya Podrostkov Odnogo iz Rayonov Moskvy. [Three-Year Survey of Arterial Blood Pressure Evolution in Adolescents of a Moscow District.]** *Terapevticheskiy Arkhiv* (5): 74-78, 1981, Russian.

Systolic and diastolic blood pressures, pulse rate, and anthropometric data were measured and smoking habits were surveyed over a 3-year period (1978-1980) in a sample of 242 students, boys and girls, born in 1964, and initially having an elevated arterial blood pressure. The study also examined 230 controls with initially normal blood pressure. The mean systolic pressure in boys and the mean diastolic pressure in both boys and girls increased significantly as age increased from 12-13 to 15-16 years. The survey indicated that 41.2 and 36.1 percent of the 15- to 16-year-old boys smoked more than three cigarettes/week among the population samples with initially elevated pressure and normal pressure, respectively. Both systolic and diastolic pressures were not significantly different in smoking and nonsmoking boys. The mean systolic pressure of 9th grade boys who smoked and who attended a 10-grade secondary school was significantly higher than in smoking boys in vocational and technical schools.

82–1044. Sieffert, G. F.; Keown, K.; Moore, W. S. **Pathologic Effect of Tobacco Smoke Inhalation on Arterial Intima.** *Surgical Forum* 32: 333-335, 1981.

The occurrence of major endothelial damage and associated focal platelet aggregation after smoke inhalation is demonstrated. Eleven Sprague-Dawley rats were exposed to tobacco smoke for 15-minute intervals 3 times/day producing 15 to 20 percent carboxyhemoglobin concentrations which persisted for at least 3 hours after return to fresh air Five rats were sacrificed after 6 weeks and 6 after 12 weeks. The thoracic aorta was perfusion-fixed and prepared for scanning electron microscopy. Elongation of endothelial cells, with uplifting from the basement membrane, areas of endothelial loss, pitting, crater formation, and white blood invasion were apparent. Platelet aggregation was on damaged intima as well as platelet adherence to normal-appearing intima. Quantitative studies revealed significant differences in white blood cell invasion ($p<0.004$), endothelial cell uplifting, and platelet aggregation ($p<0.04$) in the 12-week group. The findings are consistent with the hypothesis that tobacco smoke inhalation may have a causal relationship to the development of atherosclerosis.

82–1045. Simpson, W. W.; Priola, D. V.; Curtis, M. B. **Blockade of Nicotine Induced Cardiac Responses by Metoprolol in the *in Situ* Dog Heart.** *Proceedings of the Western Pharmacology Society* 24: 57-61, 1981.

An investigation of the possible antinicotine property of metoprolol found that the drug is able to exert a ganglionic blocking effect which inhibits the nicotine stimulation of acetylcholine (ACH) release from postganglionic nerve cells. Anesthesized dogs ($n=6$), placed on cardiopulmonary bypass, were injected with (1) bretylium to eliminate the nicotine-induced catecholamine release from adrenergic nerve fibers; (2) control nicotine (NIC) and ACH for initial responses; and (3) metoprolol, 4.0 to 6.0 mg, to evaluate the effect of NIC on the negative dromotropic and inotropic responses to intrinsic cardiac innervation. The effect noted should be considered when using a beta blocking agent in an experimental model involving ganglionic stimulation.

82–1046. Smirnova, I. P.; Kuklya, Yu. I. **Epidemiologicheskoye Issledovaniye Ishemicheskoy Bolezni Serdtsa i yeye Faktorov Riska v Neorganizovannoy Muzhskoy Populyatsii. [Epidemiological Study of Ischemic Heart Disease and Its Risk Factors in a Random Male Population.]** *Vrachebnoe Delo* (9): 31-33, September 1981, Russian.

A questionnaire including an inquiry about smoking habit was administered to a representative sample of 1,936 male residents of a Kiev district. In addition, the study program included anthropometric measurements, arterial blood pressure measurements, ECG recordings, and determinations of cholesterol and triglyceride contents. Ischemic heart disease (IHD) was detected in 19.4 percent of the subjects, and myocardial infarct in 2.7 percent. One out of every five subjects had arterial hypertension. About one-half of the population sample were regular smokers of at least one cigarette/day or ex-smokers who stopped smoking within 12 months preceding the study. IHD or at least one of its risk factors was detected in 85 percent of the total sample. Arterial hypertension, mostly systolic (equal to or higher than 160 mm Hg), was found to be the leading risk factor of IHD in the Kiev population. A correlation between IHD incidence and smoking was not confirmed.

82–1047. Tuomilehto, J.; Elo, J.; Nissinen, A. **Smoking Among Patients With Malignant Hypertension.** *British Medical Journal* 284(6322): 1086, April 10, 1982.

The association between smoking and malignant hypertension (MH) in patients of the North Karelia cardiovascular disease intervention project is examined. Of 56 patients, 13 of 24 males and 8 of 32 females were smokers. In both sexes, smoking was more common in the youngest and oldest age groups than in the middle-aged group. With the exception of MH patients, no correlation was found between smoking and blood pressure. Patients with MH tended to smoke more than those with less severe stages of hypertension, and seemed more likely to quit than normotensive smokers. A possible factor in smoking which leads to such complications as MH is cadmium exposure, which causes tubular dysfunctions.

82–1048. Ueshima, H.; Iida, M.; Shimamoto, T.; Konishi, M.; Tanigaki, M.; Nakanishi, N.; Takayama, Y.; Ozawa, H.; Kojima, S.; Komachi, Y. **High-Density Lipoprotein-Cholesterol Levels in Japan.** *Journal of the American Medical Association* 247(14): 1985-1987, April 9, 1982.

Results of a Japanese study suggest that levels of high-density lipoprotein (HDL)-cholesterol may be at least partly affected by environmental factors such as smoking. A survey of HDL-cholesterol levels of six Japanese population groups with different lifestyles (1,804 men and 1,561 women) found that the HDL-cholesterol levels of Japanese men aged 40 to 69 years were approximately 55 mg/dL, or about 10 mg/dL higher than those of American men. Smoking appears to be more popular and prevalent among Japanese men than among U.S. men. Differences in HDL-cholesterol levels in the Japanese population and between the populations of Japan and the United States may be at least partly due to smoking and other lifestyle factors such as alcohol consumption, exercise, and obesity. (Auth. Abs. Mod.)

CARDIOVASCULAR DISEASES

82–1049. Vessey, M. P. **Oral Contraceptives and Cardiovascular Disease: Some Questions and Answers.** *British Medical Journal* 284(6316): 615-616, February 27, 1982.

A review of the data from various epidemiological studies on oral contraception and cardiovascular disease reveals a consensus that fatal cardiovascular events are most likely when high-dose pills are used by older women, especially those who smoke or who have other risk factors. Some researchers have been unconvinced that the excess of deaths in women using the pill have been almost entirely attributable to smoking. An excess risk has been observed both in nonsmokers and in comparisons of users and nonusers of oral contraceptives who smoke similar numbers of cigarettes a day. Nonetheless, the risk of a fatal arterial event attributable to use of the pill is heavily concentrated in smokers, even though in venous thromboembolism smoking does not appear to be a factor.

82–1050. Wilson, P. W.; Garrison, R. J.; Castelli, W. P.; Feinleib, M.; McNamara, P. M.; Kannel, W. B. **Prevalence of Coronary Heart Disease in the Framingham Offspring Study: Role of Lipoprotein Cholesterols.** *American Journal of Cardiology* 46(4): 649-654, October 1980.

Of 1,312 men aged 35 to 54 years in the Framingham Offspring Study, 43 had clinically recognized coronary heart disease (CHD) at the initial examination, 26 of whom had previously had a myocardial infarction. Of 1,296 women in the same age range, only 11 had coronary disease and 3 of these had a prior myocardial infarction. The prevalence of CHD in men was strongly associated with age, smoking, high density lipoprotein (HDL), low density lipoprotein (LDL), and total cholesterol. When multivariate logistic regression analysis was used, these factors retained their significant association. The total cholesterol/HDL cholesterol ratio was also strongly associated with CHD in the multivariate analysis. It is concluded that both HDL and LDL cholesterol are strongly and independently associated with CHD prevalence, whereas the level of very low density lipoprotein cholesterol makes no significant independent contribution. (Auth. Abs. Mod.)

See also, **82–0954, 82–0957, 82–0986, 82–1011, 82–1076, 82–1126**

PREGNANCY AND INFANT HEALTH

82–1051. Bureau, M. A.; Monette, J.; Shapcott, D.; Pare, C.; Mathieu, J.-L.; Lippe, J.; Blovin, D.; Berthiaume, Y.; Begin, R. **Carboxyhemoglobin Concentration in Fetal Cord Blood and in Blood of Mothers Who Smoked During Labor.** *Pediatrics* 69(3): 371-373, March 1982.

A study of carboxyhemoglobin (HbCO) levels in mothers who smoked during pregnancy and labor (n = 134) and in the cord blood of their newborns found the HbCO levels in cord blood to be much higher than those in maternal blood. Information was gathered from the women about their age, smoking habits, and HbCO levels on arrival at the hospital and during delivery. Newborns were also measured for cord blood levels of HbCO at birth. A significant correlation was discovered between daily cigarette consumption and fetal HbCO and maternal levels measured at admission and at delivery. Maternal smoking produced a much higher HbCO concentration in the fetus than in the mother.

82–1052. Heinrichs, W. L. **Environment and Reproduction.** In: Semm, K.; Mettler, L. (Editors). *Human Reproduction*, Excerpta Medica International Congress Series 551, 1981, pp. 269-277.

The teratogenic effects on reproduction of many potentially harmful agents in the environment are explored. These substances include radiation, sound, chemicals (drugs, pesticides, smoking), malnutrition, and other physiological disturbances. The severity of such a public health risk is proportional to the specificity of the agent, the dose of exposure, the distribution and route of exposure, and the time of exposure in relation to reproductive events. Those factors specifically influencing reproductive function involve the individual's state of somatic development, reproductive capacity, and potential in reproductive performance. For instance, the effect of cigarette smoking on reducing fetal weight influences the growth potential until adolescence, and can be permanent. However, when onset of smoking occurs after puberty, somatic development may not be affected, but reproductive senescence (menopause) is advanced. Potential damage from environmental agents is discussed in terms of the mechanisms of reproductive toxicity, certain reproductive processes subject to chemical injury, and adverse effects on reproductive capacity and performance.

82–1053. Jouppila, P.; Kirkinen, P.; Eik-Nes, S.; Koivula, A. **Fetal and Intervillous Blood Flow Measurements in Late Pregnancy.** In: Kurjak, A.; Kratochwil, A. (Editors). *Recent Advances in Ultrasound Diagnosis 3.* Excerpta Medica International Congress Series 553, 1981, pp. 226-233.

Although placental circulation decreased after the maternal smoking of one cigarette, fetal blood flow was affected only minimally. Data from Doppler ultrasound and real-time ultrasonography, performed on the umbilical vein and fetal thoracic descending aorta, were reviewed in 54 cases in which the newborn was under the 10th weight percentile. Both maternal and fetal pulse rates increased after one cigarette, though the immediate effect on fetal blood flow was slight. The circulation adaptation mechanism in the fetus seems able to tolerate the decrease in the placental circulation of smoking mothers.

82-1054. Kline, J.; Levin, B.; Stein, Z.; Susser, M.; Warburton, D. **Epidemiologic Detection of Low Dose Effects on the Developing Fetus.** *Environmental Health Perspectives* 42: 119-126, December 1981.

The adverse effects of smoking and drinking exposures on fetal health are discussed, including data which suggest a dose—response relationship between daily cigarette consumption and both spontaneous abortion and decreased birth weight. A dose—response relationship was also found between drinking and spontaneous abortion, but not for drinking and birth weight. There appeared to be no interaction between smoking and drinking for spontaneous abortion. Also mentioned are questions that occur in trying to determine "safe" cigarette and alcohol consumption levels during pregnancy.

82-1055. Moore, L. G.; Rounds, S. S.; Jahnigen, D.; Grover, R. F.; Reeves, J. T. **Infant Birth Weight Is Related to Maternal Arterial Oxygenation at High Altitude.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 52(3): 695-699, March 1982.

Maternal characteristics acting to lower arterial oxygen content may contribute to smaller infant birth weight at high altitudes. Arterial oxygenation was measured serially during pregnancy and again postpartum in 44 (35 nonsmokers, 9 smokers) residents of Leadville, Colorado (elevation 3,100 meters). Three maternal characteristics identified as being related to birth weight were: ventilation, hemoglobin concentration, and smoking habits. Mothers of smaller babies (<2,900 grams) compared with mothers of larger babies (>3,500 grams) were characterized by hypoventilation, no change or a decrease in ventilation and arterial O_2 saturation from early to late gestation, and a falling hemoglobin concentration that combined to lower arterial O_2 content in the third trimester. Maternal smoking at 3,100 meters was associated with a twofold to threefold greater reduction in infant birth weight (-546 grams) than reported from sea level. Thus, maternal arterial oxygenation during pregnancy may be important for predicting fetal growth retardation and the process of adaptation to high altitude. (Auth. Abs. Mod.)

82-1056. Rayburn, W. F.; Motley, M. E.; Zuspan, F. P. **Conditions Affecting Nonstress Test Results.** *Obstetrics and Gynecology* 59(4): 490-493, April 1982.

Smoking, maternal fasting, amniocentesis, medication, time of day, caffeine, and age of mother were examined in relation to false nonreactive results in nonstress tests. During a 16-month period, nonstress tests (NSTs) were performed in 315 pregnancies; 64 (12 percent) of the initial 534 satisfactory tests had nonreactive patterns. Approximately one-third (22 of 64, 34 percent) of these nonreactive tests either remained nonreactive or were followed by a positive or suspicious contraction stress test that same day. When compared with pregnancies that gave reactive test results, a nonreactive pattern was not associated with maternal obesity, fasting, certain prescribed medications (methyldopa, phenobarbital, insulin), time of day, or recent amniocentesis. A cigarette had been smoked prior to 108 (20 percent) of the tests. Risk of a nonreactive test was 18.5 percent after smoking compared to 10.3 percent if the patient had not smoked, which may explain a falsely nonreactive result. Testing during a fetal rest or quiet period could also give such results. (Auth. Abs. Mod.)

82-1057. Shannon, D. C.; Kelly, D. H. **SIDs and Near-SIDs. (First of Two Parts).** *New England Journal of Medicine* 306(16): 959-965, April 22, 1982.

In a review of theories on the causes of sudden infant death (SID), it is noted that maternal smoking during pregnancy is related to SID incidence. Risk of SID is also increased if the mother is less than 20 years of age, unmarried and poor, delayed or failed to seek prenatal care, was ill during pregnancy or had a short interval between pregnancies, had a previous fetal loss, or abused narcotics. Smoking may exert an effect on placental arteries through carbon monoxide, or via the factor of inadequate weight for gestational age. The nature of SID is also discussed with respect to pathology, infection and immunity, chemistry and toxicology, maternal epidemiology, genetics, and cardiovascular factors.

82-1058. Sheveleva, G. A.; Kiryushchenkov, A. P. **Vliyaniye Nikotina na Razvitiye Ploda i Potomstva. [Effect of Nicotine on Development of the Fetus and Offspring.]** *Akusherstvo i Ginekologiya* (6): 31-34, 1981, Russian.

Nicotine extract from tobacco was administered subcutaneously to 150 pregnant rats in doses of 0.01, 0.05, and 5 mg/kg at various stages of fetus development or during the whole pregnancy. The aftereffects of nicotine were studied at various ontogenic stages. Nicotine administration at a 5 mg/kg dose during preimplantation and implantation periods caused an increase in embryonic mortality. Nicotine at a 0.05 mg/kg dose during the same gestation periods caused a decrease in placental mass and placentary coefficient. At a 0.01 mg/kg dose the fetal mass decreased. Hemorrhages, subcutaneous and in the body cavity; subcutaneous dropsy; and hydronephrosis were observed in all fetuses in varying degrees, depending on the nicotine dose. A smaller increase in body weight of the offspring (compared to controls), and various functional changes were observed in up to 2-month-old offspring of rats given 0.05 or 5 mg/kg nicotine during the whole pregnancy. Also, the respiration rate was increased at these dose levels. Thus, the dependence of the embryotropic effect of nicotine on its dose was established. The dose of 0.01 mg/kg was found to be near the threshold of embryotoxic effect and the effect on offspring development.

82–1059. Vahakangas, K.; Hirn, M.; Pelkonen, O. **Effect of Maternal Cigarette Smoke Exposure on Foetuses of Inbred Strains of Mice Responsive and Non-Responsive to Polycyclic Aromatic Hydrocarbons.** *Toxicology Letters* 10(1): 81-86, January 1982.

Although cigarette smoke exposure resulted in reduced fetal weight among pregnant rats, responsiveness to polycyclic aromatic hydrocarbons (PAH) was not the main factor. To evaluate the role of genetically controlled responsiveness to PAH in the effect of cigarette smoke on birth weight, pregnant mice were exposed to cigarette smoke from day 1 to day 17 of pregnancy, and the offspring studied on day 18. An unequivocal phenotyping of fetuses with respect to responsiveness was achieved by measuring the activity of aryl hydrocarbon hydroxylase (AHH) in the fetal liver and corresponding placenta after pretreatment of the dam with β-naphthoflavone. The fetuses of dams exposed to standard nonfilter cigarettes were consistently smaller than those of the controls, whereas smoke from commercial filter cigarettes was only marginally effective. No statistically significant association with the responsiveness to PAH could be found with respect to this effect on weight. These results suggest that genetically determined responsiveness to PAH, although of importance for PAH fetotoxicity in mice, is not important in the effect of cigarette smoke on birth weight in the strains studied. Other chemicals present besides PAH may have been responsible for the weight reduction. (Auth. Abs. Mod.)

OTHER DISEASES AND CONDITIONS

82–1060. Andreasson, L.; Bjorlin, G.; Korsgaard, R.; Mattiasson, I.; Trell, E.; Trell, L. **Leucoplakia of the Oral Cavity, Smoking and Arylhydrocarbon-Hydroxylase Inducibility.** *Postgraduate Medical Journal* 58(677): 138-141, March 1982.

Study results suggest a possible association between smoking, oral leukoplakia, and high aryl hydrocarbon hydroxylase (AHH) inducibility. In a consecutive series of 53 cases of oral leukoplakia, smoking history, oro-dental status, and AHH inducibility were assessed. There was an increased association of leukoplakia with smoking, particularly in the male cases, but no remarkable findings as to dental status. Overall, there was a slightly higher frequency of cases with high AHH inducibility than expected from a normal control population. This difference was confined to the smokers, where there was a statistically highly significant overrepresentation of high AHH inducibility and underrepresentation of low AHH inducibility. The figures in the nonsmoking patients were close to the expected. (Auth. Abs. Mod.)

82–1061. Aoyama, M.; Fujise, H.; Tachi, N. **Experimental Studies on the Effect of Cigarette Smoke on the Physiological Growth, Life-Span, and Spermatogenesis in Male Mice.** *Nagoya Medical Journal* 25(4): 259-264, June 1981.

The adverse effects of long-term exposure to cigarette smoke on the cardiovascular, respiratory, and hormonal systems of male mice are described. Forty-five male mice weighing 20 g were divided into three groups: group A, group B, and controls. Group A was exposed for 50 minutes a day, and group B for 25 minutes, in a chamber containing cigarette smoke. The survival rate of the mice exposed to smoke for 360 consecutive days was far less (group A, 13.3 percent, and group B, 20 percent) than that of the control mice (53.3 percent). The group which was exposed for a longer time each day (50 minutes) tended to have a shorter lifespan than the group exposed for a shorter time (25 minutes). Physiological increase in the body weight of the exposed mice was considerably more retarded than that of the control mice. Microscopic observation of the testes after 360 days of smoke exposure revealed oligo- or azoospermia. (Auth. Abs. Mod.)

82–1062. Aziz, S.; El-Gengehy, M. T.; Othman, A.; El Hofy, O. A.; Aly, M. A.; Said, E. **Effect of Smoking on Diabetic Retinopathy.** *Journal of the Egyptian Medical Association* 61(11-12): 743-750, 1978.

In a study of diabetics, smoking was found to be a risk factor for proliferative retinopathy. The effect of smoking on the fundus was studied in 138 diabetic patients. The smoking habit in diabetics was more common in the middle and old age group than in the younger age group. The incidence of proliferative retinopathy rose with increased tobacco consumption. No association was found between the duration of diabetes mellitus with proliferative retinopathy in nonsmokers, in contrast to smokers, where a temporal association was evident. There was no correlation between the degree of retinopathy and smoking in patients with < 20 years duration of diabetes, but there was a correlation in cases of patients who had the disease longer than 20 years. There was no relationship between the age of patients and the occurrence of proliferative retinopathy. It is concluded that tobacco and the resultant hypoxia aggravate the microangiopathy and accelerate the damage of the already damaged diabetic capillaries. Smoking should be prohibited in cases of patients with diabetes mellitus to prevent proliferative retinopathy and consequent blindness. (Auth. Abs. Mod.)

82–1063. Burgess, E. M.; Matsen, F. A., III.; Wyss, C. R.; Simmons, C. W. **Segmental Transcutaneous Measurements of Po_2 in Patients Requiring Below-the-Knee Amputation for Peripheral Vascular Insufficiency.** *Journal of Bone and Joint Surgery* 64-A(3): 378-382, March 1982.

In estimating the healing potential of patients requiring amputation for peripheral vascular insufficiency, only two factors were found to be significant: the incidence of smoking, and the segmental transcutaneous Po_2 values. Fifteen patients with below-the-knee transcutaneous Po_2 values of 40 millimeters of mercury or more had no delay in healing of the below-the-knee amputation. Seventeen of nineteen patients with values of more than zero but less than 48 millimeters of mercury had healing at the below-the-knee level; two healed after local revision. The three patients who had below-the-knee values of zero required reamputation above the knee. Six of the seven patients with a failed below-the-knee amputation were smokers, whereas only half of the patients with healed distal amputations were smokers. (Auth. Abs. Mod.)

82–1064. Gouveia, J.; Hercend, T.; Lemaigre, G.; Mathe, G.; Gros, F.; Santelli, G.; Homasson, J. P.; Angebault, M.; Lededente, A.; Parrot, R.; Gaillard, J. P.; Bonniot, J. P.; Marsac, J.; Pretet, S. **Degree of Bronchial Metaplasia in Heavy Smokers and Its Regression After Treatment With a Retinoid.** *Lancet* 1(8274): 710-712, March 27, 1982.

Bronchoscopy and bronchial biopsy were used to evaluate metaplasia in 67 heavy smokers (55 men and 12 women, 29 to 71 years of age), and to assess the effects of treatment with the retinoid, etretinate. The index of metaplasia (IM) was found to be >15 percent in 34 subjects and <15 percent in 33. No significant association between HLA phenotypes and IM was found because of the small number of subjects. The subjects with an IM of >15 percent were treated for 6 months with 25 mg/day of retinoid. Treatment consisted of clinical examination, liver function and serum creatinine tests, and electrocardiograms before the start of the trial, and at 1 month, 3 months, and 6 months of treatment. At 6 months a second bronchoscopic examination was done with further biopsy assessments. Twelve subjects completed the course with IM lower in ten and unchanged in one. The difference between the mean values of the IM before and after treatment in the 11 subjects was found to be significant ($p<0.01$).

82–1065. Harries, A. D.; Baird, A.; Rhodes, J. **Non-Smoking: A Feature of Ulcerative Colitis.** *British Medical Journal* 284(6317): 706, March 6, 1982.

Smoking was uncommon among a study group of ulcerative colitis patients. Questionnaires were used to gather information about smoking habits from patients with ulcerative colitis ($n=230$), Crohn's disease ($n=192$), and from a control group ($n=230$) matched for age and sex with the ulcerative colitis patients. Subjects were classified as current smokers, ex-smokers, and occasional or nonsmokers. Current cigarette smokers comprised 8 percent of the ulcerative colitis patients, compared to 42 percent among the Crohn's disease group and 44 percent of the controls. The low smoking rate among the ulcerative colitis patients was partly due to a larger proportion never having smoked, as well as a similar proportion having quit smoking, usually before bowel symptoms began. The cause of the different results among the groups was unclear, although it was suggested that ulcerative colitis patients might be more affected by health education.

82–1066. Hodges, M. E. **Time To Give Up Smokers?** *Bibra Bulletin* pp. 359-364, September 1981.

A review of the literature on passive smoking concludes that, with the exception of the unborn child, the evidence of harmful effects is limited. Long-term harmful effects on healthy adults have not been conclusively demonstrated, but atmospheres laden with tobacco smoke cause annoyance and irritation to eyes, noses, and throats of nonsmokers and a fair proportion of smokers. It has been reported that *N*-nitrosamine concentrations in sidestream smoke are 50 times greater than in mainstream smoke and recent studies suggest that exposure to benzo(*a*)pyrene may increase by about one-third (to 2/100,000) the rate of lung carcinoma among nonsmokers with regular and fairly heavy exposure to tobacco smoke. Recently, there has been a trend to create more nonsmoking areas in public places, but the issue of restrictions remains controversial. It may be an issue upon which toxicological evidence will have little impact. Since a number of studies indicate a relationship between parental smoking and respiratory disorders in children, it is recommended that parents avoid exposing their children, particularly the very young, to tobacco smoke.

82–1067. Kandarkar, S. V.; Hasgekar, N. N.; Sirsat, S. M. **Optical and Ultrastructural Pathology of Vitamin A Pretreated Hamster Cheek Pouch—Exposed to Lime ($Ca(OH)_2$) and Tobacco Over Total Life Span.** *Neoplasma* 28(6): 729-737, 1981.

Long-term oral exposure in hamsters to betel quid constituents (lime and tobacco) failed to induce malignancy. A study attempted to induce malignancy in the hamster cheek pouch epithelium by exposure over the total lifespan of the animals—100 to 110 weeks. Parallel conditioning by exposure to vitamin A was also included. The cheek pouch epithelium of animals exposed to the test substances for total lifespan showed only epithelial dysplasia of a marked degree, but no evidence of malignancy. It is concluded that it is not possible to simulate the time—dose relationship in human addiction to tobacco or to betel chew in experimental animals due to their comparatively short lifespan. (Auth. Abs. Mod.)

82–1068. Khan, M. A.; Provenza, D. V.; Seibel, W. **The Effects of Nicotine on Mouse First Molar Tooth Germs in Organ Culture.** *Journal de Biologie Buccale* 9(4): 335-348, December 1981.

Varying doses of nicotine were examined for their possible effects on first molar tooth germs from mice. Mandibular first molars, extirpated from 18-day mouse fetuses, were cultured. Two-day cultures were treated with 78, 117, 156, and 312 μg/ml nicotine sulfate. Untreated control tooth germs demonstrated normal growth in vitro. The peripheral cell layer of mesenchymal papilla and the inner enamel epithelium differentiated into odontoblasts and ameloblasts, respectively, with subsequent elaboration of extracellular matrix. Tooth germs treated with 78 μg/ml nicotine were only slightly affected, whereas higher doses of the drug produced extensive cell damage. Dental papilla appeared more sensitive than the enamel organ. Large necrotic foci were present in the pulp mesenchyme involving in some cases the odontogenic layer and severely limiting production of predentin. The basement membrane in these areas was partially disrupted. The inner enamel epithelium was not affected by 78 μg/ml dose of nicotine; however, higher doses produced extensive cell necrosis in this layer. Although enamel matrix was abundantly present in untreated controls, the tooth germs exposed to 117, 156, and 312 μg/ml nicotine sulfate failed to elaborate this extracellular matrix. Tooth germs exposed to nicotine for 24 hours followed by culturing in control medium demonstrated complete recovery. Treated cultures maintained in vitro for 7-9 days in control media had replaced the necrotic cells in the ameloblastic and odontoblastic layers and demonstrated abundant dentin and enamel matrices. (Auth. Abs. Mod.)

82–1069. Kuliczkowski, K. **Badania Stezenia Olowiu we Krwi i Aktywnosci Dehydratazy Kwasu Delta-Aminolewulinowego (ALAD) w Erytrocytach w Zaleznosci od Plci, Wieku, Palenia Tytoniu i Picia Alkoholu w Grupie Ludnosci Narazonej na Dzialanie Pylow Przemyslowych. [Studies of Lead Concentration in Blood and Activity of Delta-Aminolevulinic Acid Dehydratase (ALAD) in Erythrocytes, Depending on Sex, Age, Smoking Habit and Alcohol Drinking in a Population Sample Exposed to Industrial Dust.]** *Medycyna Pracy* 32(2): 99-107, 1981, Polish.

A population sample of 180 women and 219 men, aged 15 to 78 years, mostly farmers, who lived near two Polish copper works that emitted dust with very high lead content, was studied. Data on smoking habit and alcohol consumption were obtained through interviews. Blood lead concentration and Δ-aminolevulinic acid dehydratase (ALAD) activity in erythrocytes were determined in samples taken from each subject in the morning in a fasting state. The differences between the nondrinking, smoking men and the nondrinking, nonsmoking men were not found to be statistically significant for either lead or ALAD activity. Among the female subjects, the difference in mean blood lead concentration between the groups was not significant. However, the mean ALAD activity in the group of nondrinking, smoking women (2.63 units/ml) was significantly lower than in the group of nondrinking, nonsmoking women (6.32 units/ml). This difference between the sexes with respect to the mean ALAD activity is explained in terms of biochemical changes in women due to the synergistic effect of smoking and lead poisoning.

82–1070. Lancet. **Passive Smoking: FOREST, GASP, and Facts.** *Lancet* 1(8271): 548-549, March 6, 1982.

The issue of freedom—cigarette smokers vs. nonsmokers faced with passive smoking—is discussed. The evidence of harm through passive smoking effects, although largely based on effects on the family, presents a problem for the influential smokers' lobby, which has claimed that passive smoking is not harmful. In addition, it seems clear that providing the needed epidemiological evidence may not be as difficult as it once seemed. In the United States, the Surgeon General, and in the United Kingdom, the Royal College of Physicians of London have not yet committed themselves on passive smoking. Some rethinking now seems justified, not because neutrality will be seized on by FOREST (Freedom Organization for the Right to Enjoy Smoking Tobacco) to the detriment of GASP (Group Against Smoking in Public), but on scientific grounds.

82–1071. Lander, T.; Standl, E.; Dexel, T.; Siess, E. A.; Naethke, M. E.; Albert, E. D.; Scholz, S. **Untersuchung der Basalmembrandicke bei Patienten mit Typ I-Diabetes unter Beruecksichtigung der Diabetischen Retinopathie, des Zigarettenkonsums und der HLA-Antigene. [Study of the Basement Membrane Thickness in Patients With Type I Diabetes With Regard to Diabetic Retinopathy, Cigarette Consumption and HLA-Antigens.]** *Verhandlungen der Deutschen Gesellschaft fur Innere Medizin* 87: 110-113, April 26-30, 1981, German.

Muscle capillary basement membranes and retinopathy development were studied in 54 young male insulin-dependent diabetics. The duration of the disease had ranged from 0.1 to 26 years. Retinopathy in various stages of severity was found in 37 of the 54 patients. The basement membrane thickness correlated directly with the duration of diabetes. It was significantly lower in the patients without retinopathy. The average thickness in the 22 diabetics who had smoked over 15 cigarettes/day for many years was 2,210 A versus 1,648 A in 15 nonsmokers with equal average duration of the disease (12.7 vs. 12.8 years). The cited data confirmed that patients with clear symptoms of diabetic retinopathy and long duration of diabetes exhibit a thicker basement membrane than those without retinopathy. Cigarette smoking appears to be a significant factor in the thickening of the basement membrane and subsequent development of diabetic retinopathy.

82–1072. Lee, P. N. **Passive Smoking. (Letter).** *Lancet* 1(8275): 791, April 3, 1982.

In a response to a previous editorial (*Lancet* 1(8271): 548-549, March 6, 1982), it is argued that the evidence upholding passive smoking as harmful is insufficient. The four studies cited as support for the risks of passive smoking are criticized for various methodological shortcomings such as: statistical analyses which are shown to produce spurious results; unusual or too small study populations; and problems in interpreting results. See also 82-1070.

82-1073. Lewis, D. J. **The Use of Horseradish Peroxidase To Demonstrate Degenerate Cells in Rat Larynx After Acute Tobacco Smoke Exposure.** *Toxicology Letters* 9(3): 195-199, November 1981.

An investigation used horseradish peroxidase (HRP) to detect degenerate cells in rat laryngeal epithelia after one exposure to tobacco smoke. The HRP was administered topically to laryngeal epithelia of rats exposed to tobacco smoke for a single 20-minute period. The HRP demonstrated the distribution of degenerate and/or injured cells. These cells were specifically located in the ciliated epithelium in the ventrolateral region, at the base of the epiglottis. It is demonstrated that the cells were unable to exclude HRP, suggesting that the plasmalemma was either ruptured, damaged, or more permeable, indicating a degree of cell injury. (Auth. Abs. Mod.)

82-1074. Marks, I. N.; Wright, J. P.; Denyer, M.; Hatfield, A.; Girdwood, A. H.; Lucke, W. **Ranitidine Heals Duodenal Ulcers.** *South African Medical Journal* 61(5): 152-154, January 30, 1982.

In a group of ulcer patients, smoking had no effect on the healing rates of patients treated with ranitidine, and only a marginally adverse effect on those treated with a placebo. A double-blind placebo-controlled study of ranitidine (150 mg given twice daily for 4 weeks) was carried out in 65 ambulatory patients with endoscopically proven duodenal ulceration. Over 80 percent of the patients were smokers. Endoscopic healing (with or without residual erosion) was noted in 28 of the 34 ranitidine-treated patients (82 percent) and in 14 of the 31 placebo-treated patients (45 percent) ($p < 0.005$). (Auth. Abs. Mod.)

82-1075. Mattheus, A.; Heise, H. **Der Einfluss von Umweltnoxen auf die Fertilitaet. [Injurious Environmental Influences on Fertility.]** *Dermatosen Beruf Umwelt* 29(5): 135-138, 1981, German.

The possible factors in reproductive alterations in men, or diseases such as inhibition of sperm generation or testes atrophy, are surveyed, including published studies and the authors' own investigations. Nicotine abuse is cited as one of the potentially controllable factors of sperm generation inhibition. Controversial data are found in the literature about the influence of tobacco consumption on spermatozoa motility. Although a significant relationship has not been established between thiocyanate levels in sperm plasma and spermatozoa motility, in many cases, severe motility disturbances were noted in men who smoked 20 to 40 cigarettes/day. The necessity for a wide-ranging health information campaign is stressed because of increasing nicotine abuse and its negative consequences for human health.

82-1076. McGill, H. C., Jr. **The Human Model.** In: Gori, G. B. (Editor). *Proceedings of the Tobacco Smoke Inhalation Workshop on Bioassay Models and Inhalation Toxicology.* (Draft). U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, January 28, 1981, pp. 13-30.

Problems in the general use of animals as patterns copied from the human model in disease research are considered. An important difference between human and animal models is the genetic heterogeneity of the human population in contrast to the relatively homogenous strains of laboratory animals. Epidemiologic studies of humans may deal with many thousands of subjects, which is usually not possible in animal studies. Shortened animal lifetimes mean that exposures to injurious agents cannot be the same as for humans. Atherosclerosis and tobacco smoke inhalation are described as examples of human models in disease research. Cigarette smoking apparently acts in part by accelerating the progression of lesions, by affecting blood chemical variables, and by predisposing to the terminal occlusive episode. Smoking's association with chronic obstructive pulmonary disease and cancer involve different organ systems, with different anatomy, functions, and responses. See also 81-1114.

82-1077. Modeer, T.; Lavstedt, S.; Ahlund, C. **Relation Between Tobacco Consumption and Oral Health in Swedish Schoolchildren.** *Acta Dermatologica Scandinavica* 38(4): 223-227, 1980.

A group of 232 schoolchildren aged 13-14 years in the municipality of Huddinge, on the outskirts of Stockholm, was examined to study the effects of smoking and oral use of snuff in relation to oral health. Of the schoolchildren 21.5 percent smoked regularly. Snuff was taken regularly by 11 percent of the boys, but none of the girls. Snuff was present in the oral cavity for an average of 3.5 hours every day. The number of cigarettes was found to be a significant ($p < 0.01$) predictor to the dependent variable, plaque, after controlling for sex of schoolchildren and frequency of toothbrushing. Snuff use showed a significant correlation to gingival index after controlling for plaque. The results showed that use of snuff may influence the gingival tissue directly, whereas smoking affects plaque accumulation. (Auth. Abs. Mod.)

82-1078. Moshal, M. G.; Spitaels, J. M.; Khan, F.; Mason, J.; Naidoo, V.; Schlemmer, L. **Factors Affecting Duodenal Ulcer Healing.** *South African Medical Journal* 61(6): 202-206, February 6, 1982.

No significant differences in healing rates were found between smoking and nonsmoking duodenal ulcer patients who had participated in four double-blind endoscopically controlled trials carried out in 1978-19[illegible]. Drugs utilized were: a new histamine H_2-receptor antagonist ranitidine vs. placebo; the colloidal bismuth ulcer-coating preparation bicitropeptide vs. cimetidine; the tricyclic antidepressant trimipramine vs. placebo; and the disaccharide coating preparation sucralfate vs. placebo. Factors analyzed were smoking, alcohol consumption, age, length of history, sex, and race. The prevalence of smoking and alcohol use, age, and length of history varied in the four trials. Pronouncements regarding these factors based on small trials are therefore unreliable. There were no overall significant differences in healing rates whether the patient smoked, consumed alcohol, was above or below 40 years of age, or had a history shorter or longer than 1 year. Comparison of drug and placebo responses revealed that the differences between these were most significant in: nonsmokers ($p=0.0010$) compared with smokers ($p=0.0288$); teetotallers ($p<0.005$) compared with alcohol consumers (not significant, NS); history under 1 year ($p<0.005$) compared with over 1 year ($p<0.025$); males (<0.01) compared with females (NS); and Indians ($p<0.005$) compared with blacks (NS). (Auth. Abs. Mod.)

82–1079. Pantin, C.F.A.; Merrett, T. G. **Smoking and IgE Levels. (Letter).** *British Medical Journal* 284(6317): 744, March 6, 1982.

In a response to a previous article (*British Medical Journal* 283(6301): 1215-1217, November 7, 1981) which correlated smoking and increased immunoglobulin E (IgE) levels, a study is cited which gave different results. The only significant difference in IgE levels found in the latter study was with respect to sex, though this was concentrated in the smoking group. In another survey, males over age 70 had higher IgE levels than females. Thus, IgE may increase with age, owing to longer exposure to smoke and allergens, and would be higher among males since men are more likely to be heavy smokers. See also 82-0506.

82–1080. Peden, N. R.; Callachan, H.; Shepherd, D. M.; Wormsley, K. G. **Gastric Mucosal Histamine and Histamine Methyltransferase in Patients With Duodenal Ulcer.** *Gut* 23(1): 58-62, January 1982.

In a study of duodenal ulcer patients, it was concluded that cigarette smoking may have accounted for reduced fundic mucosal histamine levels. Histamine and histamine methyltransferase (HMT) were measured in the gastric mucosa from 110 patients with duodenal ulcer and 62 control subjects. Both antral and fundic mucosa had similar levels of HMT activity despite antral mucosa containing significantly less histamine. Patients with duodenal ulcer had significantly lower levels of fundic mucosal HMT activity and lower concentrations of fundic mucosal histamine than control subjects. An additional finding was that men who were cigarette smokers had significantly lower concentrations of fundic mucosal histamine (but not HMT) than nonsmokers and, as there was an excess of cigarette smokers among patients with duodenal ulcer, this may explain the reduced concentrations of fundic mucosal histamine in these patients. (Auth. Abs. Mod.)

82–1081. Pogrel, M. A. **Sublingual Keratosis and Malignant Transformation.** *Journal of Oral Pathology* 8(3): 176-178, June 1979.

Leukoplakia of the floor of the mouth and ventral surface of the tongue appear to carry greater risk of malignant transformation. In a retrospective study of 19 cases, 8 admitted to being heavy smokers (>20 cigarettes/day). Three of the nineteen patients (16 percent) developed sublingual carcinoma within 7 years of diagnosis of leukoplakia. Two of the three were smokers, one of whom also reported high alcohol intake. Other oral sites have shown only a 5 percent transformation in recent studies.

82–1082. Roberts, D. M.; Wilson, J. A.; Ratcliffe, G. E.; Waring, A. J.; Reilly, M. J.; Lloyd, J. S. **Clinical Trial of Ranitidine in the Treatment of Peptic Ulcer.** *British Journal of Clinical Practice* 36(1): 9-12, January 1982.

Outpatients with peptic ulcer attending British military hospitals have been shown to be relatively resistant to treatment with a conventional H_2-receptor antagonist. From this population 80 patients with peptic ulcer were entered into a prospective double-blind placebo controlled trial of treatment with ranitidine 100 mg three times a day, followed by an open study for nonhealers. A ranitidine heal-rate of 54.1 percent after 4 weeks compared very favorably with the placebo 4-week heal-rate of 7.7 percent. The 8-week cumulative heal-rate for those who received ranitidine in the closed part of the trial was 69 percent and for those given ranitidine in the open part of the trial it was 94 percent. Above average alcohol consumption was associated with resistance to healing in response to ranitidine, but age and drug compliance did not influence the outcome. Surprisingly, no association between cigarette smoking and resistance to treatment was found, although the literature suggests otherwise. A high proportion, 70.6 percent, of the patients were smokers, but this does not differ appreciably for the 72 percent incidence among all army personnel and the 70 percent incidence among young men in the general population. However, it is possible that this renders them relatively intractible as a "blanket" effect obscuring differences among subgroups. Further research in this area is necessary. (Auth. Abs. Mod.)

82–1083. Weber, A. **Passivrauchen, Luftqualitat, Massnahmen. [Passive Smoking, Air Quality, and Protective Measures.]** *Sozial-und Praeventivmedizin* 26(3): 182-184, 1981, German.

Laboratory and field studies have shown that acute irritations and annoyances can be tolerated until a smoke concentration corresponding to 1 to 2 ppm is reached. Protective measures for passive smokers are recommended if the carbon monoxide due to tobacco smoke exceeds 1 ppm, and necessary if it exceeds 2 ppm. The most important measures are (1) the reduction of the pollutants at the source by a smoking prohibition or a separation into smoker and nonsmoker rooms or areas; (2) air cleaning by filters; and (3) ventilation based on calculations. Recommendations are given for ventilation so that the above-mentioned limit values are not exceeded. (Auth. Abs.)

82–1084. Weiss, W. **Smoking: Burning a Hole in the Balance Sheet.** *Personnel Management* 13(5): 24-29, May 1981.

The smoking habit is reviewed in terms of its cost to industry. Companies that prohibit smoking in the workplace and hire only nonsmokers could save up to 10 percent in salaries and wages, 30 percent in health and fire insurance premiums, 50 percent in furniture depreciation, 50 percent in cleaning expenses, and 75 percent in disability benefits. Nine nonsmokers can do the work of ten smokers because of the time wasted in consuming cigarettes, thus saving in payroll costs. In addition, a smoking ban improves employee morale, which in turn improves productivity. Hiring talented personnel may be easier because of favorable reactions to the no-smoking policy and improved working conditions. Customers are shown to be respectful and compliant with the rules. Examples are given from a small U.S. electronics firm, a nightclub, the British Health Service, a chemical manufacturer, and other companies.

See also, **82–0971, 82–1024, 82–1057**

82–1085. Zovic, D. **Higijenski Znacaj Negativnog Dejstva Duvanskog Dima na Jonizaciju Vazduha u Radnoj Prostoriji. [Hygienic Significance of the Negative Effect of Tobacco Smoke on Ionization of Air in the Work Environment.]** *Vojnosanitetski Pregled* 37(2): 98-101, March-April 1980, Serbo-Croatian.

Positive and negative ions and microclimate parameters (temperature, relative humidity, atmospheric pressure) were measured in the air of a 3.6 x 3.6 x 4 m naturally ventilated office in the presence of cigarette smoke, with or without a lighted stearin candle, and in the absence of smoke. Ambient smoke originated from smoking about three cigarettes/hour. Measurements were taken hourly for 4 days between 7:00 a.m. and 2:00 p.m. inside and outside the office. It was determined that the average concentration of positive and negative ions in 1 cm percent of air in the office: (1) decreased by 53.5 percent in the presence of smoke compared to the concentration in the absence of smoke; (2) increased by 160 percent with a lighted stearin candle in the absence of smoke, and (3) increased by 213 percent when a stearin candle was lighted following 4 hours of cigarette smoking. The deionizing effect of tobacco smoke on the air in a room is known to be the cause of subjective and objective symptoms of health troubles. The flame of a stearin candle partly compensates for the loss of ions due to smoking and eliminates the disagreeable smell of tobacco smoke, but it is not considered a substitute for smoking prevention measures.

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

82–1086. Aaro, L. E.; Hauknes, A.; Berglund, E.-L. **Smoking Among Norwegian Schoolchildren 1975-1980. II. The Influence of the Social Environment.** *Scandinavian Journal of Psychology* 22: 297-309, 1981.

Results of a 1975 survey of Norwegian schoolchildren indicated that the social environment of the student had a definite effect on smoking behavior. All primary schools had been asked to carry out local studies of the smoking habits of all pupils in grades 6-9 (ages 12-15). A sample of 6,968 questionnaires from the pupils formed the empirical basis of the present study. Among ninth graders (age 15) coming from homes where both parents smoked and the children were permitted to smoke, 67 percent of the boys and 78 percent of the girls smoked daily. Among children from homes where both parents were nonsmokers and did not permit their children to smoke, only 9 percent of the boys and 11 percent of the girls smoked daily. Thus, the probability of being a daily smoker can generally be regarded as the additive effect of milieu predictors. Where both parents smoked, older sisters and brothers smoked, best friends smoked, and the pupils were permitted to smoke, practically all 15-year-olds were daily smokers. In contrast, an environment of nonsmokers and with no permission to smoke reduced the probability of being a daily smoker to almost zero. (Auth. Abs. Mod.) For part I of this series, see 82-0507.

82–1087. Biener, K. **Tabak-, Alkohol- und Tablettenkonsum Berufstaetiger Frauen—Repraesentativstudie in der Schweiz. [Tobacco, Alcohol and Tablet Consumption by Professional Women—Representative Swiss Studies.]** In: *Frau und Sucht: Beobachtungen, Erfahrungen, Therapieansatze.* Hamm, Hoheneck-Verlag Gmblt, 1981, pp. 62-70, German.

The smoking habits of 1,033 professional women, aged 20 to 65 years, from randomly selected northern Switzerland enterprises, were surveyed and compared to the smoking habits of 1,260 professional men from the same companies. Of the groups, 40 percent of the women were smokers compared to 48 percent of the men. An impressive 38 percent of the smoking women, aged 20 to 25 years, started smoking before the age of 16 compared to only 3 percent of those aged 40 to 45. Only 4 percent of the professional women were ex-smokers who used to smoke 20 or more cigarettes/day compared to 14 percent of their male counterparts. The older the woman, the harder it was for her to stop smoking. The women were relatively less motivated than men to stop smoking: only 48 percent of the women smokers expressed this wish vs. 78 percent of the men. In comparison, only 11 percent of the nonprofessional wives used to smoke cigarettes and only 5 percent of them had smoked 20 or more cigarettes per day.

82–1088. Buchkremer, G. **Arbeitsgruppe 4. Psychologie, Praevention und Therapie des Rauchens bei der Frau. [Task Group 4. Psychology, Prevention and Therapy of Smoking by Women.]** In: *Frau und Sucht: Beobachtungen, Erfahrungen, Therapieansatze.* Hamm, Hoheneck-Verlag Gmblt, 1981, pp. 151-159, German.

Recent surveys demonstrate a steady increase in smoking by women in the United States and Germany. Statistics show that women start smoking at an increasingly younger age, cease smoking at a later age than men, and are less motivated than men to stop smoking. Also, women inhale more frequently and smoke their cigarettes to the end more often than men. A strong trend toward antisocial activities and imitation of masculine behavior are described as sex-specific characteristics of smoking women. Delayed development of newborn infants, perinatal mortality of the infants, and their more frequent illnesses during the first 2 years are the main smoking-related health problems in children of smoking mothers. An increased risk of thromboembolism was observed in smoking women who took oral contraceptives at the same time. Specific smoking prevention measures for women and future mothers are indicated and the most important smoking therapies are tabulated.

82–1089. Buchkremer, G. **Bericht der Arbeitsgruppe 4. Psychologie, Praevention und Therapie des Rauchens. [Report of the Task Group 4. Psychology, Prevention and Therapy of Smoking.]** In: *Frau und Sucht: Beobachtungen, Erfahrungen, Therapieansatze.* Hamm, Hoheneck-Verlag Gmblt, 1981, pp. 159-160, German.

The task force attributes the lack of interest in the health effects of smoking to an insufficient awareness of the problem. The group found a more frequent conflict toward smoking in women than in men or adolescents. Men must undergo necessary changes in parallel with the new orientation of women. In adapting smoking therapy to women, overcoming women-specific conflicts, the unsuitability of drug therapy, and consideration of the emotional and imaginative element are emphasized. Preventive measures were discussed in groups according to age. It is concluded that preventive measures against smoking must be considered within the larger concept of health education and psychosocial measures. Development of smoking prevention measures financed with taxes on tobacco products is recommended.

82–1090. Check, W. A. **New Knowledge About Nicotine Effects.** *Journal of the American Medical Association* 247(17): 2333, 2337-2338, May 7, 1982.

New research findings on the physiological and psychological implications of low-tar, low-nicotine cigarettes indicate that nicotine dependence in smokers could be better controlled by a low-tar, high-nicotine combination. Evidence indicates that nicotine is responsible for many physiological and psychological effects, with alterations in metabolism and mood considered major reasons why people smoke and have difficulty when trying to quit. It is suggested that some of these nicotine effects are mediated through the brain by specific receptors concentrating nicotine from the plasma to levels about five times higher than those in the plasma. Approximately 90 percent of the nicotine delivered to the lungs goes directly to the brain (in only 7 seconds), producing a "bolus" effect: the very high concentration of nicotine in the blood immediately after a puff serves as a powerful "arousal modulator" that can either energize or calm. It is concluded that a low-tar, *high*-nicotine cigarette that would provide an easy "fix" but prevent the carcinogenicity of high tar levels would be a preferable alternative to increased consumption of low-nicotine, low-tar cigarettes.

82–1091. Cherek, D. R. **Effects of Smoking Different Doses of Nicotine on Human Aggressive Behavior.** *Psychopharmacology* 75(4): 339-345, December 1981.

The effects of not smoking, of smoking a low-nicotine dose (0.42 mg/cigarette), and of smoking a high-nicotine dose (2.19 mg/cigarette) on human nonaggressive and aggressive responding were determined. A nonaggressive response, which resulted in the accumulation of money, was continuously available to the subject. Two different aggressive responses were also available: the subtraction of money from and the presentation of a 1-s blast of white noise to a (fictitious) person. Aggressive responding was elicited by subtracting money from the research subjects, which was attributed to a fictitious person paired with the research subject randomly each day. Nicotine, administered with experimental cigarettes, produced dose-dependent decreases in both types of aggressive responding elicited by low- or high-frequency subtractions of money attributed to another person. Generally, the more aggressive response option, i.e., subtraction of money from another person, decreased following nicotine administration. Smoking the same doses of nicotine increased nonaggressive monetary reinforced responding. This indicates that the suppressant effect of nicotine on aggressive responding was not due to a nonspecific depressant action. (Auth. Abs. Mod.)

82–1092. Costa, P. T., Jr.; McCrae, R. R. **Stress, Smoking Motives, and Psychological Well-Being: The Illusory Benefits of Smoking.** *Advances in Behaviour Research and Therapy* 3(4): 125-150, 1981.

The deleterious physical effects of smoking have long been documented, and other research suggests that smoking does not provide psychological benefits for smokers, although smokers often claim that there are compensatory psychological benefits which accompany the use of cigarettes for sedation or pleasure. Studies undertaken to test the hypothesis that, for some individuals, certain kinds of cigarette smoking may contribute to psychological well-being are summarized. Measures of life stress, age, and the personality dimensions of neuroticism, extraversion, and openness to experience were collected on a sample of 1,101 adult male volunteers. Smoking status, smoking motives, and psychological well-being were also measured. Results showed that (1) both personality and smoking motives show a replicable factor structure and are stable over many years; (2) age, life stress, and demographic variables show little relation to psychological well-being, whereas the personality variables of neuroticism and extraversion show stronger relations; (3) smoking is associated with neuroticism, but not with extraversion; and (4) neither smoking itself nor smoking for any particular motivation shows any effect on psychological well-being. It is concluded that the psychological "benefits" reported for smoking are illusory, and that alternative coping strategies should be pursued. (Auth. Abs. Mod.)

82-1093. Couto, C.L.M.; Castro, E.J.S.; Rossoni, E. L.; Rangel, M.C.M. **Adolescencia e Tabagismo. [Adolescents and Tobacco.]** *Separata da Revista Amrigs* 25(1): 26-37, January-March 1981, Portuguese.

The smoking habits of 2,417 students, aged 10 to 19 years, at three Porto Alegre (RS), Brazil, schools were investigated by questionnaire. Motivation to smoke, attempts to quit, daily consumption, job influence, parental and peer group influence, advertising influence, respiratory symptoms, parental attitudes, knowledge of health effects of smoking, socioeconomic status, and school performance were measured. The data revealed that 52.8 percent of the students had experimented, and 20.6 percent were regular smokers. The proportion of female smokers was higher than that of male smokers (25 vs. 16.8 percent). The mean starting age was 13.7 years. The highest prevalence of smokers was in the lower socioeconomic bracket (42.6 vs. 25.7 percent) and in the higher age groups (5.3 percent of the 10- to 13-year-olds vs. 38.4 percent of the 17- to 19-year-olds). Among the smokers, 64.3 percent reported a cough, and 44.9 percent a hack, which was correlated with smoking time and consumption per day. Daily quantity smoked was greater in the older students (2.1 percent of the 10- to 13-year-olds smoked more than 10 cigarettes per day in contrast to 32.1 percent of those 17 to 19 years old). Smokers had a lower mean performance in school of 1 year compared to nonsmokers of the same class. (Auth. Abs. Mod.)

82-1094. Davidsson, D.; Olafsson, H.; Sigfusson, N.; Bjornsson, O. J.; Olafsson, O.; Karlsson, S. **Reykingavenjur Islenzkra Karla A Aldrinum 34-61 Ars. [Smoking Habits of Icelandic Males Aged 34-61 Years.]** Report AV From The Health Survey in the Reykjavik Area. Stage I, 1967-68, Men. Reykjavik, Icelandic Heart Association, 1981, 90 pp., Icelandic.

A survey showed that approximately 64 percent of men aged 34 to 61 years in the Reykjavik area are smokers, decreasing with age from 70 percent at 35 years to about 60 percent at age 60. Approximately 25 percent of men in their late thirties smoke cigarettes only, with a negligible decline with age. About 13 percent, aged 34 to 61, smoke cigarettes as well as other tobacco products. Cigarette smoking prevalence generally decreases from about 40 percent at age 30 to 35 percent at age 60. Inhalers among cigarette smokers decrease from 90 percent in the youngest age groups to 70 percent at age 60. About 6 percent of all smokers use filtered cigarettes. Camel was the favorite brand of almost 80 percent of cigarette smokers, 60 percent of whom smoke 15-24 cigarettes per day (75 percent in the youngest group and less than 50 percent of the oldest). Age at onset was 15-19 for 50 percent of smokers and 20-24 years for 30 percent. Ex-cigarette smoking increased from 30 percent in the youngest group to about 38 percent in the oldest. Percentage of heavy smokers (>25 cigarettes/day) was lower in the ex-smoker group than in the current smoker group. Reasons for quitting were mainly health and economic. For pipe smoking 50 percent of the entire group were users at one time and 25 percent are current users. About 45 percent of the youngest group have smoked cigars and 35 percent of the oldest group. Currently 25 and 15 percent are cigar smokers in the youngest and oldest groups, respectively. (Auth. Abs. Mod.)

82-1095. Deutsche Hauptstelle Gegen die Suchtgefahren. **Rauchen oder Gesundheit: Politische, Praeventive und Therapeutische Aspekte. [Smoking or Health: Political, Preventive and Therapeutical Aspects.]** Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, 211 pp., German.

This is a collection of papers presented at the May 1980 Congress in Bonn-Bad Godesberg on the occasion of World Health Day. Smoking is treated as a medical and political problem on an international scale. Smoking prevention measures are reported for Finland, Sweden, Austria, Switzerland, and West Germany. Smoking prevention as a problem of health education in West German schools is examined. Smoking habits of West German students are described, and motivation for smoking is studied in Swiss youth. Blood tests for cancer in smokers are reported, and the risks of smoking during pregnancy are discussed. Legal aspects of nonsmokers' protection in West Germany are explained. West German and Swiss specialists analyze their observations and results of smoking withdrawal therapies.

82-1096. DiClemente, C. C.; Prochaska, J. O. **Self-Change and Therapy Change of Smoking Behavior: A Comparison of Processes of Change in Cessation and Maintenance.** *Addictive Behaviors* 7(2): 133-142, 1982.

Cigarette smokers who quit on their own were compared with subjects from two commercial cessation programs: Aversion Group and Behavior Management Group. Subjects were administered a Change-Process Questionnaire and a demographic and smoking-history questionnaire within 7 weeks of successful cessation, then interviewed again in 5 months. Using a transtheoretical model of change, six verbal and four behavioral processes of change and three stages of change (Decision to Change, Active Change, Maintenance) were analyzed. Subjects in each treatment group were middle-class, heavy-smoking adults. The change-process analysis of cessation discriminated between the self-quitters and therapy quitters and between the two groups of therapy subjects on five variables. Stages of change interacted with the processes of change in the cessation of smoking behavior. Verbal processes were seen as important in making the decision to change while action processes were critical for breaking the actual smoking habit. Maintenance of cessation was related to, but not dependent on, how subjects actively changed smoking behavior. (Auth. Abs.)

82-1097. Dougherty, J.; Miller, D.; Todd, G.; Kostenbauder, H. B. **Reinforcing and Other Behavioral Effects of Nicotine.** *Neuroscience and Biobehavioral Reviews* 5(4): 487-495, Winter 1981.

A wide range of nicotine self-administration literature was reviewed to determine whether nicotine can act as a reinforcing stimulus for behavior leading to its administration. Published findings of intravenous nicotine self-injection indicate that the reinforcing properties of nicotine are weak when the drug is made available according to continuous reinforcement (CRF) or fixed-ratio (FR) schedules. CRF self-injection rates are generally only two to three times saline control levels, and self-injection frequency is largely insensitive to changes in unit dose. In contrast, drugs of the psychomotor stimulant, opiate, and sedative-hypnotic classes, with similar pharmacokinetic parameters, maintain much higher self-injection rates and show systematic changes in rate with unit dose variations. Recent studies using interval and second-order schedules of nicotine presentation have been more successful in maintaining higher rates of self-administration behavior. Systematic dose— response functions have also been found under these conditions. Food deprivation, species and strain differences, circadian rhythms, and duration of exposure to the drug also appear to be important variables in determining self-injection rate. Finally, the rapid development of tolerance to the effects of nicotine may account for changes in the pattern of self-administration within daily sessions and the differential sensitivity of those patterns to nicotine pretreatment. (Auth. Abs. Mod.)

82-1098. Duryea, E.; Krueter, M. **Declarative Consistency: An Inquiry Into Health Decision Making Skills in Students.** *Eta Sigma Gamman* 12(1): 2-6, Spring/Summer 1980.

Results of a smoking survey of 726 fifth, seventh, ninth, and eleventh grade students in Ohio, Utah, and California, indicate that sound decisionmaking skills need to be taught in school health education programs to encourage consistency in thinking through health decisions. The four-step expectancy model questionnaire used addressed the following: (1) does the ability to declare beliefs and values consistently throughout the process of making a health decision become more prominent with increasing grade level, (2) will students with a behavioral intention of "no" toward the smoking decision demonstrate higher levels of declarative consistency than students with a "yes" behavioral intention, and (3) will students with a "maybe" intention have a stronger measure of declarative consistency than subjects with a "yes" intention. Previous research has indicated that schoolchildren not only face abstract information and ambiguous social guidelines, but may frequently have to deal with peer pressure situations where, due to high emotional arousal, errors in judgment can occur, due in part to a lowering of efficiency in cognitive functioning. The disappointing performance of the subjects with respect to their declarative consistency ratings indicates the need for curricular inclusion of sound decisionmaking skills, since even students who may have good reasons for making a health choice may not necessarily value them as important in the decision itself. Any program that attempts to teach sound decisionmaking must also take into account the element of peer pressure, and combining the ability to think through health choices and the ability to respond to social situations confidently may form the strongest strategy to date to deter youth from smoking.

82-1099. Eastwood, M. R. **Smoking Profiles of Patients Admitted for Neurosis. (Letter).** *British Journal of Psychiatry* 140: 102, January 1982.

One of the authors of a 1971 study (*Lancet* 1(7690): 107-108, January 16, 1971) which found smoking and neuroticism to be unrelated responds to criticism (*British Journal of Psychiatry* 139: 43-46, July 1981) that the study was unconvincing, as all psychiatric disorders were included and neuroticism was not measured. However, 95 percent of the sample suffered from neurotic illness; thus the study was valid. Since the frequency of smoking is twice that of psychiatric disorders in the general population, cultural factors must play the greater role. Smoking is increasing among the young, while the prevalence of neurotic illness is in the middle-aged group. Where smoking reduction occurs, it is unlikely that it is due to recovery from a neurotic illness. The theory that smoking is a panacea for neuroticism does not seem tenable for the population as a whole. See also 71-0325, 81-1907.

82-1100. Eiser, J. R. **Addiction as Attribution: Cognitive Processes in Giving Up Smoking.** In: Eiser, J. R. (Editor). *Social Psychology and Behavioral Medicine.* New York, John Wiley and Sons, 1982, pp. 281-299.

Smoking qualifies as an addiction in behavioral and pharmacological criteria: the process of relapse is the same as with alcohol and opiates. The popular stereotype of addiction involves being "hooked," with little hope of breaking the habit, or that one is sick, and therefore not personally responsible for an unhealthy habit. However, nonsmokers are more likely to label the average smoker as addicted than are the smokers themselves, and nonsmokers consistently underestimate the amount of pleasure obtained by smoking. In smokers, there is an association between self-labeling of addicted and cognitive dissonance. Those who were dissonant were much more likely to be frightened by the health consequences of their habit. Though most smokers acknowledge the health risks, the knowledge they have is not necessarily accurate. Smokers hold less negative beliefs about smoking's effect on health than nonsmokers, and 18 percent of smokers surveyed (vs. 7 percent of ex-smokers) believed after 10 years damage was irreversible and there was no point in quitting. Among those who attempted to quit and succeeded, the most powerful predictor of their success was the longest period of abstinence managed in the past, followed by the perceived importance of avoiding cancer, and their perceived chances of doing so if they quit. Self-expectancy of success was also a reliable predictor, whereas those who perceived of themselves as hooked predictably failed. Self-labeling as sick did not discriminate successes from failures. What is needed is to separate the concept of addiction from its implications of helplessness and diminished or nonexistent responsibility.

82–1101. Evans, R. I.; Henderson, A.; Hill, P.; Raines, B. **Smoking in Children and Adolescents: Psychosocial Determinants and Prevention Strategies.** In: Krasnegor, N. A. (Editor). National Institute on Drug Abuse Research Monograph 26, U.S. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (ADM) 79-882, August 1979, pp. 69-96.

Smoking among children and adolescents is described in terms of present smoking patterns and beliefs; several conceptual models in developmental and social psychology are considered. Psychosocial factors that can affect a young person's decision to smoke include changing sex roles, parental smoking habits, parental acceptance of smoking by children, smoking siblings, rebellion against family authority, peer pressures, school environment, mass media, personal characteristics, and perceptions of the hazards of smoking. Present prevention programs are critically evaluated, specifically public information campaigns and school programs. Recommendations for further research and prevention programs are included.

82–1102. Gordon, L. V.; Haynes, D. K. **Parents' Attitudes Toward Cigarette Smoking and Their Children's Future Smoking.** *Health Education* 13(2): 17-21, March-April 1982.

Substantial differences in attitudes toward smoking were found between smoking and nonsmoking parents of fourth grade schoolchildren. In Rochester, New York, children in nine schools received a smoker education program, which involved education at school and homework with parental participation. In four other schools the program took place at school only. Parents of children in both programs were later surveyed for smoking attitudes. While there were no significant differences between the parents in the homework and nonhomework samples on the attitude items surveyed, there were noticeable differences between smoking and nonsmoking parents. A majority of both smoking and nonsmoking parents acknowledged smoking's possible health hazards. A majority of nonsmokers preferred others not to smoke in their vicinity, and strongly favored prohibition of smoking in public areas as well as cigarette tax increases. Nonsmokers were unwilling to consider unrestricted smoking as a right, a position endorsed by a majority of the smokers. Although a majority of both groups opposed future smoking by their children, nonsmokers had a significantly larger majority. Smoking parents, however, were more in favor of fourth grade smoking education programs.

82–1103. Ibsen, K. K. **Smoking Habits in 9000 Danish Schoolchildren.** *Acta Paediatrica Scandinavica* 71(1): 131-134, January 1982.

Results of a sample survey indicated a decline in the initiation of smoking among Danish children. A stratified sample of 9,158 Danish schoolchildren from 39 schools and 118 children from a handball club team championship participated in a questionnaire survey of smoking habits. Compared with previous investigations, a decrease in the percentage of children who had tried to smoke and in the children who actually smoked was observed. In most of the classes, smokers now constitute a numerical minority. In most of the older age groups, a preponderance of girl smokers is now present. Most of the smokers started to smoke out of curiosity or because their companions were smokers, whereas only 5 percent started to smoke because their parents were smokers. Children in the capital became regular smokers earlier than children in rural areas. Only 8 percent of the children participating in a championship for handball teams were smokers whereas 31 percent of the remainder of the sample in the same age group were smokers. If more children could be encouraged to participate in sports, it is possible that attitudes which could reinforce smoking health education could be established. (Auth. Abs. Mod.)

82–1104. Jaffe, J. H.; Kanzler, M. **Smoking as a Psychiatric Disorder.** In: Pickens, R. W.; Heston, L. L. (Editors). *Psychiatric Factors in Drug Abuse.* Conference on Psychiatric Factors in Drug Abuse, University of Minnesota, March 4-6, 1979, pp. 239-265.

Four issues must be considered in approaching smoking as a psychiatric disorder. First, the extent to which tobacco use resembles other drug-using behaviors: long-term health effects aside, tobacco use does not disable the way alcohol or opiates do. Yet nicotine tolerance occurs, as does a withdrawal syndrome when the smoker attempts to quit. Also, smoking is more common among illicit drug users. Second, the diagnostic implications of including tobacco use with alcoholism and other drug dependence may mean labeling millions of smokers as having a mental disorder. In addition, the quantity smoked does not always separate the addict from the experimenter or occasional smoker, so mental disorder/addiction cannot be defined in terms of nicotine intake. Third, the factors leading to habituation must be taken into account. These factors are not necessarily pharmacological. Smokers do not perfectly titrate nicotine levels. However, neither do alcoholics or opium users; thus a lack of nicotine titration does not exempt tobacco from being an addictive substance. Finally, the contribution psychiatry may make in understanding and treating smoking behavior and the future of the tobacco habit are discussed.

82–1105. Jaffe, J. H.; Kanzler, M. **Nicotine: Tobacco Use, Abuse, and Dependence.** In: Lowinson, J. H.; Ruiz, P. (Editors). *Substance Abuse. Clinical Problems and Perspectives.* Baltimore, Williams and Wilkins, 1981, pp. 256-273.

Tobacco consumption is discussed as a form of drug abuse, and the American Psychiatric Association's criteria for tobacco withdrawal and tobacco dependence are mentioned. Both psychological and pharmacological factors can affect smoking behavior, and while nicotine is probably the main reinforcing component of cigarette smoking, it may not be the sole reinforcer. The peripheral effects of nicotine seem to be of less importance in reinforcement than the central effects. There appears to be a withdrawal syndrome that follows quitting smoking. Some heavy smokers may try to regulate plasma nicotine levels by adjusting their smoking behavior. Several types of smoking cessation are discussed, including stopping on one's own, hypnosis, use of other drugs, behavior therapy, and several multifaceted programs. Differences between tobacco consumption and other types of drug dependence are discussed, and the possible effects of lower yield cigarettes are mentioned.

82–1106. Jaffe, J. H.; Petersen, R.; Hodgson, R. **Tobacco—The Devil's Weed.** In: *Addictions: Issues and Answers.* The Life Cycle Series. New York, Harper and Row, 1980, pp. 80-96.

The smoking habit in all its aspects is reviewed. Tobacco was introduced to Europe and Asia after Columbus found it in the New World and took the leaf back to Spain. Tobacco soon became known as a medicinal herb. Since then, despite efforts to prevent it, whether based on economics, religion, criminal punishment, or health, the smoking habit has continued. Aspects of the habit discussed include: why people become and remain smokers; the attractions of nicotine; smoking's relationship to anger and frustration; smoking as an addiction; the problems involved in quitting, and the relapse rates of those who do; the link between cigarettes, cancer, and other health hazards; and the current inefficacy of health education campaigns to wipe out the habit.

82–1107. Kozlowski, L. T. **Psychosocial Influences on Cigarette Smoking.** In: Krasnegor, N. A. (Editor). National Institute on Drug Abuse Research Monograph 26, U.S. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (ADM) 79-882, August 1979, pp. 97-125.

The psychosocial influences which maintain the smoking habit and which deter cessation are reviewed. Personal factors which help propagate cigarette use include extraversion, neuroticism, antisocial tendencies, the internal vs. external locus of control, miscellaneous personality variables, attitudes toward drug taking, smoking typologies, and multiple drug use. Social variables are family and peer pressure, social class and social mobility, and sex roles. The above factors are discussed relative to smoking maintenance and in terms of potential predictors or inhibitors of smoking cessation. General influences on smoking, such as the mass media, economic pressures, and cross cultural perspectives, are also considered.

82–1108. Krasnegor, N. A. (Editor). **The Behavioral Aspects of Smoking.** U.S. Department of Health, Education, and Welfare, Public Health Service, National Institute on Drug Abuse Research Monograph Series No. 26, DHEW Publication No. (ADM) 79-882, August 1979, 199 pp.

Five papers are presented from Part II of the 1979 Report of the Surgeon General on Smoking and Health on the behavioral aspects of smoking, particularly as this relates to the addictive and dependency processes associated with cigarette smoking. The papers discuss: (1) biological influences on cigarette smoking; (2) behavioral factors in the establishment, maintenance, and cessation of smoking; (3) smoking in children and adolescents—psychosocial determinants and preventive strategies; (4) psychosocial influences on cigarette smoking; and (5) modification of smoking behavior. Each contains extensive references. In addition, an introduction has been added which offers an overview of cigarette smoking from an applied behavioral analysis perspective; reviews what is known concerning withdrawal, relapse, and abstinence; and suggests new directions for research. See also 80-1304.

82–1109. Loupekine, F. **Nicotine Rush. (Letter).** *New Scientist* 93(1297): 742, March 18, 1982.

Contrary to an earlier statement (*New Scientist* 93(1293): 459-460, February 18, 1982) that tobacco smokers adjust the number of cigarettes, the frequency of puffing, and depth of smoke inhalation to a dose corresponding to their nicotine addiction, it is suggested that the sudden rush of nicotine craved by smokers can be obtained from low nicotine cigarettes without such adjustments. Also, to alleviate craving for those trying to quit, a low blood level can be obtained from nicotine chewing gum or chewing tobacco. See also 82-0812.

82–1110. Moger, J. **"Dohanyzas Vagy Egeszseg Valasszon"!—Avagy mit Mutat az 1980-as ev Dohanyfogyasztasa Hazankban? [Have Your Choice—Smoking or Health. What Does the Tobacco Consumption of the Year 1980 in Hungary Show?]** *Dohanyipar* (3): 94-96, July-September 1981, Hungarian.

Tobacco consumption in Hungary for the years 1975 to 1980 is summarized, with special emphasis on 1980. Compared with 1975 and 1979, cigarette consumption increased 8.5 percent and 3.2 percent, respectively, in 1980, in spite of price rises. While the awareness of the health effects of smoking has had no effect on the number of smokers, it has had an impact on what people are buying: (1) the proportion of filter products has reached 79 percent; (2) cigarettes with double filters account for 20 percent of total sales; (3) filter Symphonia leads sales with 33.8 percent of total consumption; (4) Sopianae, Helikon, and Marlboro have had dynamic market increases; and (5) the demand for Feeske and Symphonia has decreased significantly. A 2 percent consumption increase is predicted for 1981 as well as a moderate continuation in cigarette purchase modifications. (Auth. Abs. Mod.)

82–1111. Pomerleau, O. F. **Behavioral Factors in the Establishment, Maintenance, and Cessation of Smoking.** In: Krasnegor, N. A. (Editor). National Institute on Drug Abuse Research Monograph 26, U.S. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (ADM) 79-882, August 1979, pp. 47-67.

The complex nature of the behavioral aspects of smoking is examined and the contributions made by experimental behavioral research to the treatment of smoking are evaluated. Two theories, the social learning model and the nicotine addiction model, are discussed in detail, but neither model alone can provide a complete understanding of smoking. A more recent model, the opponent process theory, attempts to link the psychological and physiological factors involved in the maintenance of smoking. These three models are used as the basis of a reexamination of the phenomena of acquisition, perpetuation, and termination of smoking, to develop a context for directing research, improving treatment, and for guiding social policy. The specific areas of recommended research are the phenomenon of withdrawal, the reinforcing effects of nicotine, the role of nicotine antagonists and blockers, and the behavioral pharmacology of cigarette smoking.

82–1112. Silverstein, B.; Kelly, E.; Swan, J.; Kozlowski, L. T. **Physiological Predisposition Toward Becoming a Cigarette Smoker: Experimental Evidence for a Sex Difference.** *Addictive Behaviors* 7(1): 83-86, 1982.

Physiological factors that influence nicotine intake during early smoking experiences of nonsmokers may help determine who becomes a cigarette smoker. In an experiment, college seniors with little smoking experience were randomly assigned by sex to the acidic or alkaline condition by the ingestion of cranberry juice and four 340 mg tablets of glutamic acid hydrochloride or sodium bicarbonate. Following measurement of urinary pH, subjects inhaled eight 2-sec puffs of a Marlboro cigarette (nicotine, 1.1 mg; tar, 17 mg). Subjects willing to make additional attempts to smoke were matched according to pH grouping. A second experiment replicated the first on a group of college freshmen; however, subjects were asked to rinse out their mouths with water and chew a piece of gum following ingestion of the test substances to eliminate aftertaste. Acidic subjects also received a fifth glutamic acid hydrochloride tablet. Results indicated that nonsmoking females whose urine was acidified were more willing, after smoking one cigarette, to volunteer to make additional attempts than were females with alkaline urine. Males did not exhibit this effect. These findings may contribute to the understanding of increases in smoking among adolescent females.

82–1113. Smith, R. **Drinking and Smoking in the Land of the Long White Cloud.** *British Medical Journal* 284(6321): 1029-1031, April 3, 1982.

The smoking and drinking problems of New Zealand are discussed, with special reference to New Zealanders' slow response to the smoking issue. New Zealand does not have a very active antismoking program or active pressure group. Attempts to create a New Zealand version of the British Action on Smoking and Health (ASH) have encountered difficulties; nevertheless, smoking rates have declined. According to the 1976 New Zealand Census, smokers comprised 38 percent of the men and 32 percent of the women. Preliminary analysis of the 1981 census indicated that smokers comprised 35 percent of the men and 29 percent of the women.

82–1114. Svobodova, A. **Zkusenosti Deti Skolniho Veku s Kourenim Cigaret. [Experience of School-Age Children With Cigarette Smoking.]** *Ceskoslovenska Pediatrie* 36(7): 400-402, July 1981, Czechoslovakian.

The smoking habits of schoolchildren aged 5 to 13 were determined by a survey of a representative sample of 2,500 children, their parents, and school headmasters in Czechoslovakia. Although a number of children started smoking after their eighth year, a majority had smoked their first cigarette between the ages of 10 and 13. Boys started smoking earlier than girls, at a ratio of 3 to 2. Over 60 percent of all the children continued smoking after their first cigarette. At the age of 13, 55 percent of the children were occasional smokers (about 10 cigarettes in 3 months), and 6 percent were daily smokers (typically 1 to 2 cigarettes). A high risk of continued smoking was present more often for boys than girls, for children with low scholastic achievement, those whose parents smoked or did not pay attention to their children, undisciplined individuals, and inactive or rather unpopular students. Smoking was found to be highly correlated with the consumption of beer, wine, or spirits. Teaching children in school and at youth organizations about the dangers of smoking, as well as the health education of teachers and of health workers, are suggested to prevent smoking by children and adolescents.

82–1115. Tagliacozzo, R.; Vaughn, S. **Stress and Smoking in Hospital Nurses.** *American Journal of Public Health* 72(5): 441-448, May 1982.

The prevalence of smoking of female hospital nurses working in direct patient care, as measured by a questionnaire sent to the entire nursing staff of the University of Michigan Hospital, was found to be considerably lower than in the U.S. nurse population surveyed in 1975 and in the general female population of the same age range surveyed in 1978. The questionnaire also assessed the association between work-related stress and smoking. Significantly higher scores were obtained for smokers than for nonsmokers on scales concerning the physical and emotional stress of the job and the dissatisfaction with its rewards. The results supported more weakly the association of smoking with the perception of stress induced by role ambiguity, by nurse—doctor conflict, and by conflict between nurses. Some personal and situational variables were found to interact with perception of stress and smoking. (Auth. Abs. Mod.)

82–1116. Urberg, K.; Robbins, R. L. **Adolescents' Perceptions of the Costs and Benefits Associated With Cigarette Smoking: Sex Differences and Peer Influence.** *Journal of Youth and Adolescence* 10(5): 353-361, October 1981.

The perceptions of young adolescents of the costs and benefits of cigarette smoking were examined as a function of the sex of the adolescent and peer smoking habits. The sample consisted of 155 white middle-class male and female adolescents, aged 12 to 15. The results indicate that endorsement of particular costs and benefits was related to the respondent's sex and whether or not the respondent had friends who smoked. The girls seemed to view smoking as a sign of rebellion or autonomy, while the boys seemed to view smoking cigarettes as a social coping mechanism. The effect of having friends who smoke was always mediated by the sex of the adolescents. Boys who have friends who smoke have attitudes that appear more conducive to smoking than do boys with nonsmoking friends. This relationship did not hold for girls. It is suggested that smoking education and intervention be tailored to the sex of the adolescent. Adolescent girls may need assistance in finding safer ways of showing independence and autonomy. Adolescent boys may need to be shown alternatives to smoking for being comfortable in social settings. (Auth. Abs. Mod.)

See also, **82–0946, 82–1006, 82–1117, 82–1201**

SMOKING PREVENTION AND INTERVENTION

82–1117. Atkinson, A. B.; Skegg, J. L. **Anti-Smoking Publicity and the Demand for Tobacco in the U.K.** *Manchester School of Economic and Social Studies* 41(3): 265-282, September 1978.

The conclusion of a 1971 study of the demand for tobacco that "while the effect of antismoking propaganda is measurable and statistically significant, and appears to be increasing, it remains small," is questioned. A reexamination of the data shows that even when correctly stated, these findings are open to serious doubt and different conclusions should be drawn. It appears that publicity had the effect of causing a sudden fall in consumption in 1962, 1965, and 1971, with consumption returning gradually to its previous level. The overall fall was small (around 5 percent) and was accounted for entirely by men, while women's consumption was affected only through increases in taxation. There has been a downward trend in the weight of tobacco consumed which is associated in part with the switch to filter cigarettes and partly with the reduction in their average weight. How far this is attributed to publicity is an open question. Publicity can take many different forms resulting in various effects. The reported results do not necessarily imply that different or more sustained publicity could not have a more significant effect on consumption.

82–1118. Botvin, G. J.; Eng, A. **The Efficacy of a Multicomponent Approach to the Prevention of Cigarette Smoking.** *Preventive Medicine* 11(2): 199-211, March 1982.

A 12-session multicomponent peer leadership smoking prevention program had a significant effect on the prevention of smoking in a study population of 426 seventh grade students. The program focused on the acquisition of basic life skills and the improvement of personal competence, with particular emphasis on coping with social influences to smoke. Experimental and control groups were compared in terms of self-reported smoking status and several knowledge and psychological variables hypothesized to be related to smoking onset. In addition, saliva thiocyanate (SCN) levels were analyzed in a subsample of students. A significant reduction in new smoking was seen in students in the experimental group, based on both the analysis of self-reported smoking status and SCN levels. There also were significant end-of-treatment differences between the experimental and control groups on three of the knowledge variables and two of the psychological variables. At 1-year followup, significantly fewer of the students in the experimental group had become regular (weekly) cigarette smokers compared with those in the control group. These results indicate the short-term effectiveness of a broad-spectrum smoking prevention program that may be applicable to several areas of health education. (Auth. Abs. Mod.)

82–1119. Brink, C.J.H. **Smokers' Rights. (Letter).** *South African Medical Journal* 61(2): 34, January 9, 1982.

Suggestions for nonsmokers are presented to supplement an article which recommends measures for controlling and reducing smoking in South Africa (*South African Medical Journal* 59(18): 617-618, April 25, 1981). Since smokers regard their habit as a "right," nonsmokers should become more assertive to protect their rights to a smoke-free environment. It is recommended that this group form an antismoking league, wear special badges to identify their cause, object on principle when smokers light up in nonsmoking areas, and point to the badge while politely requesting that the offenders refrain from smoking. See 81-1976.

82–1120. Casewit, C. W. **The Stop Smoking Book for Teens.** New York, Julian Messner, 1980, 160 pp.

This straightforward compendium of ideas about smoking is addressed to teenagers, but would be useful for any smoker who wishes to quit. Everything from smoking and pregnancy to the economic savings and other advantages of quitting is covered. All types of cessation programs, including hypnosis and behavioral therapy are reported in detail, and problems such as withdrawal symptoms and rebound eating are discussed. Sections of the book include: (1) why young people smoke cigarettes, (2) what smoking does to you, (3) how to psych yourself to quit, (4) where to get valuable help, (5) hypnosis and "B.T." (behavior therapy), (6) a mix of programs for big (and little) spenders, and (7) quitting forever. A reading list, which includes additional material on the consequences of smoking as well as on specific cessation strategies, is provided.

82–1121. Colletti, G.; Supnick, J. A.; Abueg, F. R. **Assessment of the Relationship Between Self-Reported Smoking Rate and Ecolyzer Measurement.** *Addictive Behaviors* 7(2): 183-188, 1982.

The use of carbon monoxide measurement as a means of validating self-reported smoking data was supported during studies involving smoking reduction. The relationship between self-reported smoking rates and Ecolyzer measurements was assessed in a series of four studies. Factors such as the reactive effects of prior measurement and the time period of administration were examined. In general, a close relationship between Ecolyzer readings and self-reported smoking rates was found. Prior Ecolyzer testing did not appear to reduce the strength of the relationship of Ecolyzer measurement to self-report at subsequent administrations. Thus, carbon monoxide measurement is considered a valid means of corroborating self-reported data. (Auth. Abs. Mod.)

82–1122. Cooper, R.; Soltero, I.; Stamler, J.; Mojonnier, L.; Hall, Y.; Moss, D.; Berkson, D. M.; Tokich, T. **Smoking Cessation in the Chicago Coronary Prevention Evaluation Program.** *Journal of the National Medical Association* 74(4): 349-355, April 1982.

Quit-rates for cigarette smokers in a lifestyle intervention program aimed at reducing coronary risk were 24 percent for all participants and 34 percent for nondropouts. The Chicago Coronary Prevention Evaluation Program (CPEP) recruited 519 coronary-prone male volunteers, originally aged 40 to 59 years, between 1958 and 1968. The primary emphasis was modification of diet (calorie and lipid composition), with smoking cessation deferred until after a period of diet change and weight loss. Recidivism remained very low during participation in the program. Half of the smokers who quit did so after being in the program more than 2 years. The cessation rate increased after the publication of the 1964 Report of the Surgeon General on Smoking and Health, reflecting the change in social attitudes. The specific reasons and circumstances for quitting were not recorded. (Auth. Abs. Mod.)

82–1123. Davidson, S. **Smoking and Alcohol Consumption Advice Given by Health Care Professionals.** *Journal of Obstetric, Gynecologic and Neonatal Nursing* 10(4): 256-258, July/August 1981.

Maternal consumption of tobacco and alcohol can harm infant development. A survey conducted in Vancouver, British Columbia, of 189 women during their 8th month of pregnancy and again 1 month postpartum suggests that health professionals' advice to expectant and nursing mothers is not in accordance with the research evidence. Questionnaire data revealed that only 68 percent (125) of the women received information about smoking during the prenatal period. Of these, 90 percent were advised not to smoke at all. Fewer women (57 percent) received advice about alcohol, with 35 percent being encouraged to quit and 64 percent being told that drinking in moderation was permissible. Indications are that although the majority of women are advised about smoking during pregnancy, and the advice coincides with the research evidence, the same is not true for alcohol. The counsel was most often given by nurses and childbirth educators, with physicians being the next most frequent source.

82–1124. Ellis, L.B.M.; Raines, J. R.; Hakanson, N. **Health Education Using Microcomputers. II: One Year in the Clinic.** *Preventive Medicine* 11(2): 212-224, March 1982.

A personal computer system dedicated to health education was placed in the waiting area of a general medical clinic serving a low-income urban community in south-central Minneapolis. The five health lessons available on this system discuss smoking, weight control, exercise, alcohol consumption, and other health behaviors. Specifically, the lesson "Why Do You Smoke?" determines a cigarette smoker's primary reasons for smoking based on the answers to 18 multiple-choice questions, gives medical reasons to stop smoking, and ways to decrease health risk if the user can't or won't stop. The system was evaluated through two questions asked at the end of every lesson, a survey of clinic staff after 6 and 12 months of operation, and a survey of a sample of clinic patients at the end of the year trial. All lessons were rated as appropriate by 85 percent or more of their raters, with "Why Do You Smoke?" receiving highest honors (96 percent at 6 months and 100 percent at 1 year). Over 1,600 uses were recorded during the first year of operation. The computer has proved to be a cost-effective method of health education in clinical settings acceptable to both patients and staff. A prospective study was recommended to determine whether computer-based health education can make an appreciable difference in health behavior. If such can be demonstrated, a new and very versatile medium can be added to the tools of the health educator. (Auth. Abs. Mod.)

82–1125. Goldberg, M. E.; Gorn, G. J. **Increasing the Involvement of Teenage Cigarette Smokers in Antismoking Campaigns.** *Journal of Communication* 32(1): 75-86, Winter 1982.

An antismoking campaign in which older teenagers (18 years old) worked to discourage smoking among younger teenagers (13 years old) apparently reduced smoking among the older teenagers. Educational material was presented to older teenagers in the first stage of the project. In the second stage, the older teenagers were then requested to act as change agents in trying to alter smoking-related attitudes and behavior of the younger teenagers. Information gathered on the behavior of the older teenagers showed an apparent reduction or cessation of smoking among many of them. The results suggested that the older teenagers were affected by their antismoking involvement with younger teenagers.

82–1126. Goshtautas, A. A.; Rugyavichyus, M. Z. **Izucheniye Rezul'tatov Tselenapravlennykh Meropriyatiy po Bor'be s Kureniyem u Muzhchin Srednego Vozrasta. [Results of Purposeful Anti-Smoking Measures in Middle-Aged Men.]** *Terapevticheskiy Arkhiv* 53(5): 118-121, 1981, Russian.

In the framework of prevention of ischemic heart disease, 131 male residents of Kaunas, aged 40 to 59 years, regular smokers of at least one cigarette/day, were screened for a group education smoking cessation program. After the first 1-hour session, 57 men (43.5 percent of the original sample) agreed to participate in the program. Among those who refused to participate, 32 did so because of lack of motivation to quit. After 3 months, home visits revealed that 19.7 percent of the original sample stopped smoking, 11 percent reduced their smoking by 25 percent or more, and 13.4 percent did not succeed in their attempt to quit smoking. Among the participants in the program, 29.6 percent did not smoke after 3 months vs. 12.3 percent of the nonparticipants. Thus, the percentage of men who discontinued smoking increased by 13.4 as the result of group education. The average number of cigarettes/day decreased significantly for all men in the original sample, but more so in the program participants. The greater effect of antismoking education of the participants in group sessions is attributed to psychological support.

82–1127. Jeffery, R. W.; Danaher, B. G.; Killen, J.; Farquhar, J. W.; Kinnier, R. **Self-Administered Programs for Health Behavior Change: Smoking Cessation and Weight Reduction by Mail.** *Addictive Behaviors* 7(1): 57-63, 1982.

Results of a home correspondence program for either smoking cessation or weight loss showed that the approach produced significant changes in health behavior. Forty smokers and 47 overweight individuals were recruited and assigned to one of four programs for changing their respective health behaviors: (1) a home correspondence program conducted entirely by mail, (2) the mail program with supplemental financial contingency contracts tied to completion of written homework, (3) the correspondence program supplemented both by homework contracts and by regular calls to a telephone answering system, and (4) a standard behavioral group program which acted as a comparison condition. Results showed that all three correspondence approaches produced significant changes in health behaviors equal to the changes experienced by participants in the standard group program. The implication of these findings for development of low-cost programs for disease prevention are discussed. (Auth. Abs. Mod.)

82–1128. King, J.; Eiser, J. R. **A Strategy for Counselling Pregnant Smokers.** *Health Education Journal* 40(3): 66-68, 1981.

Indications are that the counseling strategy described had the effect of reducing smoking among pregnant women who had not quit by their first visit to an antenatal clinic in Bristol, England. A study was made of 300 pregnant women; 150 were counseled and 150 formed the control group who were not counseled. Both groups completed questionnaires at every clinic visit and at delivery. Counseling took place during each clinic visit and in the women's homes, starting 2 months after the initial clinic visit, and at monthly intervals thereafter until delivery. During the initial interview, the project was explained to the women, information about their smoking history was obtained, information about the effects of smoking on fetal health was supplied, advice on how to stop or reduce smoking was given, and an individual strategy was planned with the women. Counseling appeared to be accepted better by primiparous mothers than multiparous ones, and to have more effect in the more personal setting of the mother's home. Actual results of the study are not reported; however, summary data provided indicate that the strategy was effective in either stopping smoking or reducing the amount smoked.

82–1129. Lando, H. A. **Inexpensive Methods for Recruiting Subjects to Smoking Cessation Programs.** *Addictive Behaviors* 7(1): 79-81, 1982.

Results of an investigation to assess the effectiveness of specific publicity techniques in recruiting subjects for smoking cessation programs found that personal contact was important. The study was conducted because researchers in the area of smoking cessation have often been plagued with difficulties in securing adequate subject samples. The specific publicity techniques (e.g., standard news releases, taped public service announcements, posters, letters to employers) in recruiting subjects were used in a metropolitan area of approximately 250,000 residents and in a university community of approximately 40,000 residents. Results were extremely encouraging, with the greatest impact being in notices to employers, accounting for nearly half the total responses. The friend/family category suggested that word of mouth was an important source of information for subjects. Newspaper announcements were effective, but radio and television announcements had less impact than expected. Posters and letters to physicians did not appear to be effective. (Auth. Abs. Mod.)

82-1130. Lando, H. A.; McGovern, P. G. **Three-Year Data on a Behavioral Treatment for Smoking: A Follow-Up Note.** *Addictive Behaviors* 7(2): 177-181, 1982.

Followup data at 12, 18, 24, and 36 months are given for a previous smoking cessation study (*Addictive Behaviors* 6(2): 123-133, 1981, and 7(2): 143-154, 1982). These studies compared two-stage treatment (aversion, maintenance) against three-stage treatment (preparation, aversion, and maintenance) under conditions of either intensive or minimal experimenter contact. One-year followup data indicated the superiority of the two-stage intensive contact procedure. Followup results continue to favor this intervention. Long-term abstinence in this condition consistently exceeded 40 percent as opposed to less than 25 percent in the other conditions. (Auth. Abs. Mod.) See also 82-0569 and 82-1141.

82-1131. New York State Bureau of Drug Education. **Annotated Resource Guide for Alcohol, Tobacco and Other Drug Abuse/Misuse Prevention Education Programs—Secondary Level.** University of the State of New York/State Education Department, Bureau of Drug Education, Albany, 1979, 173 pp.

An annotated guide is provided to help secondary school health education staff (primarily teachers) identify materials and resources for use in planning, developing, and conducting programs for substance abuse prevention/education. It also serves as a source for locating references to augment awareness and understanding of substance use and abuse, and to improve the leadership, communication, and coordination skills needed to reduce alcohol and drug abuse in the schools. Although many more items were reviewed than were chosen for listing, this guide is neither a list of recommended materials nor a list of all the suitable drug education materials available. It simply narrows the field of selection for those who have felt overwhelmed by the task of identifying titles, sources, and ordering information for sound instructional aids in substance abuse prevention education. Topical reviews of materials covering alcohol and alcoholism; tobacco; other drugs and general substance abuse; self-image, values, and skills; and general health education are provided. The section on tobacco includes materials for students, adults, and teachers, as well as materials to help smokers quit.

82-1132. New York State Bureau of Drug Education. **Annotated Resource Guide for Alcohol, Tobacco, and Other Drug Abuse/Misuse Prevention Education Programs—Elementary Level.** University of the State of New York/State Education Department, Bureau of Drug Education, Albany, 1978, 87 pp.

Earlier attempts to design a comprehensive drug abuse curriculum underemphasized attitudinal and behavioral goals. To rectify this situation a more comprehensive curriculum was designed, supplemented by a guide of recommended materials so as to narrow the field of selection and provide guidance for teachers who have felt overwhelmed by the task. Listings of printed materials, audiovisual materials, and regional and community services were selected for this guide based on several assumptions: (1) individual responsibility for one's health should be stressed; (2) materials should aim to develop self-awareness, self-confidence, coping and decisionmaking skills, and ability to handle peer pressure; (3) appropriate information should be given to provide a basis upon which sound choices are made; (4) material should be integrated to involve children in their own learning; (5) programs should use a variety of approaches to meet the needs presented by individual differences; (6) programs should provide for extension into the community through contacts with parents, parent groups, and cooperative efforts with community health agencies and professionals.

82-1133. Orleans, C. S.; Shipley, R. H. **Worksite Smoking Cessation Initiatives: Review and Recommendations.** *Addictive Behaviors* 7(1): 1-16, 1982.

Offering health lifestyle change assistance in the workplace represents a major challenge for behavioral scientists and behavior therapists in the 1980s. Business and industry are showing special interest in employee smoking cessation. A wide range of worksite smoking cessation activities and related research are reviewed and six types of intervention are discussed: educational campaigns, policies restricting workplace smoking, self-help programs, physical examination and physician advice, incentive programs, and actual smoking cessation services. Existing research is critically examined. Practical recommendations for improving and systematically evaluating worksite quit-smoking initiatives are presented. (Auth. Abs.)

82-1134. Pechacek, T. F.; McAlister, A. L. **Strategies for the Modification of Smoking Behavior: Treatment and Prevention.** In: Ferguson, J. M.; Taylor, C. B. (Editors). *The Comprehensive Handbook of Behavioral Medicine. Volume 3; Extended*

Applications and Issues. Jamaica, New York, Spectrum Publications, 1980, pp. 257-298.

A review of current smoking intervention and cessation strategies is presented. The magnitude of the problem of quitting—90 percent of today's smokers have tried or want to quit—and factors controlling smoking behavior are examined. The overview of preventive and interventive programs includes various studies designed to deter the onset of smoking, strategies aimed at adult and general populations, and a variety of social learning and behavioral modification approaches to cessation. The latter category covers self-control strategies, aversion methods, controlled smoking, maintenance of nonsmoking, and programs aimed at multiple risk populations. Methodological issues are discussed, and recommendations for future research and treatments are given.

82-1135. Peltier, B.; Telch, M. J.; Coates, T. J. **Smoking Cessation With Adolescents: A Comparison of Recruitment Strategies.** *Addictive Behaviors* 7(1): 71-73, 1982.

Two methods for recruiting high school students into a voluntary smoking cessation program are discussed. A person-to-person approach was compared to an approach that resembled normal school publicity procedures. Active personal recruitment (normal procedures plus leaflet distribution, in-class announcements, person-to-person recruiting, and peer-assisted recruiting) produced 31 volunteers, while the normal procedures (posters, public address system announcements, and notices in daily bulletins) produced only one. Subsequent application of the active approach at sites where only static procedures had been used produced an additional 21 volunteers. Results suggest that active person-to-person recruitment may be an important component of a smoking cessation effort with high school students. (Auth. Abs. Mod.)

82-1136. Prochaska, J. O.; Lapsanski, D. V. **Life Changes, Cessation and Maintenance of Smoking: A Preliminary Report.** *Psychological Reports* 50(2): 609-610, 1982.

Positive lifestyle changes appear to be inversely related to success in smoking cessation and maintenance. The degree to which positive and negative lifestyle changes in the 6 months prior to treatment were related to initial smoking cessation and to maintenance at a 4-month followup was assessed. Thirty-five participants in a smoking cessation clinic completed a Life Experiences Survey in which the impact of changes were rated from very positive (+3) to very negative (-3). At the completion of the 6-week therapy program, 20 subjects who had initially quit smoking during treatment and 8 who had maintained cessation at 4 months reported significantly fewer positive lifestyle changes prior to treatment. Negative lifestyle changes were unrelated to success. It is speculated that those subjects who have experienced recent positive lifestyle changes will be less motivated to try and attain additional changes such as the cessation of smoking.

82-1137. Raw, M. No Smoking, Please. *UNESCO Courier* (1): 32, January 1982.

Since 1973 there has been a slow but steady decline in tobacco consumption in the United Kingdom. This decline can be attributed in part to the following antismoking activities: (1) health education efforts including publication of research findings, holding of world conferences, and formation of the Health Education Council; (2) legislation, especially the banning of cigarette advertising on TV; (3) nonlegislative pressure on the tobacco and advertising industries which has resulted in voluntary agreements; (4) social control, such as the restriction of smoking in public places; (5) price control, a powerful means of restricting consumption (not yet implemented by the government); (6) smoking withdrawal clinics; and (7) direct advice to patients by health care professionals. Because of the considerable revenue derived from tobacco consumption, stronger government antismoking action is not anticipated.

82-1138. Schneider, L.; Klein, B.; Murphy, K. M. **Governmental Regulation of Cigarette Health Information.** *Journal of Law and Economics* 14: 575-612, December 1981.

Results of a study to estimate the effects of the 1953 and 1964 cigarette health information "shocks," and related government regulations, indicate that consumers have reacted quite vigorously to such information. The primary response to the 1953 report by the American Cancer Society and the British Medical Research Council, which claimed that death rates were significantly higher for cigarette smokers than for nonsmokers, was a movement by cigarette smokers to filter cigarettes with their lower tobacco-per-cigarette content. If changes in the per capita consumption of tobacco rather than in the number of cigarettes is considered, the negative effect of the 1953 report is large, showing more than a 15 percent reduction in consumption by 1964. Further, the 1964 U.S. Surgeon General's Report, which concluded that smoking was causally related to lung cancer, intensified the consumer movement away from tobacco consumption. The cumulative effect of the two health reports had produced a reduction in per capita tobacco consumption of about 25 percent. In addition, by 1971, the antismoking commercials required by the Federal Communication Commission's Fairness Doctrine had further reduced per capita consumption by another 5 percent, for a total reduction of 30 percent. Within this context, Congress imposed a ban in 1971 on all cigarette broadcast advertising. While many previous studies have reported a negative impact on cigarette consumption due to the ban, results here indicated that the ban actually increased consumption. The decrease in advertising expenditures appears to have led to a lower (nonquality-adjusted) cigarette price and hence to increased consumption. Further, the ban increased consumption via the elimination of the antismoking commercials. Finally, the ban substantially increased the cost to firms of introducing new low-tar brands, and the cost to consumers of obtaining information about these newer brands, thus slowing down the movement to these lower-tar cigarettes. (Auth. Abs. Mod.)

82-1139. University of Southern California, Cancer Center Office of Cancer Communications. **Breathe Easy. A Guide to Stop-Smoking Programs in Southern California. (Brochure—Directory).** Los Angeles, University of Southern California Cancer Center Office of Cancer Communications, (Undated), 17 pp.

Organizations offering smoking cessation programs in Southern California are listed in this pamphlet which also includes advice on how to stop smoking and tips from ex-smokers. Information on individual programs includes name of sponsoring organization, address and contact telephone number, type of program, location, length, and cost. Program costs range from free and nominal fees to as much as 500 dollars. Most programs are based on group therapy employing behavior modification, but self-help, aversive therapy, and one-on-one approaches are available. Additional services (smokers hotline and information services) are also listed.

See also, **82-1036, 82-1088, 82-1089, 82-1095, 82-1106, 82-1202**

SMOKING CESSATION METHODS

82-1140. Bradshaw, J. C. **Therapeutic Cigarette-Substitute.** United States Patent No. 4,149,548, April 17, 1979, 5 pp.

A simulated cigarette which overcomes the disadvantages of actual cigarette smoking, while providing psychological advantages to the would-be smoker, is described. The object is constructed of chewable, chemically-resistant plastic, the ends being coated with edible material containing tobacco-like flavors and, preferably, caffeine. In the central mid-part of the cylinder is a septum, rupturable by stress on the article, and containing sodium hydroxide plus hydrochloric acid in separate enclosures. When ruptured, the chemicals inside the compartment would react to produce water and heat sufficient to warm the opposite ends of the simulated cigarette.

82-1141. Lando, H. A. **A Factorial Analysis of Preparation, Aversion, and Maintenance in the Elimination of Smoking.** *Addictive Behaviors* 7(2): 143-154, 1982.

A dismantling strategy for cessation of smoking, in which subjects were assigned to treatment stages (preparation, aversion, maintenance) or to combinations of stages in a factorial design, is discussed. It was hypothesized that multistage conditions would be superior to single stage conditions and that maintenance strategies would retard relapse. Forty men and 33 women were randomly assigned to one of seven conditions. Results supported both hypotheses although significant effects were no longer evident at 12-month followup. Considerable relapse occurred in all conditions, and booster sessions appeared to be ineffective. Continuous reassignment at the various stages disrupted group cohesion, helping to nullify the effects of treatment. Interpretation of the findings is limited, however, by a relatively small subject enrollment in each condition. It is concluded that attempts to isolate extremely precise treatment elements are unlikely to be successful, and further research should focus on more global treatment elements and the importance of group cohesiveness. (Auth. Abs. Mod.)

82-1142. Newman, A.; Bloom, R. **Smoking Reduction: A Comparison of the Effectiveness of Rapid Smoking and Increasing Delay Training.** *Addictive Behaviors* 7(1): 93-96, 1982.

Increasing the time before smoking a lit cigarette was found to facilitate better self-control than rapid smoking according to a study that compared the relative effectiveness of both procedures. One week before treatment, 20 subjects (10 male, 10 female) self-monitored the number of cigarettes smoked. On the days of treatment, subjects were given pre- and postsession self-control tests; subjects were given a lit cigarette and were asked to withstand the temptation of taking a puff during an imposed delay period. After 5 days of 2-hour sessions in which subjects' smoking was paced with either increasing delays or rapid smoking, subjects again were asked to self-monitor their smoking for 1 week. Both groups were not significantly different in the number of cigarettes smoked. However, the findings indicate that increasing delay training resulted in superior self-control on a resistance-to-temptation task than did rapid smoking. (Auth. Abs. Mod.)

82-1143. Pechacek, T. F. **Modification of Smoking Behavior.** In: Krasnegor, N. A. (Editor). National Institute on Drug Abuse Research Monograph 26, U.S. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (ADM) 79-882, August 1979, pp. 127-188.

A review of smoking cessation and behavior modification programs is presented. Problems in methodology, such as the lack of reliability in self-reported cessation and the lack of appropriate followup in many studies, are discussed. An overview of current strategies includes drug treatments, hypnosis, social psychological approaches, and learning/behavior modification methods such as self-control (stimulus control, contingency contracting, and others), and aversion strategies (electric shock, covert sensitization, smoke aversion, satiation). The medical risks of aversive smoking are reviewed, and less stressful alternatives suggested. Multicomponent interventions, some treatment innovations, and the problems involved in controlled smoking are detailed. Final considerations include maintenance of achieved cessation and recommendations for ongoing research, such as maximizing unaided cessation.

82–1144. Prue, D. M.; Krapfl, J. E.; Martin, J. E. **Brand Fading: The Effects of Gradual Changes to Low Tar and Nicotine Cigarettes on Smoking Rate, Carbon Monoxide, and Thiocyanate Levels.** *Behavior Therapy* 12(3): 400-416, June 1981.

When smokers gradually and progressively reduce the tar and nicotine intake of their cigarette, it is demonstrated that there are corresponding decreases in the poisonous gas intake as indicated by decreased carbon monoxide (CO) and saliva thiocyanate (SCN) levels. The effects of progressive reductions in the tar and nicotine content of cigarettes consumed by individuals in a smoking treatment program were examined in nine smokers (five males and four females) by monitoring CO levels, SCN levels, and consumption rate. Reductions were made by changing the smokers' brands of cigarettes every 2 weeks until smokers were consuming cigarettes with 3.0 mg or less of tar and 0.4 mg or less of nicotine. The data show that, although smokers occasionally had higher levels of CO or SCN during brand fading, no smoker sustained increases over baseline levels while smoking cigarettes with less than 3.0 mg tar and 0.4 mg nicotine. The change in mean level from baseline to final brand change condition represents a 46 percent decrease for CO level and a 44 percent decrease for SCN level. Even smokers who showed increased rates of cigarette consumption had decreased levels of these poisonous gases.

TOBACCO PRODUCT ADDITIVES

82–1145. Cline, W. K.; Martin, R. H. **Wrapper for Smoking Articles Containing Magnesium Oxide.** United States Patent No. 4,231,377, November 4, 1980, 5 pp.

A wrapper for smoking articles such as cigarettes, cigars, and the like containing at least 15 percent by weight magnesium oxide or its hydrate and at least 0.5 percent by weight of a specific chemical adjuvant, such as the alkali metal acetates, carbonates, citrates, nitrates, or tartrates, is described. The combination of magnesium oxide or its hydrate with any of the chemical adjuvants significantly reduces visible sidestream smoke that emanates during static burning. The wrapper may comprise conventional cigarette paper with magnesium oxide and the adjuvant incorporated therein as the filler, or either or both of the additives may be applied to the paper as a coating. Wrappers containing the additives can be used in place of conventional wrappers for smoking articles or used as an inner wrapper in combination with a conventional outer wrapping of cigarette paper or cigar wrap. (Auth. Abs.)

82–1146. Greene, R. K.; Geiszler, W. A., Jr. **Post-Treatment of Humic Acid-Dyed Paper.** United States Patent No. 4,238,283, December 9, 1980, 5 pp.

A process to improve the humic acid-treated paper or sheet material in which tobacco or any other smoking product is rolled for the fabrication of cigarettes, cigars, or the like, is described. The paper wrappers are posttreated with magnesium sulfate to fix or render the humic acid insoluble. Paper treated in this manner provides a product of acceptable brown color. Smoking articles wrapped in the treated paper evolve a reduced amount of carbon monoxide on smoking under normal conditions in comparison to currently available brown papers stained with humic acid. (Auth. Abs. Mod.)

82–1147. Hall, J. B.; Sprecker, M. A.; Shuster, E. J.; Schmitt, F. L.; Vinals, J. F. **Use of Substituted Dimethyl Dihydroxy Benzene and Cyclohexadiene Compounds for Augmenting or Enhancing the Taste and/or Aroma of Smoking Tobaccos.** United States Patent No. 4,173,982, November 13, 1979, 17 pp.

A process using one or more of the compounds: 3,6-dimethyl-2,4-dioxocyclohexanecarbonitrile; 2,4-dihydroxy-3,6-dimethylbenzonitrile; and 2,4-dihydroxy-3,6-dimethyl-1-acetophenone combined with other perfume compositions to serve as substitute(s) for "Oak Moss" aroma in the manufacture of tobacco products is described. These compounds are inexpensive and can be prepared from readily available materials. One or more of these substituted dimethyl dihydroxy benzene and cyclohexadiene compounds will create sweet, smoky, woody, and "Oak Moss"-like aromas in tobacco, prior to and during smoking in the mainstream and sidestream smoke. They may be added to filter tip materials, seam paste, and packaging materials.

82–1148. Light, K. K.; Spencer, B. M.; Vinals, J. F.; Kiwala, J.; Vock, M. H.; Shuster, E. J. **Processes for Preparing Cyclohexadiene Derivatives.** United States Patent No. 4,186,149, January 29, 1980, 19 pp.

A process for preparing alkyl-substituted cyclohexadiene derivatives for use as flavor and aroma enhancers in tobacco is described. Preparation includes reacting an alkyl-substituted hydroxycyclohexene of a similar structure (formula illustrated) with mineral acid selected from the group consisting of dilute hydrochloric acid, diluted phosphoric acid, and dilute sulfuric acid, where the alkyl substituent is one to five carbons in length. The products produced by this process are useful for their organoleptic properties in tobaccos and other substances imparting sweet, hay-like, heliotropine-like, black pepper-like, woody, tobacco-like, and "Oriental" aromas and flavors.

82–1149. Mookherjee, B. D.; Wilson, R. A.; Schmitt, F. L.; Vinals, J. F.; Kiwala, J. **1-(2,6,6-Trimethyl-1,3-Cyclohexadien-1-yl)-1,3-Butanedione and Organoleptic Uses Thereof.** United States Patent No. 4,174,286, November 13, 1979, 19 pp.

The organoleptic use of 1-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-1,3-butanedione as an additive for smoking tobacco and other consumable materials is described. Novel tobacco and flavoring compositions having sweet, fruity, berry-like, fresh squeezed fruit (apple and grape) juice-like, hay-like, and tea-like aromas prior to smoking with sweet, hay/tea aromas in the mainstream on smoking and sweet, tobacco-like, and "fresh cigarette pack" aromas in the sidestream on smoking may be provided by 1-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-1,3- butanedione. An aroma and flavoring concentrate containing 1-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-1,3-butanedione and, if desired, one or more other flavoring additives, may be added to the smoking tobacco material or tobacco substitute, to the filter, or to the leaf or paper wrapper. (Auth. Abs. Mod.)

82–1150. Mookherjee, B. D.; Wilson, R. A.; Vock, M. H.; Vinals, J. F.; Kiwala, J.; Schmitt, F. L.; Granda, E. J. **Organoleptic Uses of 3-Hydroxy-1-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-1-butanone.** United States Patent No. 4,235,247, November 25, 1980, 16 pp.

The taste- and aroma-modifying properties of 3-hydroxy-1-(2,6,6-trimethyl-1,3-cyclohexadien-1-yl)-1-butanone are described. Tobacco aromas with rose and grape notes, and hay-like and methane-like undertones are possible. This compound is also useful in smoking tobacco flavoring compositions having sweet, dried fruit-like, floral, and woody aromas prior to smoking, and sweet and dried fruit aroma characteristics in the mainstream and in the sidestream smoke while smoking the tobacco. (Auth. Abs. Mod.)

82–1151. Mussinan, C. J.; Mookherjee, B. D.; Vock, M. H.; Schmitt, F. L.; Granda, E. J.; Vinals, J. F.; Kiwala, J. **Flavoring With a Mixture of Cis-3-hexenal, Trans-2-hexenal, Cis-3-hexenyl Formate, Cis-3-hexenol and Cis-3-hexenyl-cis-3-hexenoate.** United States Patent No. 4,241,098, December 23, 1980, 29 pp.

A method for enhancing the aroma and flavor of tobacco products and foodstuffs with certain cis-3-hexenyl derivatives is presented. The preparation of the derivatives by reacting cis-3-hexenol with pyridinium chlorochromate is described. These compounds are useful in smoking tobaccos, colognes, foodstuffs, chewing gums, toothpastes, medicinal products, and perfumes. In tobacco, a sweet, fruity, cooling green aroma is imparted, prior to and during smoking in the mainstream and sidestream smoke.

82–1152. Mussinan, C. J.; Mookherjee, B. D.; Vock, M. H.; Schmitt, F. L.; Shuster, E. J.; Sanders, J. M.; Light, B. M.; Granda, E. J. **3-(2-Chloroallyloxy)-2(10)-Pinene.** United States Patent No. 4,173,711, November 6, 1979, 46 pp.

Described are processes and compositions for augmenting or enhancing the flavor and/or aroma of consumable materials including chewing tobacco, foodstuffs, chewing gums, medicinal products, toothpastes, perfumes, colognes, and perfumed articles using as the essential ingredient at least one C_{10}-terpene alkyl, alkenyl, haloalkenyl, and alkynyl ether and diether. In treating chewing tobacco, 100 pounds of tobacco are sprayed with a mixture including 0.4 parts by weight of the flavorant composition and redried to a moisture content of 20 percent. On chewing, the resultant tobacco has an excellent, substantially consistent, long-lasting licorice nuance in conjunction with the tobacco note. (Auth. Abs. Mod.)

82–1153. Mussinan, C. J.; Mookherjee, B. D.; Vock, M. H.; Vinals, J. F.; Kiwala, J.; Schmitt, F. L. **Preparation of a Caryophyllene Alcohol Mixture.** United States Patent No. 4,229,599, October 21, 1980, 24 pp.

A process is described for preparing a mixture of isomers of caryophyllene alcohol and dihydrocaryophyllene alcohol which can be used in the manufacture of tobacco products to impart woody, cigar box-like, and oriental aromas prior to and on smoking in both the mainstream and the sidestream smoke. The mixture includes 10,10-dimethyl-2,6-dimethylene bicyclo (7.2.-0)undecan-5-ol produced by isomerizing caryophyllene oxide or dihydrocaryophyllene oxide using an aluminum isopropylate catalyst in a toluene solvent at temperatures in the range of from about 100°C up to about 150°C. The additives may be blended alone or with other flavoring agents in the tobacco (or suitable substitute) or added to the filter, the leaf wrapper, or the paper wrapper. The proportion of flavoring additives may be varied in accordance with taste, although satisfactory results are obtained if the total mixture by weight to smoking material is between 50 ppm and 1,500 ppm. (Auth. Abs. Mod.)

82–1154. Rowsell, D. G.; Spring, D. J.; Hems, R. **Alicyclic Amides Having a Physiological Cooling Effect.** United States Patent No. 4,248,859, February 3, 1981, 15 pp.

A group of alkyl-substituted alicyclic carboxylic acids, esters, and amides, which have a pronounced physiological cooling activity but are without the strong minty odor characteristic of menthol, are disclosed. They have relatively low volatility, and are substantially nontoxic. As additives, they can be incorporated directly into tobacco or filter material, and are effective on the mucous membranes of the oral and nasal mucosa when the tobacco or tobacco-containing article is smoked, chewed, or inhaled.

82–1155. Sanders, E. B.; Houminer, Y. **Smoking Compositions.** United States Patent No. 4,318,418, March 9, 1982, 9 pp.

An invention providing tobacco and nontobacco smoking compositions which contain a pyrazine-containing polyheterocyclic compound as a flavorant additive is described. In one embodiment, tobacco compositions which contain a polyheterocyclic flavorant additive such as 1,2-bis(3,5,6-trimethyl-2-pyrazyl)ethane are provided. Under smoking conditions, the polyheterocyclic additive conditions the mainstream and sidestream smoke, producing a considerably smoother flavor. (Auth. Abs. Mod.)

82–1156. Schreck, R. P.; Light, K. K.; Hall, J. B.; Schmitt, F. L.; Vock, M. H.; Schreiber, W. L.; Vinals, J. F.; Kiwala, J. **Alkyl Esters of 1-Alkanoyl Cycloalkanols, Process for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,327,749, May 4, 1982, 33 pp.

An organoleptically improved smoking tobacco product and additives, and methods of manufacture are described. Sweet, minty, cooling, spicy, anisic, berry-like, fruity, pineapple-like, woody, green, pepper, or dill-like aroma and taste nuances are provided prior to and on smoking in both the mainstream and the sidestream smoke. The nuances created or enhanced may be readily controlled and maintained at the desired uniform level regardless of variations in the tobacco components of the blend. The smoking tobacco materials, or a suitable substitute (e.g., dried lettuce leaves), are processed with an aroma and flavor additive containing as an active ingredient at least one or more 1-acylcycloalkyl alkanoate derivative. In addition, other flavoring and aroma additives may be added to the smoking materials or substituted either separately or in admixture. The smoking material may be shredded, cured, cased, or blended tobacco, reconstituted tobacco, tobacco substitutes (e.g., lettuce leaves), or mixtures. The proportions of flavoring additives may be varied in accordance with taste. The additives may be dissolved in a suitable solvent and the resulting solution sprayed on the tobacco material, or the material may be dipped into the solution. Only a portion of the smoking tobacco or substitute need be treated, which can be blended with other tobaccos before the ultimate tobacco product is formed.

82–1157. Schulte-Elte, K.-H.; Fankhauser, P.; Ohloff, G. **Perfume Compositions Containing Spirane Derivatives.** United States Patent No. 4,252,693, February 24, 1981, 5 pp.

New spirane derivatives useful as odor- and flavor-modifying ingredients in tobacco products are reported. These compounds are also suitable for preparing perfumes, and in the manufacture of artificial flavors in foodstuffs, animal feeds, beverages, and pharmaceutical preparations. The derivatives impart fresh, green, and flowery odors reminiscent of mint leaves, clary sage or black currant, and fruity, green, and woody flavors.

82–1158. Sprecker, M. A.; Hall, J. B.; Schmitt, F. L. **Use of Benzodionones in Augmenting or Enhancing the Aroma and Taste of a Foodstuff.** United States Patent No. 4,241,097, December 23, 1980, 16 pp.

Two bicyclic compounds, 1,4-benzodioxan-2-one and trans-hexahydro-1,4-benzodioxan-2-one, useful for augmenting or enhancing the flavor and/or aroma of consumable materials such as tobacco, are described. Sweet, spicy, vanilla-like, woody, hay, tobacco-like aromas prior to smoking and sweet, spicy, hay, tobacco-like aromas on smoking are provided both in the mainstream and sidestream smoke. The additives may be added to the smoking tobacco material, to the filter, or to the leaf or paper wrapper. The proportions may be varied in accordance with taste, but satisfactory results have been obtained with a total proportion by weight of between 250 ppm and 1,500 ppm.

82–1159. Sprecker, M. A.; Sanders, J. M.; Schreiber, W. L.; Watkins, H.; Vinals, J. F.; Shuster, E. J.; O'Rourke, T. J.; Hagedorn, M. L.; Klemarczyk, P. **Bridged Ketones and Process for Preparing Same.** United States Patent No. 4,250,338, February 10, 1981, 33 pp.

The generic structure and processing of tricyclic ketone derivatives, used for aroma enhancement of smoking tobacco and other compositions, is described. The process can be used to augment, enhance, or impart sweet, floral, woody, spicy, leathery, and amber aroma nuances to smoking tobacco prior to smoking, and sweet aromas with natural tobacco taste and aroma nuances upon smoking in the mainstream and in the sidestream smoke. It can also be used to impart, augment, or enhance low-keyed oily, woody, amber, leathery, warm spice, earthy camphoraceous, patchouli-like, balsamic-like, green, cardamom-like, vetiver-like, sweet woody, or minty nuances in or to perfumed compositions or perfumed articles or colognes. (Auth. Abs. Mod.)

82–1160. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Use of 2-Oxabicyclooctane Derivative in Augmenting or Enhancing the Aroma or Taste of Smoking Tobaccos and Smoking Tobacco Articles.** United States Patent No. 4,320,771, March 23, 1982, 21 pp.

Described is the use of a derivative of a 2-oxabicyclo (2.2.2)octane which is 1,3,5,5-tetramethyl-2-oxabicyclo (2.2.2)octane for augmenting or enhancing the aroma or taste of smoking tobaccos and smoking tobacco articles. The substance imparts sweet, earthy, cooling, citrus-like aroma and taste nuances before and during smoking in the mainstream and sidestream smoke. The substance can also be used for other consumable materials, including perfumes, perfumed articles, colognes, foodstuffs, chewing gums, toothpastes, and medicinal products. (Auth. Abs. Mod.)

82–1161. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **2-Oxabicyclooctane Derivatives, Processes for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,242,269, December 30, 1980, 30 pp.

A process using 2-oxabicyclooctane derivatives for flavor and aroma enhancement in smoking tobacco and other consumable materials is described. The oxabicylooctane derivatives augment or enhance the clove bud-like characteristics of smoking tobacco by imparting thereto a clove bud aroma and taste nuance prior to and on smoking in the mainstream and in the sidestream smoke. An aroma and flavoring concentrate containing one or more of the oxabicyclooctane derivatives and, if desired, one or more other flavoring additives may be added to the smoking tobacco material or tobacco substitute, to the filter, or to the leaf or paper wrapper, or to a filter which is part of the smoking article. This patent is part of a series involving the use of 2-oxabicyclooctanes. (Auth. Abs. Mod.)

82–1162. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **2-Oxabicyclooctane Derivatives, Processes for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,250,342, February 10, 1981, 31 pp.'

Derivatives of 2-oxabicyclo(2.2.2)octanes are described, for possible use as flavor/aroma additives. Their precursors, the cyclohexenemethanols, and their organometallic salts are included. These compounds are useful in augmenting the flavors and/or fragrances of such consumable materials as smoking tobaccos, perfumes, perfumed articles, colognes, foodstuffs, chewing gums, toothpastes, and medicinal products by adding a small amount of at least one of the compounds described. The clove-like characteristics of smoking tobacco are enhanced by imparting a clove bud aroma and taste nuance in the mainstream and in the sidestream smoke. This patent is part of a series involving the use of 2-oxabicyclooctanes. (Auth. Abs. Mod.)

82–1163. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **2-Oxabicyclooctane Derivatives, Processes for Preparing Same and Organoleptic Uses Therefor.** United States Patent No. 4,222,941, September 16, 1980, 31 pp.

Derivatives of 2-oxabicyclo(2.2.2)octanes are described, for possible use as flavor/aroma additives. Their precursors, the cyclohexenemethanols, and their organometallic salts are included. These compounds are useful in augmenting or enhancing the flavors and/or fragrances of such consumable materials as smoking tobaccos, perfumes, perfumed articles, colognes, foodstuffs, chewing gums, toothpastes, and medicinal products by adding a small amount of at least one of the compounds described. The oxabicyclooctane derivatives presented augment or enhance the clove bud-like characteristics of smoking tobacco by imparting a clove bud aroma and taste nuance upon smoking in the mainstream and sidestream smoke. This patent is part of a series involving the use of 2-oxabicyclooctanes. (Auth. Abs. Mod.)

82–1164. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Cyclohexene Methanol Derivatives in Perfume.** United States Patent No. 4,234,463, November 18, 1980, 19 pp.

The derivatives of 2-oxabicyclo(2.2.2)octanes, their organoleptic properties, and their precursors (cyclohexene methanols) used as tobacco flavorants, are described. Also examined are their organometallic salts. These compounds are also useful in perfumery, foodstuffs, and medicinal product flavors. The oxabicyclooctane derivatives impart to tobacco a clove bud-like aroma and taste nuance prior to and during smoking in both the mainstream and sidestream smoke. These flavorants may be added to tobacco or tobacco substitutes and incorporated into materials such as seam paste, filter tip, and packaging materials. This patent is one of a series utilizing oxabicyclooctane derivatives as tobacco additives.

82–1165. Trenkle, R. W.; Mookherjee, B. D.; Hall, J. B.; Kasper, R.; Vock, M. H.; Schreck, R.; Granda, E. J.; Vinals, J. F. **Process for Preparing Acyl Trimethyl Cyclohexene Derivatives and Use of Intermediates Therefor in Augmenting or Enhancing the Aroma or Taste of a Consumable Material.** United States Patent No. 4,250,332, February 10, 1981, 33 pp.

A process for preparing β-damascone is described, starting with 2,6,6-trimethyl cyclohex-2-enone, and proceeding according to a specified reaction sequence. The organoleptic properties of the compound are also detailed, being useful in augmenting or enhancing the taste or aroma of a consumable material, e.g., chewing tobacco, smoking tobacco, foodstuff, chewing gum, medicinal product, perfume, perfumed article (e.g., drier-added fabric softener articles and solid or liquid anionic, cationic, or nonionic detergents), and cologne. (Auth. Abs. Mod.)

82–1166. Trenkle, R. W.; Mookherjee, B. D.; Kasper, R.; Vock, M. H.; Vinals, J. F.; Kiwala, J.; Schmitt, F. L. **1-Acyl-2,6,6-Trimethylcyclohexene Derivatives, Processes for Producing Same, Intermediates Therefor and Organoleptic Uses Thereof.** United States Patent No. 4,172,850, October 30, 1979, 33 pp.

A method for producing organoleptically improved smoking tobacco product and additives is described. The process overcomes problems in providing musty-floral, slightly sweet, fruity, damascenone-like aroma and taste nuances prior to smoking, and sweet, Virginia tobacco-like notes on smoking in the mainstream and sidestream smoke. These nuances are created, enhanced, and can be readily controlled and maintained at the desired uniform level regardless of variations in the tobacco components of the blend. The method comprises an aroma and flavor additive containing as an active ingredient at least one 1-acyl-2,6,6-trimethylcyclohexene derivative prepared according to the process is added to tobacco materials or substitutes (dried lettuce leaves). In addition, other flavoring and aroma additives may be added to the materials either separately or in admixture with the one or more 1-acyl-2,6,6-trimethylcyclohexene derivatives.

82–1167. Winter, M. **Heterocyclic Derivatives as Flavoring Agents.** United States Patent No. 4,262,030, April 14, 1981, 11 pp.

The use of 1,3-oxathiane and 1,3-oxathiolane derivatives as tobacco flavor and aroma enhancers is described. These compounds are also recommended in the manufacture of artificial flavors, foodstuffs, animal feeds, beverages, pharmaceutical preparations, and perfumes. The derivatives described are suitable for use in natural tobacco and tobacco substitutes, for smoking in pipes, cigars, and cigarettes, and for chewing or for snuff. Depending on the nature of the compositions to which they are added, the compounds impart flavors such as fruity, green, alliaceous, caramel-like, or slightly burnt and rubbery notes.

82–1168. Yoshida, T.; Hall, J. B. **Process for Preparing Ketones Using Zinc Acetate Condensation Catalysts, Products Produced Thereby and Organoleptic Uses of Same.** United States Patent No. 4,169,109, September 25, 1979, 31 pp.

A process for the production of alpha, beta unsaturated ketones as tobacco flavorants by reaction of an aldehyde with a ketone in the presence of a catalyst consisting essentially of either zinc acetate or zinc acetate dihydrate is described. The ketones obtained are suitable for flavor and aroma enhancement in smoking tobacco and tobacco substitutes, as well as in other consumable materials. The invention may provide or enhance Virginia-type tobacco aroma and taste nuances. Other flavor and aroma additives may also be used, separately or together with the ketone(s). An aroma and flavor material which contains the ketone(s), as well as one or more of the other additives if so decided, may be added to smoking tobacco, the filter, or the leaf or paper wrapper. (Auth. Abs. Mod.)

TOBACCO MANUFACTURING AND PROCESSING

82–1169. Baxter, S.; Cuangani, P. D. **Filter Elements.** United States Patent No. 4,232,130, November 4, 1980, 18 pp.

A polyolefin filter for tobacco products and the like is described. The extruded element is an open-cell foamed polyethylene or polypropylene having a hardness greater than 50 percent, a ΔP_{10} less than 15 centimeters water gauge, and a TPM filtration efficiency greater than 20 percent. The filter rod can be made by extruding polyethylene containing less than 0.2 moles of a volatile blowing agent per 100 grams of resin at a melt temperature between 130°C and 150°C, the temperature of the die being between 120°C and 136°C. Higher melt temperature and die temperature are required for polypropylenes. (Auth. Abs. Mod.)

82–1170. Bolt, R. C. **Apparatus for Perforating a Moving Web.** United States Patent No. 4,219,727, August 26, 1980, 11 pp.

In this ventilated cigarette-making system, the tipping paper is perforated before assembly of the cigarettes using a spark perforator which is controlled by a feedback arrangement, in response to the resultant air permeability of the finished cigarettes, or the permeability of the tipping paper as monitored before assembly. Preferably the testing of the finished cigarettes is done in a modified Molins "CID 4" cigarette testing device. The averaged test signal is used to control the frequency of power supply to the spark perforator, whose electrodes are loaded with capacitors so that the change in frequency results in a change in the current flow through the electrodes, and thus the size of the holes produced. (Auth. Abs.)

82–1171. Calleson, D. A.; Sigmon, N. A. **Apparatus for Measuring Smoke Dilution in a Vented-Filter Cigarette.** United States Patent No. 4,171,635, October 23, 1979, 10 pp.

An invention which provides a means of measuring the degree of smoke dilution caused by air ventilation in a vented-filter cigarette is described. The process is comprised of relatively simple components. As a puff generator draws a volume of smoke and air through the vented-filter cigarette, air is drawn from a ventilation cylinder to cause a piston to travel to a dead center position. The amount of movement of this piston is a measure of an amount of air drawn through the ventilation path of the filter by the puff generator. The amount of travel can be related to the total possible stroke of the piston so as to produce a direct digital read-out of the degree of ventilation. (Auth. Abs. Mod.)

82–1172. Cartwright, W. F.; Cornell, A.; Higgins, D. B.; Mikkelson, R. P. **Filter Cigar.** United States Patent No. 4,243,053, January 6, 1981, 8 pp.

A manufacturing process for a filter-tipped large cigar is described. The process comprises a filtered large cigar having an outer cigar wrapper and an inner cigar binder, both spirally wrapped about a circumferentially shaped preformed filter butted tightly against a tobacco filler charge. A reinforcement strip, bonded to the binder, includes a layer of high wet strength reinforcing tape bonded to flame-extinguishing foil and circumferentially wrapped over the interface between the filter and the filler charge to prevent separation of the filter and the filler charge and to extinguish the flame prior to ignition of the filter. The tobacco filler charge may be virtually any conventional large cigar tobacco filler charge and preferably consists principally of tobacco and tobacco blends. The tobacco filler charge may be treated and additives added to enhance its burn, taste, and aroma properties. Additives may also be added to the tobacco filler material to enhance its taste and aroma characteristics. (Auth. Abs. Mod.)

82–1173. Gretz, H.; Rudszinat, W.; Lorenzen, H.-C. **Method and Apparatus for Perforating the Envelopes of Filter Cigarettes or the Like.** United States Patent No. 4,249,545, February 10, 1981, 21 pp.

A manufacturing process designed to provide the desired quantity of air flow through cigarettes is described. The making of perforations is regulated automatically in dependency on one or more variables including (1) the resistance of the filler to axial flow of a gaseous fluid, (2) the permeability of the entire tubular envelope, (3) the resistance which the entire filler or the tobacco filler of a filter cigarette offers to flow of a gaseous fluid or to penetration of radiation across successive increments of the filler, (4) the resistance which successive increments of a web of cigarette paper offer to the penetration of air or light, and/or (5) the quantity of tobacco particles in the smokers' products. The perforations are formed by laser beams, and the regulating step includes changing the intensity of laser beams and/or the length of intervals of impingement of laser beams upon the envelopes. (Auth. Abs. Mod.)

82–1174. Hall, F. V. **Apparatus and Method for Providing a Cigarette Filter With an Aeration Groove.** United States Patent No. 4,232,574, November 11, 1980, 8 pp.

An apparatus and method for altering the filter of a cigarette with an annular peripheral groove to enable air to pass directly into the fibrous body of the filter material from apertures in the tipping paper is described. Ordinary plug wrap paper, instead of a high porosity tea bag paper, is used to wrap the fibrous filter material. The annular groove can be formed by a pair of rotating knives and a rotatable spacer with radial cutting edges which rout out material from each filter plug during conveyance on an alignment drum. Alternatively, a plurality of closely spaced rotating knives which cut through the plug wrap paper only may be used to form a series of closely spaced annular grooves or slits in the filter periphery. (Auth. Abs. Mod.)

82–1175. Hardwick, M. J.; McMeekan, K. R. **Smoking Product.** United States Patent No. 4,326,544, April 27, 1982, 4 pp.

A method of making a fuel for use in a tobacco substitute is described, in which a three-dimensional needled felt web of viscose fibers is subjected to controlled pyrolysis until the organic residue contains at least 90 percent carbon by weight. The resulting felt web, after loading with combustion-modifying agents and volatile ingredients, is divided into pieces for smoking. The processing of the viscose fibers in the form of a felt web has a number of advantages: the fibers are particularly cohesive in the felt and thus material-handling problems are reduced; the three-dimensional lay of the fiber gives good resilient resistance providing good filling capacity with an acceptable hardness and pressure drop; a significant reduction in carbon weight per standard cigarette is produced; and ashing characteristics are improved. The web also provides a convenient form for application of the various additives during pre- and post-pyrolysis treatment. Such fillers or additives, or their precursors, may include carriers for the volatile agents, catalysts for the pyrolysis, and combustion-modifying agents. Subsequent to the pyrolysis, further additives may be applied to the pyrolysed felt, particularly by spraying on or by passing the felt through a dip. Such subsequently applied additives may include combustion modifying agents, nicotine, smoke-producing agents, and flavoring agents.

82–1176. Higgins, D. B.; Godfrey, F. D., Jr. **Method and Apparatus for Manufacturing Filter Cigar.** United States Patent No 4,256,124, March 17, 1981, 21 pp.

A method and an apparatus are described for the manufacture of a filtered large cigar. Steps in the process include simultaneously introducing preformed cigar filters, tobacco filler material, and a sheet of cigar binder material onto the rolling table of a cigar-rolling machine. A reinforcement strip including layers of reinforcing tape and a nonflammable foil is also introduced onto the rolling table. In this manner, during the subsequent rolling operation, the reinforcement strip becomes circumferentially wrapped about and bonded to the binder over the interface between the filter and filter material. Depending on the relative placement on the table of the reinforcement strip and binder, the reinforcement strip will be positioned either over or under the binder. Upon completion of the rolling operation, the filter is tightly located against the filler charge, and the bunch is then crimped. The crimped bunch is then wrapped in either a natural leaf or a manufactured cigar wrapper. (Auth. Abs.)

82–1177. Johnson, R. R. **Cigarette Filter.** United States Patent No. 4,256,122, March 17, 1981, 4 pp.

A process to improve air ventilation of cigarette filters and cigarettes by means of flow-directing grooves is described. The grooves direct ventilating air either to the tobacco end of the filter or to the mouth end of the filter or a combination thereof. The filter includes a porous filter rod, circumscribed by a nonporous or smoke-impervious wrapper which provides the filter rod with grooves extending a preselected distance longitudinally. Tipping material circumscribes the nonporous wrapper and is provided with flow-through openings in communication with the grooves. The grooves may be oriented to extend to the mouth end of the filter when connected to a cigarette, to the tobacco end of the filter when connected to the cigarette, or to nonconnecting grooves which extend from each end of the filter element to a preselected distance. (Auth. Abs. Mod.)

82–1178. Keith, C. H. **Particle Size Studies on Tobacco Smoke.** *Beitraege zur Tabakforschung International* 11(3): 123-131, January 1982.

A cyanoacrylate fixation technique was applied directly to cigarette smoke to evaluate the effects of dilution and filtration on particle size. Undiluted smoke has particles ranging from mainly <0.1 to a few at >0.5 micrometers. The median and mass median diameters are 0.20 and 0.32 micrometers for an unfiltered middle puff. The smoke was treated by injection of cyanoacrylate vapor, collection of a sample of fixed particles on membrane filters, and image analysis to separate, size, and count particles, followed by aging of undiluted particles for 1.4 seconds to double the number and mass median diameters and decrease particle concentration. It was determined that (1) different puffs on unfiltered cigarettes have little effect on size distribution and particulate concentration, (2) filtration removes both larger and smaller particles and tends to skew distribution to smaller sizes, (3) reduction in particulate concentration in filtered smoke is as expected from nicotine removal efficiencies, (4) filter dilution skews distribution to larger sizes, while tobacco column dilution has little effect, and (5) particulate concentration correlates well with overall dilution levels. (Auth. Abs. Mod.)

82–1179. Keritsis, G. D. **Method for Denitration of Tobacco Employing Electrodialysis.** United States Patent No. 4,253,929, March 3, 1981, 11 pp.

A process for denitrating tobacco with minimal losses of desirable tobacco solubles employing electrodialysis is disclosed. The process comprises subjecting an aqueous tobacco extract to membrane electrodialysis such that nitrate salts are extracted, without substantial loss of desirable tobacco solubles. The denitrated extract when combined with denitrated insoluble fibrous tobacco materials may be employed to produce tobacco products having reduced nitrogen oxide delivery. (Auth. Abs.)

82–1180. Kiefer, J. E.; Mumpower, R. C., II. **Hot Melt Adhesive for Bonding Filter Tow, and Filter Elements Bonded Thereby.** United States Patent No. 4,221,226, September 9, 1980, 5 pp.

A hot melt adhesive for textile tow, particularly for textile tow from which tobacco smoke filter rods are manufactured, is described. Included in the description is a tobacco smoke filter element with fibers which are bonded together at their contact points with the hot melt adhesive. The properties of this adhesive include a very low viscosity and very low tackiness while in the molten state, but a very good adhesion to either crystalline or amorphous fibers at ambient temperatures. In this manner, tobacco smoke filters may be made from crimped textile tows of either crystalline or amorphous fibers, such as those from cellulose acetate, poly-(ethylene terephthalate), polypropylene, and polyethylene polymers. (Auth. Abs. Mod.)

82–1181. Koch, F.-P.; Pinck, P.; Petersen, K.-E.; Lange N.; Koehler, E.; Bornfleth, U. **Apparatus for Perforating Cigarette Paper or the Like.** United States Patent No. 4,265,254, May 5, 1981, 12 pp.

A means of perforating wrapping material or the tubular wrappers of discrete filter cigarettes is described, for either unit length or double unit length in a filter tipping machine. The apparatus may also be used in perforating wrappers of plain cigarettes, plain or filter-tipped cigars, cigarillos, or for the wrappers of filter mouthpieces. The invention has a laser which is energizable at a frequency depending on the speed of the main prime mover of the machine. A beam is emitted which is guided by a pivotable reflector to impinge alternately upon different portions of a single optical system, or upon several discrete optical systems, which focus the beam upon the wrapping material or wrappers. The beam forms two or more rows or patterns of perforations in the web, or wrappers of successive cigarettes. The pivotable reflector is optional if a diffraction grating is placed in front of a single optical system. The grating splits the beam into several discrete beams, each of which is focused upon a different portion of the web or of the wrapper of a cigarette. (Auth. Abs. Mod.)

82–1182. Ligeti, E. C. **Tobacco Smoke Filter.** United States Patent No. 4,254,782, March 10, 1981, 6 pp.

An improved tobacco smoke filter with a novel arrangement of restricted passageways and expansion chambers is described. The filter entrains the smoke so that greater diffusion occurs in a first expansion chamber and greater and more efficient mixing with the ambient air occurs in a second expansion chamber than was previously possible. With this filter, more tar and nicotine are removed and greater cooling of the smoke is provided. Indexing and indicating means are provided on the device so that the user may easily select the amount of ambient air desired for admission into the second expansion chamber. (Auth. Abs.)

82–1183. Martin, J. R.; Sides, L. W. **Smoking Product and Process for Manufacturing Same.** United States Patent No. 4,326,543, April 27, 1982, 7 pp.

A manufacturing process for filter-tipped cigarettes, cigars, and similar smoking products which employs self-supporting nonwrapped fibrous filters is described. In this process the tobacco-containing portions are attached to the fibrous filters by tipping paper that is coated in a specified manner with a hot-melt adhesive. The tipping paper is normally substantially nonporous and the means for permitting the ingress of air comprises perforations located adjacent to the filter. The hot-melt adhesive is composed of an ethylene/vinyl acetate copolymer, a wax, and a polymeric resin derived from styrene, and/or a styrene derivative, with the adhesive activated at temperatures within the range of 60°C to 110°C. (Auth. Abs. Mod.)

82–1184. Miano, R. R.; Keith, C. H. **Smoking Material.** United States Patent No. 4,233,993, November 18, 1980, 4 pp.

A tobacco substitute exhibiting lower particulate matter yield upon combustion and a pressure drop resulting in a more smokable column are described. This is achieved by incorporating a "blowing agent" into the matrix material. Compounds which will, upon heating, produce a gas by vaporization or gaseous decomposition are designated as blowing agents. The matrix material, blowing agent, combustion modifiers, and other additives are added to water and formed into a film. The wet film is then heated to a temperature sufficient to cause gasification of the blowing agent. The resultant film has a porous nature and a burning rate analogous to that of tobacco.

82–1185. Nienow, J. F.; Irving, C. L.; Shaw, L. V. **Measuring Cigarette Characteristics During Pyrolysis Smoking.** United States Patent No. 4,227,400, October 14, 1980, 14 pp.

An invention relating to the automatic measurement of cigarette rod firmness and length, and more particularly to the measurement of rod firmness behind the coal of a burning cigarette, is described. It automatically feeds, mounts, smokes cigarettes of given blends, and measures the firmness of the cigarettes before they are lit as well as during smoking while the firmness is reflecting the effects of heat, blend, and moisture on the tobacco column. It also measures the remaining length of the burning cigarette so as to provide a firmness value for a particular blend for a given length of the cigarette smoked. A feed hopper that stores a large supply of cigarettes to be automatically advanced for smoking and testing is included. In addition, the apparatus has a self-zeroing feature in which a measuring foot and a reference foot are automatically disposed against the cigarette under test. The firmness is determined by the difference in the depth to which the measuring foot deflects the wrapper beyond where the reference foot has come to rest. (Auth. Abs. Mod.)

82–1186. Rainer, N. B.; Bailey, C. V. **Cigarette Filter Material Comprising Compounds of Iron in High Oxidation States.** United States Patent No. 4,246,910, January 27, 1981, 4 pp.

A tobacco filter material capable of removing cyanides, particularly hydrogen cyanide, from cigarette smoke is described. The inert gas-permeable granular support materials are impregnated with alkali ferrates comprised of iron compounds having an oxidation state of +4, +5, or +6. When they are used as a granular filter material in combination with a tobacco product, hydrogen cyanide can be significantly reduced in the smoke obtained from the tobacco product. Activated carbon or alumina impregnated with sodium permanganate and a basic sodium compound may also be combined with the granular filter material. The permanganate-alumina composition, when blended with the ferrate filter material, is especially effective in reducing the amount of nitrogen oxides present in tobacco smoke. Still other materials generally known in the cigarette filter art may be utilized to either selectively remove other components of the smoke, or flavor compounds may be used in combination with the filter material. (Auth. Abs. Mod.)

82–1187. Sawada, H.; Kotani, M.; Sato, K.; Izawa, T.; Sugimori, K.; Katanosaka, A. **Smoke Filter Material and Use Thereof.** United States Patent No. 4,246,009, January 20, 1981, 5 pp.

Smoke filter material which has a superior capacity for removing injurious gaseous, liquid, and solid components from smoke is described. The material is especially efficient for tobacco smoke, with a high rate of removal of injurious components contained therein, especially nicotine and tar, as well as minute solid particles and acetaldehyde in the gaseous phase. This is accomplished without adversely affecting the flavor and taste of tobacco. The filter material is composed of a fiber matrix and a water-swellable synthetic mica. (Auth. Abs. Mod.)

82–1188. Siren, M. J. **Filter Material and a Method of Manufacturing and Using the Same.** United States Patent No. 4,242,226, December 30, 1980, 6 pp.

A manufacturing process for a filter material in the form of a matrix of activated carbon is described. At least one metal taken from the group Ca, Mg, Ba, Al, Cu, and the transition metals are dispersed throughout the filter material. These are obtained by reacting cations of these metals with anionic groups bound to a polyhexose derivative. For a material particularly suitable for filtering tobacco smoke, calcium cations are used in a quantity that is at least approximately 10 percent by weight, and preferably approximately 15 percent by weight. The resulting filter material will selectively remove cadmium from tobacco smoke. A conversion of carbon monoxide to carbon dioxide with the oxygen present in the smoke is achieved catalytically when, in addition to the calcium salt, Fe-cations and/or Cu-cations are used in a quantity that is at least one-tenth of the Ca-content. The Fe and Cu are conveniently incorporated in oxide or chloride form. (Auth. Abs. Mod.)

82–1189. Smith, E. A. **Cigarette Holder.** United States Patent No. 4,267,849, May 19, 1981, 6 pp.

An improved cigarette holder, which has a built-in retrieving member with a hook on one end and a tamping handle on the other for removing and replacing filtering material (absorbent cotton), is described. Replacing filter material can be costly (using discardable holders or manufactured filter elements), or inconvenient (replacing cotton in holder can be untidy work). This easily disassembled holder provides a means for conveniently inserting and removing the filtering material. The plastic material from which the cigarette holder is formed can be translucent or transparent, so the smoker can observe the tar accumulated within the filtering material. This may act as a deterrent to continued smoking as well as giving an indication as to the need for replacing the filtering material.

82–1190. Tamaoki, A.; Tanaka, S.; Tomioka, S.; Kunimune, K. **Tobacco Filters and Method for Forming Same.** United States Patent No. 4,261,373, April 14, 1981, 9 pp.

A method for producing molded tobacco filters characterized by a plurality of individual fiber bundles, consisting of both twisted and unstructured polymer fiber bundles, is described. These filters, which are useful in providing various preferable effects such as improvement in pressure drop and balanced adsorption of smoke component, consist of a first and second component by themselves, or a mixture of at least 20 percent by weight of the described fibers and another kind of fiber, so as to give a variety of cross sections to the fiber bundles. These first and second components, having different melting points, by 10°C or more, are subjected to a heat treatment at a temperature lower than the melting point of the first component but higher than the melting point of the second, to stabilize the structure by self-adhesion. The multibundle structure is then cooled and cut into filter plugs.

82–1191. Utsch, F. V. **Process for the Expansion of Tobacco.** United States Patent No. 4,235,250, November 25, 1980, 11 pp.

An improved process for the expansion of tobacco, which employs carbon dioxide as the expansion agent, is presented. Tobacco puffing or expansion procedures have been described as a means fo. reducing the "tar" content by reduction in the delivery of smoke. The process involves (1) contacting tobacco with carbon dioxide gas at a pressure of at least 250 psig for a time sufficient to impregnate the tobacco with the carbon dioxide gas to form a gaseous carbon dioxide-tobacco system; (2) releasing the pressure; and (3) subjecting the carbon dioxide-treated tobacco to rapid heating conditions to remove the carbon dioxide and thereby expand the tobacco. The improved process consists of cooling the gaseous carbon dioxide and tobacco system in step (1) to a temperature close to the saturation temperature of carbon dioxide but not lower than -23°C. (Auth. Abs. Mod.)

See also, **82–1140**

TOBACCO ECONOMICS

82–1192. Burry, R. D. **1981 Strong Tobacco Production Year; Usage Gains, Particularly Smokeless.** *Tobacco International* 184(4): 10-12, 14, 16, 18, 21, February 19, 1982.

Tobacco equities showed a substantial upward revaluation during 1981. The strongest performer was United States Tobacco (40 percent increase relative to the market), followed by Philip Morris (21 percent). Demand slackened after the September price increase, though total units received by retailers for the entire year rose by 1.0 percent. Domestic cigarette consumption continues to expand at a pace slightly ahead of the overall population growth. The fastest expanding parts of the market are the two moist smokeless lines, fine-cut chewing tobacco and snuff. Retail cigarette prices rose 8.0 percent, just below the consumer price index. Domestic advertising expenditures accelerated sharply as manufacturers competed for positions in the low-tar sector. Cigarette production and shipments, price trends, and nontobacco outlays are also discussed.

82–1193. Feinschreiber, R. **Tax Incentives for Exports of Tobacco Products.** *Tobacco International* 184(4): 52, 54, 57, February 19, 1982.

Tobacco product manufacturers can begin saving tax dollars by using a Domestic International Sales Corporation program (DISC), a tax incentive for exports, and a DISC subsidiary known as a Foreign International Sales Corporation (FISC). Manufacturers who participate in the DISC program acquire an extended period of time to pay taxes on export profits. Two major benefits of DISC are the grouping and ungrouping of transactions and the use of FISC. Although relatively few companies have set up a FISC, it does provide three major benefits for a manufacturer of tobacco products: increases the amount of income eligible for a tax deferral, increases the duration of the tax deferral, and enables DISC to meet its annual assets test.

82–1194. Hall, R. **Future Prosperity Hinges on Flue-Cured Exports.** *Tobacco Reporter* 109(4): 55-56, April 1982.

If the U.S. tobacco industry is to survive and profit during the eighties, the United States must gain a greater share of the expanding foreign market. Although cigarette production has continued to increase since 1977, consumption of flue-cured tobacco has declined each year. This is due to the switch to filter cigarettes, and decreasing tar and nicotine levels in cigarettes which use less flue-cured tobaccos. Increasing prices relative to other tobaccos have also contributed to the shrinking market share. Domestic pricing policies have tended to limit market size, thus deterring potential gains in the U.S. share of world exports in an age of an expanding export market. Because costs to U.S. growers have increased as fast as or faster than the support price, the "cost—price" squeeze has resulted in a drop in the number of producers, hired workers, and acreage used per crop. The prosperity of the flue-cured tobacco industry depends on rebuilding the market and establishing pricing policies which can compete in the foreign market.

82–1195. Morris, J. E. **This Tobacco Business. Part XV: Help.** *Tobacco International* 184(4): 28-38, February 19, 1982.

The relationship between the world's economic structure and the tobacco industry is discussed. The northern hemisphere (excluding China) has one-quarter of the world's population and four-fifths of its income. The south, including China, has 3 billion people, three-quarters of the world's population, but is living on one-fifth of the world's income. Over 90 percent of the world's manufacturing industry is in the north Subdividing the world's cigarette production for 1980 in the same way, the following comparisons are evident: north 61.7 percent, south 5.1 percent, Union of Soviet Socialist Republics and Eastern Europe 16 percent, and China 17.2 percent. Average per capita consumption is 1,445 and 786 cigarettes per annum for the north and south, respectively. The major cigarette-producing countries of the world and their cigarette consumption per capita in comparison with their income per capita for 1980 are presented in tabular form. The indication is that developing countries will need help for the future and now is a good time for the tobacco industry to be evaluating services for assistance and prosperity. BAT, R. J. Reynolds, and Philip Morris have agreements with the People's Republic of China for cigarette manufacturing, and R. J. Reynolds Tobacco International is negotiating with the Union of Soviet Socialist Republics to grow tobacco commercially for a jointly created brand of cigarettes. As developing countries achieve greater prosperity, benefits will accrue to the tobacco industry through the enormous potentialities for increased cigarette consumption and production.

82–1196. Naguib, M. R. **Rothmans—Eastern Deal, Lengthened Filter Highlight Egypt's Tobacco Year.** *Tobacco International* 184(4): 44, 47, 50, February 19, 1982.

The Egyptian tobacco industry's 1980/1981 highlights are reviewed. (1) An agreement on a joint venture factory was made by Eastern Tobacco Company, the main Egyptian cigarette producers, and Rothmans International. Capacity will start at 3 billion to be increased gradually to 15 billion cigarettes. (2) Total tobacco consumption was 35,340 million kilograms compared with 33,291 in 1979/1980. Eastern Company, El-Nasr Company, and the private sector's share was 76.3, 16.4, and 7.3 percent, respectively. (3) Cigarette production increased by only 650 million units compared with the 2,360 million increase the previous year. Total sales of top class cigarettes have risen rapidly, however, over the past 5 years, doubling consumption. (4) Moulassed and snuff tobacco has been increasing annually but roll-your-own, chewing, and pipe tobacco has decreased. (5) Prices of domestic cigarettes have been stable under a governmental price control policy while imports are double the prices of locals. (6) A proposed smoking control law will restrict smoking in all means of transportation, will require nicotine and tar content to be labeled on all cigarette packages along with health warnings, and will restrict advertising and sales to minors. (7) A new product is being launched: Cleopatra brand cigarettes with nicotine and tar contents below 20 mg, a longer filter, and less tobacco.

82–1197. Thassitis, B. **Major Changes Faced by Greek Cigarette Market.** *Tabak Journal International* 1: 10, 12, 14, February 1982.

Some general observations can be made about the market for tobacco products in Greece. Most notably, the market is dominated almost entirely by cigarettes (99.8 percent of the total output). In 1980, 25,293 tons of cigarettes (880 cigarettes = 1 kg) were produced compared with only 911 kg of cigars and 14,800 kg of pipe tobacco. Tobacco consumption as a whole has been virtually at a standstill over the past 2 or 3 years, while imports have increased. The figures show that domestic cigarette consumption receded from 24,942 tons in 1978 and 24,912 tons in 1979 to 24,889 tons in 1980. Domestic pipe tobacco consumption fell from 21.1 tons in 1978 and 17.4 in 1979 to 14.8 in 1980 while cigar sales slumped from 1.6 tons in 1978 and 1.1 in 1979 to 0.9 in 1980. In contrast to the rapid growth in cigarette imports, exports in 1980 were only 51 tons higher than in 1978 and down 6 tons from 1979. For 1981 the following trends were seen in the cigarette market: (1) total consumption rose, (2) foreign brands produced in Greece or imported made progress, (3) filter cigarettes gained in the market (1980 share was 91.4 percent), and (4) controlled rises in retail sales prices have occurred. It is predicted that the alignment of Greece's tax system with the Common Market System will introduce a binomial tax consisting of a fixed and a proportional tax and the regulation that manufacturers must produce 880 cigarettes/kg will be abolished. These changes along with pressure for reduced tar and nicotine and the inevitable price rise will markedly affect the Greek cigarette industry.

82–1198. Tobacco Institute. **Tobacco Industry Profile 1981.** Washington, D.C., The Tobacco Institute, 1981, 4 pp.

Economic information about the tobacco industry, based primarily on 1980 data, is provided including consumption, expenditures, global production, domestic production, government tobacco programs, taxes, manufacturing, distribution and sales, supply network, and tobacco's contribution to the domestic economy. Domestic consumption totaled 630 billion cigarettes. It is noted that while per capita consumption remains stable at 3,924, it is far lower than the record high of 4,345 in 1963. Expenditures for tobacco products were estimated at 20.8 billion dollars, of which about 19.2 billion dollars were for cigarettes. Total global tobacco output was estimated at 5.2 million metric tons. In the United States, where tobacco was the sixth biggest cash crop, government tobacco programs aim to stabilize tobacco production and assure fair prices. For the United States in 1980, Federal, State, and local governments realized over 6.6 billion dollars from direct taxes on tobacco products; approximately 69,000 people were employed in tobacco manufacturing; and about 1.4 million retail outlets distributed tobacco products. The United States remains the leading importer and exporter of tobacco, thereby aiding the domestic economy by providing employment, investment, and tax money.

82-1199. U.S. Department of Agriculture. **World Cigarette Production and Trade Continued Upward in 1980.** *Foreign Agricultural Circular—Tobacco*, FT 4-18, U.S. Department of Agriculture, Foreign Agricultural Service, Washington, D.C., July 1981, 29 pp.

World cigarette production and trade statistics for 1980 are presented, past economic performance is reviewed, and possible future trends are discussed. For 1980, global cigarette production was 4,373 billion pieces, compared with 4,311 billion in 1979. The rate of increase in cigarette production has slowed in recent years to 2 percent annually, due to price increases, antismoking campaigns, and higher production costs. Most production increases are taking place in developing nations, while developed nations are experiencing smaller rates of increase. Filter-tipped cigarettes continue to comprise an increasing proportion of cigarettes in the United States and elsewhere. Trends in production and consumption in 1979 and 1980 are expected to continue through 1985. In 1980, global cigarette exports were 314 billion pieces and imports were 263 billion pieces. A detailed analysis of 1980 tobacco economic activity for both developed and developing nations is included.

LEGISLATION

82-1200. Arutt, A. **Tobacco: A Challenged Contribution.** *Interservice* 1(2): 82, 85-88, 93-94, Winter 1981.

An internal U.S. Department of Defense proposal which urges the discontinuation of cigarette sales in commissaries and the setting of prices in clubs and exchanges at the prevailing local rate is criticized. The proposal, it is argued, would adversely affect various services available to military customers. At present, tobacco products are a major sales item in military stores (more than 500 million dollars annually) which charge the military customer lower tobacco prices than do civilian stores. Under the proposal, exchanges could experience reduced revenues, and higher exchange prices would also reduce the spendable income of military personnel. Linking exchange cigarette prices to local commercial prices could also reflect regional price differences. The proposal could set a precedent for removing other products from sale in the commissaries.

82-1201. Barr, S. H.; Lambert, C. A. **Smoking Regulation in Restaurants and Health-Care Facilities. (Letter).** *New England Journal of Medicine* 306(13): 812, April 1, 1982.

A survey of attitudes toward the regulation of smoking in restaurants and health-care facilities in Massachusetts found broad-based support for a policy of separate smoking and nonsmoking areas. Field interviews, conducted at the institutions in question, asked subjects to state their preferences among four regulatory alternatives: (1) a total ban on smoking, (2) separate areas for smokers and nonsmokers, (3) a requirement for adequate ventilation, and (4) no restrictions. Of the 3,313 interviews, 60 percent took place in restaurants and 40 percent in health facilities. Median age of subjects was 30 years. Overall, 85 percent favored some kind of restriction; " separate areas" was more than twice as popular as any other alternative. Of smokers, 75 percent favored some form of restriction, while persons who had never smoked were the strongest advocates of a total ban. Ex-smokers were the likeliest to vote for "separate areas."

82-1202. Bonnie, R. J. **Regulation of Alcohol, Tobacco, and Other Drugs: The Agenda for Law Reform.** In: Harris, L. S. (Editor). National Institute on Drug Abuse Research Monograph 34, U.S. Department of Health and Human Services, Public Health Service, DHHS Pub. No. (ADM) 81-1058, February 1981, pp. 272-286.

The problem of drug regulation, based on the premise that it is desirable for government to prevent or minimize the use of any substance which can result in health impairment, is discussed. Focus is placed on how the law is best used to implement a policy built on the public health model. The chronology of the recent Federal antismoking initiative is reviewed. It is claimed that, while considerable evidence has been developed about the health effects of smoking, too little is known about the associated behavioral factors. The goals of the Department of Health and Human Services (HHS) for the 1980s are the reduction of adult smoking to below 25 percent by 1990 and the reduction of smoking in youth 12-18 years of age to below 6 percent. Specific measures recommended by HHS include stronger health warnings, bans on smoking in public places, and insurance premiums which differentiate between people who smoke and those who do not. It is noted that the tobacco industry is untouched, and nothing is said about availability, yet antismoking goals cannot be reached as long as these conditions are ignored. It is recommended that:

(1) the objective of national tobacco policy should be clearly stated; (2) Federal programming for prevention of cigarette smoking should not be limited to the initiatives of the Office on Smoking and Health; and (3) the Federal Government should work with States to reduce availability, especially through the banning of vending machines.

82-1203. Chwat, J. S. **The Year Ahead: Tobacco in Congress.** *Tobacco International* 184(4): 22, 24, 26, February 19, 1982.

The legislative battle over the future of the tobacco program is reviewed. The arguments behind the recently introduced bills are not based on traditional antitobacco grounds, but rather on the new economic policies of deregulation, free market economy, budget savings, and supply-side economics. Besides the deregulation arguments, proponents of abolishing the tobacco price support system cite the erosion of the U.S. position in the world market (from 60 to 22 percent over the last decade) as a reason for change. Opponents of deregulation argue that thousands of small farmers would suffer bankruptcy, and the free market system would be reduced to a power monopoly in the hands of a few large corporations. Extensive hearings by both sides have been scheduled throughout the tobacco region to prepare for next year's legislative proposals.

See also, **82-0960, 82-1138, 82-1196**

82-1204. Pharmaceutical Journal. **Dr. Owen Seeks Legal Changes.** *Pharmaceutical Journal* 228(6167): 332, March 20, 1982.

Major legal changes to tackle the health hazard posed by smoking, proposed by Dr. David Owen, Commons leader of the Social Democrats, include: (1) an amendment of the Medicines Act 1968 to create specialist committees to provide expert advice on the health risks of both tobacco and alcohol; (2) under Section 62 of the Act, cigarettes with excessive tar, nicotine, or carbon monoxide could be prevented from being sold or imported on the advice of said committee; and (3) a ban on sport sponsorship by the tobacco industry. A new agreement relating to sports sponsorship will allow the industry to spend more on prize money, but all advertisements for tobacco-sponsored events will have to carry a health warning.

GENERAL

82-1205. Bonham, G. S.; Wilson, R. W. **Minor Error Noted in Study on Child Health in Smokers' Families. (Letter).** *American Journal of Public Health* 72(4): 403, April 1982.

A minor error which does not affect findings in a study on children's health in families with cigarette smokers (*American Journal of Public Health* 71(3): 290-293, March 1981) is reported. Affected are the chi-square values shown in table 3. Corrected unadjusted and unweighted chi-square values are provided for the purpose of statistical accuracy and for documentation purposes. Values of the unadjusted weighted and the adjusted chi-squares would also be slightly affected. See also 81-0791.

82-1206. Garner, D. W. **No More Secret Subsidies for Cigarettes.** *Business and Society Review* (29): 52-54, Spring 1979.

When a smoker goes to the hospital, the public, through tax-supported hospitals, government health care programs, social security, and unemployment benefits, pays a large part of the bill. Smoking is responsible for a growing proportion of illness, resulting in increased medical and economic liabilities. One means of desubsidizing smoking would be to tax cigarettes to the extent of the costs of their consumption. This "safety tax" could be levied according to the liability of the brand: a completely safe brand would be free of the tax. This would provide incentive to manufacturers to develop safer cigarettes, and allow the public to recoup the expenditures made for a smoker. An alternative would be to tax according to tar and nicotine levels. However, as these are not the only dangerous elements of cigarette smoke, this would not be as effective as a true "safety tax," especially if more cigarettes are consumed to compensate for declining nicotine levels. A *high* -nicotine, low-tar cigarette is proposed to counter this problem.

82-1207. Hoffmann, D.; Wynder, E. L. **Tobacco and Tobacco Smoke—A Continuing Public Health Issue.** *Tabak Journal International* 1: 83-85, February 1982.

Low-yield cigarettes continue to be produced and developed in response to the smoking and health controversy. Studies have found a dose—response relationship between cigarette consumption and the risk of tobacco-associated cancers. Attempts have been made to decrease cancer health risks by reducing the levels of hazardous constituents which have been identified, such as polynuclear aromatic hydrocarbons, phenols, catechols, or volatile and nonvolatile tobacco-specific nitrosamines. Efforts have also been made to reduce or remove smoke levels of ciliatoxins, constituents which can impair respiratory functions, including hydrogen cyanide, acrolein, carbon monoxide, and possibly nicotine and phenols. Researchers have also suggested that nicotine and carbon monoxide may harm the cardiovascular system, and efforts have been made to reduce their levels in cigarettes also. However, some nicotine may have to remain for consumer acceptance. Tobacco substitute materials have not received wide acceptance by smokers, and smokeless tobaccos may also be hazardous. While being a nonsmoker is the only way to avoid the risks associated with cigarette smoking, attempts to reduce the toxic components in tobacco products should continue.

82–1208. Journal of Public Health Policy. **Action on Smoking and Health.** *Journal of Public Health Policy* 3(1): 9-11, March 1982.

Governmental action for discouraging the use of tobacco is urged and antismoking policy objectives and recommendations of three major international health associations are presented. A ban on tobacco advertising is advocated as essential to control major causes of death. It is estimated that in the United States, the yearly amount spent on advertising by tobacco companies is about 1 billion dollars. The continuation of the Government's tobacco support program is strongly criticized, and as an alternative, it is suggested that Congress could enact a health tax on tobacco to discourage its consumption, and use the tax proceeds to help farmers substitute other crops for tobacco. Proceeds could also be used to fund a large-scale health education program.

82–1209. Krasny, J. F.; Allen, P. J.; Maldonado, A.; Juarez, N. **Development of a Candidate Test Method for the Measurement of the Propensity of Cigarettes To Cause Smoldering Ignition of Upholstered Furniture and Mattresses.** U.S. Department of Commerce, National Bureau of Standards, National Engineering Laboratory, Center for Fire Research, Washington, D.C., October 1981, 42 pp.

A candidate test method for the measurement of the propensity of cigarettes to cause smoldering ignition of upholstered furniture and mattresses is described. It consists of placing burning cigarettes on pieces of a standard, alpha cellulose, chromatographic paper and measuring the weight loss rate of the paper/cigarette system. The results were compared to the propensity of cigarettes to ignite upholstered furniture substrates. The agreement was satisfactory. Of the approximately 30 cigarette brands investigated, most had a similar propensity to ignite the upholstered furniture substrates, but a few ignited fewer substrates, and exhibited longer times to ignition on those which ignited. Many upholstered furniture substrates resisted ignition by any cigarette, while others were ignited even by those commercial cigarettes which ranked lowest in ignition propensity in these tests. However, a substantial number of upholstered furniture types did ignite with some cigarettes but not with others. Self-extinguishing time alone does not define the propensity of a cigarette to ignite upholstered furniture. (Auth. Abs.)

82–1210. Kuller, L. H. **Who Should Provide Research Initiative and Support?** *Environmental Health Perspectives* **42: 81-88,** December 1981.

Solutions are proposed to the problem of funding low-level environmental pollution studies. The cost of doing environmental studies is substantial. Various resources are currently being utilized for public health studies, such as the Egg Board's tax on egg crates. However, there are several problems associated with many of these nongovernmental resources; i.e., studies tend to be short term and are generally not investigator oriented. One possible solution to the problem of obtaining a large funding source for environmental research, independent of special interest groups, may be a tax on dangerous consumer products, such as cigarettes and alcohol. Smoking is probably the single largest source of personal pollution. Separating the factors of air pollution, occupational exposures, and smoking in lung cancer or obstructive pulmonary disease etiology has been difficult. In trying to reduce costs in environmental studies, epidemiologists have either only crudely measured smoking and occupational variables, or not at all, and have assumed the distribution was similar. A 10 cent tax on a pack of cigarettes would yield between 2.5 and 5 billion dollars for health care and health-related research. (Auth. Abs. Mod.)

82–1211. Vogt, T. M. **Questionnaires vs Biochemical Measures of Smoking Exposure. (Letter).** *American Journal of Public Health* 72(1): 93, January 1982.

The controversy implied in a previous article (*American Journal of Public Health* 71(3): 308-311, March 1981) over questionnaires vs. biochemical methods for determining cigarette consumption in medical care settings is discussed. It is agreed that a questionnaire is the more appropriate and economical instrument in the setting of the previously mentioned study. It is further pointed out that biochemical testing is recommended in only two situations: (1) smoking cessation programs where social and peer pressures lead to underestimation of consumption; and (2) in assessing the health effects of smoking. See also 81-0902.

82–1212. Wack, J. T.; Rodin, J. **Smoking and Its Effects on Body Weight and the Systems of Caloric Regulation.** *American Journal of Clinical Nutrition* 35(2): 366-380, February 1982.

Cigarette smoking and excess body weight appear to be inversely related. Data indicate that smokers weigh less than nonsmokers and that weight gain occurs after the cessation of smoking. The popular wisdom is that this is due to differences in caloric intake: smokers weigh less because they consume less and they gain weight upon stopping smoking as a consequence of consuming more. Cross-sectional data on this point are conflicting, however, with some studies of daily caloric intake suggesting that smokers may, in fact, consume more calories per day than nonsmokers. In addition, cigarette smoking has a variety of physiological effects that may play a more important role than amount of calories consumed. Evidence regarding the effects of cigarette smoking and nicotine upon the behavioral, sensory, and metabolic components of the processes of caloric regulation and nutrition is reviewed. Within-subject studies investigating weight changes associated with the cessation and initiation of smoking are recommended. (Auth. Abs. Mod.)

AUTHOR AND ORGANIZATIONAL INDEX

Aaro, L. E., 82–1086
Aberg, A., 82–0959
Abood, L. G., 82–0932
Abueg, F. R., 82–1121
Adams, J. D., 82–0930
Agnew, J. E., 82–0982
Ahlund, C., 82–1077
Albert, E. D., 82–1071
Aleksandrov, A. A., 82–1043
Allen, P. J., 82–1209
Aly, M. A., 82–1062
Ames, R. G., 82–0962
Andersen, P., 82–0933
Andreasson, L., 82–0980, 82–1060
Angebault, M., 82–1064
Antonova, L. T., 82–1043
Aoyama, M., 82–1061
Arcos, J. C., 82–0941
Argus, M. F., 82–0941
Arrendale, R. F., 82–0921
Arutt, A., 82–1200
Ashburn, W. L., 82–1021
Atkinson, A. B., 82–1117
Aziz, S., 82–1062

Bach, B., 82–0933
Bailey, C. V., 82–1186
Baird, A., 82–1065
Baird, R. N., 82–1023
Barr, S. H., 82–1201
Battigelli, M. C., 82–0983
Baxter, S., 82–1169
Bayes, J., 82–0984
Beaglehole, R., 82–0957
Beck, G. J., 82–0985
Becker, C. G., 82–1032
Becklake, M. R., 82–0991
Begin, R., 82–1051
Bellamy, D., 82–1002
Bellon, G., 82–0986
Bengtsson, C., 82–1041
Berger, P., 82–0937
Berglund, E.-L., 82–1086
Berkson, D. M., 82–1122
Berthiaume, Y., 82–1051
Bidlack, J. M., 82–0932
Biener, K., 82–1087
Bilimoria, M. H., 82–0987
Biot, N., 82–0999
Bjorlin, G., 82–0980, 82–1060
Bjornsson, O. J., 82–1094
Black, L. F., 82–1014
Bloom, R., 82–1142
Blovin, D., 82–1051
Bohadana, A., 82–1012
Bolt, R. C., 82–1170
Bonde, G. J., 82–0933
Bone, R. C., 82–1000
Bonham, G. S., 82–1205
Bonnie, R. J., 82–1202
Bonniot, J. P., 82–1064
Booth, H., 82–0932
Bornemisza, P., 82–1024
Bornfleth, U., 82–1181
Bottiger, L. E., 82–1025
Botvin, G. J., 82–1118
Boye, N. P., 82–1017, 82–1018
Bozler, G., 82–0996
Bradshaw, J. C., 82–1140
Bredesen, J. E., 82–1017, 82–1018
Brink, C.J.H., 82–1119
†British-American Tobacco Co., Ltd., 82–0922
†Brown and Williamson Tobacco Corp., 82–1177
Buchkremer, G., 82–1088, 82–1089
Bureau, M. A., 82–1051
Burgess, E. M., 82–1063
Burke, J. A., 82–1032
Burrows, G. D., 82–0944
Burry, R. D., 82–1192
Busbee, D. L., 82–0972
Bush, L. P., 82–0920

Callachan, H., 82–1080
Calleson, D. A., 82–1171
Cantrell, E. T., 82–0972
Carlson, L. A., 82–1025
Carp, H., 82–0988
Cartwright, W. F., 82–1172
Casewit, C. W., 82–1120
Castelli, W. P., 82–1050
Castro, E.J.S., 82–1093
Caton, J. R., 82–0934
†Celanese Corp., 82–1184
Chamberlain, W. J., 82–0921
Chaplin, J. F., 82–0916, 82–0918, 82–0930
Check, W. A., 82–1090
Cherek, D. R., 82–1091
Chesebro, J. H., 82–1026
†Chisso Corp., 82–1190
Choi, N. W., 82–0974
Chowdhury, P., 82–0989
Chwat, J. S., 82–1203
Clarke, J. R., 82–0990
Clarke, S. W., 82–0982
Claude, J. R., 82–0939
Clement, J., 82–1005
Clements, J., 82–0957
Cline, W. K., 82–1145
Cluette, J. E., 82–1029
Coates, T. J., 82–1135
Cochrane, G. M., 82–0993
Cohen, B., 82–0925
Cohen, D., 82–0991
Colletti, G., 82–1121
Colome, S., 82–0958
Conn, D. L., 82–1014
Cooper, R., 82–1122
Cordeiro, R., 82–0992
Cornell, A., 82–1172
Costa, G., 82–0981
Costa, P. T., Jr., 82–1092
Couto, C.L.M., 82–1093
Cross, F. T., 82–0963
Crowther, T. S., 82–0991
Cuangani, P. D., 82–1169
Curtis, M. B., 82–1045

Dagle, G. E., 82–0963
Daicel Ltd., 82–1187
Dalbey, W. E., 82–0934
Danaher, B. G., 82–1127
Davidson, S., 82–1123
Davidsson, D., 82–1094
Davis, J. W., 82–1027
Davis, R. F., 82–1027
de Alleluia, I. B., 82–0970
Denyer, M., 82–1074
Deutsche Hauptstelle Gegen die Suchtgefahren., 82–1095
Dexel, T., 82–1071
DiClemente, C. C., 82–1096
Dougherty, J., 82–1097
Dowd, D. E., 82–1002
Doyle, C. A., 82–0985

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Drizd, T. A., 82–0948
Ducimetiere, P., 82–1028
Duryea, E., 82–1098
Dymond, H.F.D., 82–0922

†Eastman Kodak Co., 82–1180
Eastwood, M. R., 82–1099
Ecobichon, D. J., 82–0987
Efthimiou, J., 82–0993
Eik-Nes, S., 82–1053
Eiser, J. R., 82–1100, 82–1128
El-Gengehy, M. T., 82–1062
El Hofy, O. A., 82–1062
Ellis, L.B.M., 82–1124
Elo, J., 82–1047
Elveback, L. R., 82–1026
Elwood, J. H., 82–0994
Elwood, P. C., 82–0994
Eng, A., 82–1118
Eschmann, G., 82–0995
Evans, R. I., 82–1101

Fabsitz, R. R., 82–1001
Fankhauser, P., 82–1157
Farquhar, J. W., 82–1036, 82–1127
Federal Trade Commission., 82–0923
Feinleib, M., 82–1001, 82–1050
Feinschreiber, R., 82–1193
Ferris, B. G., Jr., 82–0958
Festy, B., 82–0939
Feyerabend, C., 82–0935
Filipy, R. E., 82–0963
†Firmenich SA, 82–1157, 82–1167
Flanders, W. D., 82–0964
Fleischmann, R., 82–0996
Fowler, C. J., 82–0946
Frecker, R. C., 82–0940
Friedlander, E., 82–0965
Friedman, R. J., 82–0978
Frye, R. L., 82–1026
Fujise, H., 82–1061
Fukuda, J., 82–1011
Fulton, A., 82–0944
Fuster, V., 82–1026

Gaillard, J. P., 82–1064
†Gallaher Ltd., 82–1175
Galle, J., 82–1015
Garner, D. W., 82–1206
Garrison, R. J., 82–1050
Geiszler, W. A., Jr., 82–1146
Gibbs, G. W., 82–0991
Giesler, G. J., Jr., 82–0997
Girdwood, A. H., 82–1074
Glazunov, I. S., 82–1043
Godfrey, F. D., Jr., 82–1176
Goldberg, M. E., 82–1125
Gordon, L. V., 82–1102
Gorn, G. J., 82–1125
†Goseikagaku Institute Ltd., 82–1187
Goshtautas, A. A., 82–1126
Gottlieb, L. S., 82–0966
Gouveia, J., 82–1064
Granda, E. J., 82–1150, 82–1151, 82–1152, 82–1165
Greene, R. K., 82–1146
Gretz, H., 82–1173
Gros, F., 82–1064
Grover, R. F., 82–1055
Guerin, M. R., 82–0934, 82–0936, 82–0951
Guetat, M., 82–0937
†Gulf and Western Corp., 82–1172, 82–1176
Gwinn, C., 82–1001

Haferkamp, O., 82–1015
Hagedorn, M. L., 82–1159
Haines, A. P., 82–0998
Hakanson, N., 82–1124
Hall, F. V., 82–1174
Hall, J. B., 82–1147, 82–1156, 82–1158, 82–1165, 82–1168
Hall, R., 82–1194
Hall, Y., 82–1122
Hallak, A. B., 82–0938
Hamada, M., 82–1031
Hankin, J. H., 82–0975
Hannhart, B., 82–1012
Hardwick, M. J., 82–1175
Harf, R., 82–0999
Harries, A. D., 82–1065
Hartman, M. T., 82–1021
Hasgekar, N. N., 82–1067
Hassanein, K. M., 82–1027
Hatfield, A., 82–1074
Hatsushiba, S., 82–1011
Hauknes, A., 82–1086
†Hauni-Werke Koerber and Co. KG., 82–1173, 82–1181
Haynes, D. K., 82–1102
Hegarty, K. M., 82–1029
Heilbrun, L. K., 82–0975
Heinrich, R., 82–0996
Heinrichs, W. L., 82–1052
Heise, H., 82–1075
Helgeland, A., 82–0954
Hems, R., 82–1154
Henderson, A., 82–1101
Hercend, T., 82–1064
Hernandez-Llamas, H., 82–0967
Higenbottam, T., 82–0935, 82–0993
Higgins, D. B., 82–1172, 82–1176
Hill, P., 82–1101
Hiller, F. C., 82–1000
Hinds, M. W., 82–0968
Hiramoto, M., 82–1003
Hirn, M., 82–1059
Hjermann, I., 82–0954
Hodek-Demarin, V., 82–1030
Hodges, M. E., 82–1066
Hodgson, R., 82–1106
Hoffmann, D., 82–0930, 82–1207
Hoffmeister, H., 82–0955
Hoidal, J. R., 82–0988
Hojnacki, J. L., 82–1029
Holford-Strevens, V., 82–0952
Holme, I., 82–0954
Holt, D., 82–0993
Homasson, J. P., 82–1064
Hornebeck, W., 82–0986
Houminer, Y., 82–1155
Hubert, H. B., 82–1001
Hugh-Jones, P., 82–1002
Hughes, J. A., 82–1002
Huhti, E., 82–1013
Husen, L. A., 82–0966
Hutchison, D.C.S., 82–1002

Ibsen, K. K., 82–1103
Iida, M., 82–1048
Imai, T., 82–1011
Imamura, Y., 82–1031
Imeson, J. D., 82–0998
†International Flavors and Fragrances Inc., 82–1147, 82–1148, 82–1149, 82–1150, 82–1151, 82–1152, 82–1153, 82–1156, 82–1158, 82–1159, 82–1160, 82–1161, 82–1162, 82–1163, 82–1164, 82–1165, 82–1166, 82–1168
Irving, C. L., 82–1185
Ishidate, T., 82–0975
Itoh, K., 82–1011
Izawa, T., 82–1187

Jaeger, M. J., 82–0990
Jaffe, J. H., 82–1104, 82–1105, 82–1106
Jahnigen, D., 82–1055
James, W. H., 82–0939
Janoff, A., 82–0988

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

†Japan Tobacco and Salt Public Corp., 82-1190
Jardin, P., 82-1012
Jarvik, M. E., 82-0924
Jeffery, R. W., 82-1127
Joh, T., 82-1031
Johansson, S., 82-0959
Johnson, K., 82-0960
Johnson, R. R., 82-1177
Jonsson, A., 82-0942
Jouppila, P., 82-1053
Journal of Public Health Policy., 82-1208
Journal of the American Dental Association., 82-0969
Juarez, N., 82-1209
Juguet, B., 82-0939
Junge, B., 82-0955

Kambe, M., 82-1003
Kamiyama, S., 82-0975
Kandarkar, S. V., 82-1067
Kannel, W. B., 82-1050
Kanzler, M., 82-1104, 82-1105
Karlsson, S., 82-1094
Kasper, R., 82-1165, 82-1166
Katanosaka, A., 82-1187
Keith, C. H., 82-1178, 82-1184
Keller, C., 82-0970
Kelly, D. H., 82-1057
Kelly, E., 82-1112
Keown, K., 82-1044
Keritsis, G. D., 82-1179
Kerkay, J., 82-0947
Kew, R. R., 82-1029
Khan, F., 82-1078
Khan, M. A., 82-1068
Kiefer, J. E., 82-1180
Killen, J., 82-1127
Kimball, A. W., 82-0967
King, J., 82-1128
Kinnier, R., 82-1127
Kirkinen, P., 82-1053
Kiryushchenkov, A. P., 82-1058
Kiwala, J., 82-1148, 82-1149, 82-1150, 82-1151, 82-1153, 82-1156, 82-1160, 82-1161, 82-1162, 82-1163, 82-1164, 82-1166
Klein, B., 82-1138
Klemarczyk, P., 82-1159
Kline, J., 82-1054
Kobayakawa, T., 82-1003
Koch, F.-P., 82-1181
Koehler, E., 82-1181
Koivula, A., 82-1053
Kojima, S., 82-1048
Kojima, T., 82-1011
Kokubu, T., 82-1031
Kolonel, L. N., 82-0968
Komachi, Y., 82-1048
Kongerud, J., 82-1017
Konishi, M., 82-1048
Kopf, A. W., 82-0978
Kornstad, S., 82-1018
Korsgaard, R., 82-0980, 82-1060
Kostenbauder, H. B., 82-1097
Kostowska, B., 82-0917
Kotani, M., 82-1187
Kozlowski, L. T., 82-0940, 82-1107, 82-1112
Kramer, I.R.H., 82-0971
Krapfl, J. E., 82-1144
Krasnegor, N. A., 82-1108
Krasny, J. F., 82-1209
Krause, M., 82-1007
Krueter, M., 82-1098
Kukita, H., 82-1031
Kuklya, Yu. I., 82-1046
Kuliczkowski, K., 82-1069
Kuller, L. H., 82-1210
Kummer, G., 82-1007
Kunimune, K., 82-1190

Lagrue, G., 82-0986
Lai, D. Y., 82-0941
Lambert, C. A., 82-1201
Lan, S.-P., 82-1004
Lancet., 82-1070
Lander, T., 82-1071
Lando, H. A., 82-1129, 82-1130, 82-1141
Landser, F. J., 82-1005
Lange N., 82-1181
Lapsanski, D. V., 82-1136
Lavstedt, S., 82-1077
Ledederte, A., 82-1064
Lee, J., 82-0968
Lee, M. L., 82-0926
Lee, P. N., 82-1072
Legg, P. D., 82-0918
Lei, H., 82-0940
Lellouch, J., 82-0939
Lemaigre, G., 82-1064
Leren, P., 82-0954
Levenstein, M., 82-0978
Levi, R., 82-1032
Levin, B., 82-1054
Levin, L. I., 82-0960
Levine, J., 82-0978
Lewis, D. J., 82-1073
Ligeti, E. C., 82-1182
†Liggett Group Inc., 82-1171, 82-1174
Light, B. M., 82-1152
Light, K. K., 82-1148, 82-1156
Lippe, J., 82-1051
Lippmann, M., 82-0925
Lissner, H. G., 82-1006
Little, F., 82-0982
Lloyd, J. S., 82-1082
Lorenzen, H.-C., 82-1173
Losten, F., 82-0937
Loupekine, F., 82-1109
Lucke, W., 82-1074

MacDonald, J. C., 82-0942
Madeley, J., 82-0919
Maehrlein, W., 82-1007
Maguire, K. P., 82-0944
Major, G., 82-1024
Major, I., 82-1024
Makarainen, M., 82-1013
Malchow, H., 82-0996
Maldonado, A., 82-1209
Manfreda, J., 82-0952
Marcer, G., 82-1008
Marks, I. N., 82-1074
Marsac, J., 82-1064
Martin, J. E., 82-1144
Martin, J. P., 82-0986
Martin, J. R., 82-1183
Martin, R. H., 82-1145
Martin, R. M., 82-0929
Martin, R. R., 82-0972
Masarei, J.R.L., 82-1033
Maskarinec, M. P., 82-0926
Mason, J., 82-1078
Mastrangelo, G., 82-1008
Masuda, H., 82-0975
Mathe, G., 82-1064
Mathieu, J.-L., 82-1051
Matsen, F. A., III., 82-1063
Mattheus, A., 82-1075
Mattiasson, I., 82-0980, 82-1060
Mazumder, M. K., 82-1000
McAlister, A. L., 82-1134
McCoy, E. C., 82-0943
McCrae, R. R., 82-1092
McCusker, K. T., 82-1000
McDuffie, F. C., 82-1014
McGill, H. C., Jr., 82-1076
McGovern, P. G., 82-1130
McLemore, T. L., 82-0972
McMeekan, K. R., 82-1175
McMillan, G.H.G., 82-1009
McNamara, P. M., 82-1050
Meade, T. W., 82-0998, 82-1034

---

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Meltzer, L. T., 82–0949
Merli, F., 82–0926
Merrett, T. G., 82–1079
Miano, R. R., 82–1184
Mikkelson, R. P., 82–1172
Miller, D., 82–1097
Miller, D. G., 82–0973
Miller, F., 82–0988
Mittman, C., 82–1003
Miyajima, I., 82–1011
Modeer, T., 82–1077
Moger, J., 82–1110
Mojonnier, L., 82–1122
†Molins Ltd., 82–1170
Monette, J., 82–1051
†Monsanto Chemicals Ltd., 82–1169
Mookherjee, B. D., 82–1149, 82–1150, 82–1151, 82–1152, 82–1153, 82–1165, 82–1166
Moore, L. G., 82–1055
Moore, W. S., 82–1044
Moreau, T., 82–0939
Morris, J. E., 82–1195
Morrison, A. S., 82–0965
Moser, K. M., 82–1021
Moshal, M. G., 82–1078
Moss, D., 82–1122
Motley, M. E., 82–1056
Muir, C. S., 82–0974
Mulligan, J. J., 82–1029
Mumpower, R. C., II., 82–1180
Muramatsu, M., 82–0927
Murphy, K. M., 82–1138
Mussinan, C. J., 82–1151, 82–1152, 82–1153

Naethke, M. E., 82–1071
Nagai, S., 82–1011
Naguib, M. R., 82–1196
Naidoo, V., 82–1078
Nakamura, S., 82–1035
Nakanishi, N., 82–1048
Nash, J. D., 82–1036
National Research Council., 82–1010
Nettesheim, P., 82–0934
New York State Bureau of Drug Education., 82–1131, 82–1132
Newman, A., 82–1142
Nienow, J. F., 82–1185
Nissinen, A., 82–1047
Nomura, A., 82–0975
Norman, T. R., 82–0944
Noro, M., 82–1011
Notoya, Y., 82–1011
Novotny, M., 82–0926

O'Bryan, R., 82–0990
Ochs, H. R., 82–0945
Ogoshi, M., 82–1003
Ohkubo, C., 82–1037
Ohloff, G., 82–1157
Okada, T., 82–0927
Olafsson, H., 82–1094
Olafsson, O., 82–1094
†Olin Corp., 82–1145
Olsson, J.-E., 82–1038
Omenn, G. S., 82–0956
Oreland, L., 82–0946
Orleans, C. S., 82–1133
O'Rourke, T. J., 82–1159
Orssaud, G., 82–0939
Othman, A., 82–1062
Otten, H., 82–0945
Ozawa, H., 82–1048

Pacheco, Y., 82–0999
Palmer, R. F., 82–0963
Pantin, C.F.A., 82–1079
Pare, C., 82–1051
Parrot, R., 82–1064
Pavia, D., 82–0982
Pearson, K. H., 82–0947
Pechacek, T. F., 82–1134, 82–1143
Peden, N. R., 82–1080
Pedersen, O. F., 82–0933
Pelkonen, O., 82–1059
Peltier, B., 82–1135
Pequignot, G., 82–1028
Perera, F., 82–0976
Perrin-Fayolle, M., 82–0999
Peslin, R., 82–1012
Petersen, K.-E., 82–1181
Petersen, R., 82–1106
Peto, J., 82–0977
Pharmaceutical Journal., 82–1204
†Philip Morris Inc., 82–1146, 82–1155, 82–1179, 82–1185, 82–1186, 82–1191
Piasecka-Grzeszek, A., 82–0917
Piazza, G., 82–1008
Pignal, C., 82–0937
Pinck, P., 82–1181
Pleban, P. A., 82–0947
Plunkett, E. R., 82–1039
Pogrel, M. A., 82–1081
Pomerleau, O. F., 82–1111
Poukkula, A., 82–1013
Pretet, S., 82–1064
Priola, D. V., 82–1045
Prior, I.A.M., 82–0957
Prochaska, J. O., 82–1096, 82–1136
Provenza, D. V., 82–1068
Prue, D. M., 82–1144
Pudliner, H. J., 82–0920
Puska, P., 82–1042
Pyke, F. S., 82–1033
Pyke, J. E., 82–1033
Pyorala, K., 82–1040

†R. J. Reynolds Tobacco Co., 82–1183
Radford, E. P., 82–0948
Rafnsson, V., 82–1041
Rainer, N. B., 82–1186
Raines, B., 82–1101
Raines, J. R., 82–1124
Rangel, M.C.M., 82–1093
Ratcliffe, G. E., 82–1082
Raw, M., 82–1137
Rayburn, W. F., 82–1056
Rees, R., 82–0957
Reeves, J. T., 82–1055
Reilly, M. J., 82–1082
Reist, P. C., 82–0928
Reynolds, D. T., 82–0932
Rhodes, J., 82–1065
Richard, J. L., 82–1028
Rigel, D. S., 82–0978
Robbins, R. L., 82–1116
Robert, L., 82–0986
Roberts, D. M., 82–1082
Rodin, J., 82–1212
Rosecrans, J. A., 82–0949
Rosenkranz, H. S., 82–0943
Rossiter, C. E., 82–1009
Rossoni, E. L., 82–1093
Roth, J., 82–1007
Rothman, K. J., 82–0964
Rounds, S. S., 82–1055
Rowsell, D. G., 82–1154
Rudszinat, W., 82–1173
Rugyavichyus, M. Z., 82–1126
Russell, M.A.H., 82–0935
Rutt, D. R., 82–0950
Ryan, K. C., 82–1002

Sadowski, J., 82–0917
Saia, B., 82–1008
Said, E., 82–1062
Salmond, C. E., 82–0957
Salonen, J. T., 82–1042
Sanders, E. B., 82–1155
Sanders, J. M., 82–1152, 82–1159
Santelli, G., 82–1064

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Sato, K., 82–1187
Saunders, J. W., 82–0920
Sawada, H., 82–1187
Schachter, E. N., 82–0985
Schalling, D., 82–0946
Schifflers, E., 82–0974
Schlemmer, L., 82–1078
Schlotzhauer, W. S., 82–0929
Schmitt, F. L., 82–1147, 82–1149, 82–1150, 82–1151, 82–1152, 82–1153, 82–1156, 82–1158, 82–1160, 82–1161, 82–1162, 82–1163, 82–1164, 82–1166
Schneider, L., 82–1138
Scholz, S., 82–1071
Schreck, R., 82–1165
Schreck, R. P., 82–1156
Schreiber, W. L., 82–1156, 82–1159
Schroeter, A. L., 82–1014
Schulte-Elte, K.-H., 82–1157
Schulz, V., 82–0995
Schwartz, H. R., 82–1014
Segnan, N., 82–0981
Seibel, W., 82–1068
Seidman, H., 82–0977
Selikoff, I. J., 82–0977
Sevenier, L., 82–0937
Shamarin, V. M., 82–1043
Shannon, D. C., 82–1057
Shapcott, D., 82–1051
Shaw, L. V., 82–1185
Shepherd, D. M., 82–1080
Sheveleva, G. A., 82–1058
Shimamoto, T., 82–1048
Shipley, R. H., 82–1133
Shuster, E. J., 82–1147, 82–1148, 82–1152, 82–1159
Shy, C., 82–1004
Sides, L. W., 82–1183
Sieffert, G. F., 82–1044
Siess, E. A., 82–1071
Sigfusson, N., 82–1094
Sigmon, N. A., 82–1171
Silverstein, B., 82–1112
Simmons, C. W., 82–1063
Simpson, W. W., 82–1045
Siren, M. J., 82–1188
Sirsat, S. M., 82–1067
Skegg, J. L., 82–1117
Smirnova, I. P., 82–1046
Smith, E. A., 82–1189
Smith, R., 82–1113
Snook, M. E., 82–0929
Soltero, I., 82–1122
Sombooncharoen, S., 82–0973
Spanel, R., 82–1015
Spaur, W. H., 82–0990
Speizer, F. E., 82–0958
Spencer, B. M., 82–1148
Spitaels, J. M., 82–1078
Sprecker, M. A., 82–1147, 82–1158, 82–1159, 82–1160, 82–1161, 82–1162, 82–1163, 82–1164
Spring, D. J., 82–1154
Spurr, H. W., Jr., 82–0916
Stack, D. J., 82–1029
Staecker, K.-H., 82–1006
Stamler, J., 82–1122
Standl, E., 82–1071
Stanhope, J., 82–0957
Stein, Z., 82–1054
Stemmermann, G. N., 82–0968, 82–0975
Stetter, J. R., 82–0950
Stock, S. L., 82–0979
Stokely, J. R., Jr., 82–0936, 82–0951
Stuart, B. O., 82–0963
Suciu, I., 82–1024
Sugimori, K., 82–1187
Supnick, J. A., 82–1121
Susser, M., 82–1054
Svobodova, A., 82–1114
Swan, J., 82–1112
Sweetnam, P. M., 82–0994
Sykut, E., 82–1016

Tachi, N., 82–1061
Tagliacozzo, R., 82–1115
Takayama, Y., 82–1048
Talseth, T., 82–1017, 82–1018
Tamaoki, A., 82–1190
Tanaka, S., 82–1190
Tanaka, U., 82–1011
Tanigaki, M., 82–1048
Telch, M. J., 82–1135
Tennant, F. S., Jr., 82–1019
Terr, A. I., 82–1020
Terracini, B., 82–0981
Thassitis, B., 82–1197
Thibodeau, L. A., 82–0958
Thomas, H. F., 82–0994
Tobacco Institute., 82–1198
Todd, G., 82–1097
Tokich, T., 82–1122
Tomioka, S., 82–1190
Trauth, H. A., 82–1006
Trell, E., 82–0980, 82–1060
Trell, L., 82–1060
Trenkle, R. W., 82–1165, 82–1166
Tso, T. C., 82–0930
Tsubokura, T., 82–1003
Tuia, H., 82–0957
Tuomilehto, J., 82–1042, 82–1047
Turgiss, L. E., 82–1029

U.S. Department of Agriculture., 82–1199
Ueshima, H., 82–1048
Ulvenstam, G., 82–0959
Umemura, S., 82–0927
United Kingdom Laboratory of the Government Chemist., 82–0931
University of Southern California, Cancer Center Office of Cancer Communications., 82–1139
Urberg, K., 82–1116
Utsch, F. V., 82–1191

Vahakangas, K., 82–1059
Valkonen, T., 82–1040
Van de Woestijne, K. P., 82–1005
Vassilaros, D. L., 82–0926
Vaughn, S., 82–1115
Vedin, A., 82–0959
Vessey, M. P., 82–1049
Vinals, J. F., 82–1147, 82–1148, 82–1149, 82–1150, 82–1151, 82–1153, 82–1156, 82–1159, 82–1160, 82–1161, 82–1162, 82–1163, 82–1164, 82–1165, 82–1166
Vineis, P., 82–0981
Vock, M. H., 82–1148, 82–1150, 82–1151, 82–1152, 82–1153, 82–1156, 82–1160, 82–1161, 82–1162, 82–1163, 82–1164, 82–1165, 82–1166
Vogt, T. M., 82–1211
Vorlaender, W., 82–1015

Wack, J. T., 82–1212
Wallace, J. M., 82–1021
Warburton, D., 82–1054
Ware, J. H., 82–0958
Waring, A. J., 82–1082
Warnet, J. M., 82–1028
Warren, C.P.W., 82–0952
Watkins, H., 82–1159
Watson, C. B., 82–1022
Weber, A., 82–1083
Weiss, W., 82–1084
Wiesler, D., 82–0926
Wilhelmssen, L., 82–0959
Wilhelmsson, C., 82–0959
†Wilkinson Sword Ltd., 82–1154
Williamson, R. E., 82–0929

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Wilson, J. A., 82-1082
Wilson, J. D., 82-1000
Wilson, P. W., 82-1050
Wilson, R. A., 82-1149, 82-1150
Wilson, R. W., 82-1205
Winkelstein, W., Jr., 82-0960
Winter, M., 82-1167
Wong, C., 82-0952
World Health Organization., 82-0961
Wormsley, K. G., 82-1080
Wray, N. P., 82-0972
Wright, J. P., 82-1074
Wynder, E. L., 82-1207
Wyss, C. R., 82-1063

Yallup, A. E., 82-0922
Yamakawa, H., 82-0975
Yang, H.-Y., 82-0968
Yoshida, T., 82-1168
Yoshikawa, O., 82-1011

Zahran, F., 82-0953
Zavecz, J. H., 82-1032
Zovic, D., 82-1085
Zumrick, J. L., 82-0990
Zuspan, F. P., 82-1056

---

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

SUBJECT INDEX

ABORTION
maternal smoking, possible dose—response relationship, 82–1054

ABSENTEEISM
industrial pollutants and smoking relationship, 82–1016

ACCIDENTS
cigarette smoking association in Norway, 82–0954
genetic predisposition, smoking relationship, 82–0956
smoldering ignition of mattresses and upholstery, cigarette testing, 82–1209

ADOLESCENTS
antismoking program, older teenagers interacting with younger ones, 82–1125
decisionmaking skills in health education, need for, 82–1098
elevated blood pressure, smoking relationship, 82–1043
initiation of smoking, influences and prevention methods, 82–1101
periodontal diseases in Swedish schoolchildren, snuff-dipping and smoking effect, 82–1077
physiological basis for increase in female smoking, incidence, 82–1112
psychosocial determinants of smoking habit and preventive strategies, 82–1108
recruitment technique effectiveness in high school smoking intervention programs, 82–1135
reduction in smoking, HHS goal for 1980s, 82–1202
resource guide for drug use/abuse education in New York, 82–1131
smoking behavior, differences by sex and peer behavior, 82–1116
smoking cessation strategies, handbook, 82–1120
smoking habits of 10- to 17-year-old students in Brazil, 82–1093
smoking problem in Europe, health congress papers, 82–1095
social influences, effect on smoking behavior in Norway, 82–1086
somatic development and smoking relationship, 82–1052

ADVERTISING
1971 cigarette advertising broadcast ban effect on consumption, 82–1138
recruitment techniques for high school smoking intervention programs, 82–1135
recruitment techniques for smoking intervention programs, 82–1129
restrictive legislation concerning tobacco products proposed in Egypt, 82–1196
suggested ban on tobacco promotion, 82–1208
1981 tobacco industry expenditures, 82–1192
United Kingdom, health warning requirements for tobacco-sponsored events, 82–1204
United Kingdom, televised cigarette advertising ban effect on consumption, 82–1137

AGE FACTORS
cardiovascular mortality relationship, 82–1049
coronary disease risk factor with smoking and cholesterol, 82–1050
immunoglobulin E levels, smoking effect, 82–1079
initiation of smoking by Brazilian youth, 82–1093
neuroticism and smoking relationship, 82–1099
oral contraceptive use, age, and smoking as risk factors in cardiovascular disease, 82–1039
theophylline dosage in obstructive lung disease patients, age and smoking effect, 82–1018

AGRICULTURAL PRACTICES
cultivar differences in smoke components, 82–0918
methyl bromide in insecticides used in Jordan, tobacco contaminant, 82–0938
pesticide Aldrin in poisoning of Kenyan tobacco farmers, 82–0919
residues of six herbicides on tobacco leaves, 82–0917
tobacco strains cultured in presence of nicotine, effect on nicotine content, 82–0920

AIR POLLUTION
additive and synergistic effects of smoking in the workplace, 82–0956
atmospheric sulphur and particulates, smoking role in health effects, 82–0958
attitudes toward regulation of smoking in restaurants and health-care facilities, 82–1201
bans on smoking in public places, recommended, 82–1202
benzo(*a*)pyrene, carcinogenicity and need for environmental standard, 82–0976
carbon monoxide, smoking as source of worker exposure, 82–0950
chronic respiratory diseases effect, smoking relationship, 82–1004
cigarette tax to fund health care and research, 82–1210
negative ion concentrations, effect of smoking and stearin candle, 82–1085

  protective measures recommended for passive smokers, 82-1083
  United States, relaxation of standards, pros and cons, 82-0960

ALCOHOL CONSUMPTION
  elevated blood lead concentrations, smoking relationship, 82-0939
  ethyl nitrite generation from interaction with smoking, 82-0942
  laryngeal and esophageal cancer association, smoking relationship, 82-0974
  laryngeal cancer risk, synergistic action with tobacco, 82-0964
  schoolchildren in Czechoslovakia, correlation with smoking, 82-1114
  spontaneous abortion and maternal drinking relationship, 82-1054
  tiapride as analgesic in alcoholics and smokers slated for cancer surgery, 82-0937

ALCOHOLS
  caryophyllene alcohol additive for tobacco flavor and aroma, 82-1153

ALLERGY
  cardiac reactions to tobacco glycoprotein in guinea pigs and rabbits, 82-1032

AMIDES
  alicyclic amides as tobacco-cooling additives, 82-1154

ANTIGEN-ANTIBODY REACTIONS
  response to inhaled antigens among smokers vs. nonsmokers, 82-0933
  tobacco-caused changes in the lung response, 82-0999

ANTIGENS
  ABH secretion among smokers and nonsmokers, 82-0998
  cardiac allergic reactions to tobacco glycoprotein in guinea pigs and rabbits, 82-1032
  response to inhaled antigens among smokers vs. nonsmokers, 82-0933
  tobacco, immune response of the lungs, 82-0999

ANTISMOKING CAMPAIGNS
  advertising ban and health information effects, 82-1138
  antismoking league to challenge rights of smokers, 82-1119
  industry smoking cessation programs for employees, review, 82-1133
  multicomponent prevention program for seventh grade students, 82-1118
  New Zealand, inactivity, 82-1113
  older teenagers interacting with younger ones, 82-1125
  suggested advertising ban, tobacco tax, 82-1208
  tobacco consumption effect in the United Kingdom, 82-1117, 82-1137

ANTISMOKING MATERIALS
  badges to identify and reinforce antismoking campaign, 82-1119
  directory of programs and cassette material in California, 82-1139
  resource guide for drug abuse prevention in New York elementary schools, 82-1132
  resource guide for drug use/abuse education programs in New York secondary schools, 82-1131

ANTITRYPSIN DEFICIENCY
  genetic and function loss of elastase inhibition in smokers, 82-0986

ANTITRYPSIN LEVELS
  serum content and activity, smoking effect, 82-0989

ARECA
  betel quid, cheek pouch epithelial dysplasia in hamsters, 82-1067

ARECA CHEWING
  cessation among urban youth in Thailand, 82-0973

AROMATIC HYDROCARBONS
  nitroarenes from nitrated cigarette tobacco, 82-0943
  responsiveness of fetal mice to maternal smoke exposure, 82-1059

ARRHYTHMIA
  cardiac allergic reactions to tobacco, possible relationship, 82-1032

ARSENIC
  cigarette smoke concentrations in Jordanian and U.S. cigarettes, analysis, 82-0938

ARTERIAL DISEASES
  coronary artery disease, family history, smoking and serum cholesterol as risk factors, 82-1026
  ultrasound and related techniques for diagnosis and monitoring after surgery, smoking effect, 82-1023

ARTERIES
  endothelial damage in rats with tobacco smoke inhalation, 82-1044

ARTERIOSCLEROSIS
  antitrypsin deficiency evaluation, simple method, 82-0986
  atherogenic influence of smoking in diabetics, heart disease patients, and controls, 82-1024
  tobacco smoke inhalation, causal relationship in rats, 82-1044
  tobacco smoke inhalation in etiology, 82-1076

- ultrasound and related techniques for early diagnosis in smoking populations, 82–1023
- varicose veins and smoking, association, 82–1028

ARYL HYDROCARBON HYDROXYLASE
- dose—response to cigarette smoke in rat lung, 82–0987
- high inducibility associated with smoking, 82–1060
- levels in lung cancer patients, 82–0972
- oral carcinoma, smoking and dental status relationship, 82–0980

ASBESTOS
- age vs. duration of exposure in risk assessment, smoking factor, 82–0977
- exposure effects in smokers vs. nonsmokers, 82–0991
- parenchymal fibrosis in dockyard workers, 82–1009

ASBESTOSIS
- dockyard workers, smoking habits, 82–1009

ATTITUDES
- adolescent smoking, influences and prevention methods, 82–1101
- adolescent smoking, sex and peer behavior differences, 82–1116
- illusory psychological benefits from smoking, 82–1092
- nonsmokers "rights, " how to fight smokers, 82–1119
- parents of fourth grade students, 82–1102
- psychological and physiological factors of smoking, 82–1111
- regulation of smoking in restaurants and health-care facilities, Massachusetts survey, 82–1201
- schoolchildren's smoking habits in Czechoslovakia, 82–1114
- schoolchildren's smoking habits in Norway, social influences, 82–1086
- self-labeling, concept of addiction as predictor of smoking cessation success, 82–1100
- self-quitting vs. therapeutic smoking cessation, 82–1096

AUSTRALIA
- mortality trends in men vs. women, "civilization-associated" diseases, 82–0955

AVERSIVE THERAPY
- increasing delay training comparison, efficacy, 82–1142
- medical risks, 82–1143
- treatment stage in multistage cessation program, efficacy, 82–1141

BACTERIA
- *Salmonella* mutagenicity assay for nitroarenes, 82–0943

BEHAVIOR, ANIMAL
- nicotine administration to rat brain with tonic seizure and aversive shock reaction, 82–0932
- nicotine as central nervous system stimulus, sites and mechanisms of action in rats, 82–0949
- nicotine self-administration factors, 82–1097

BEHAVIOR, HUMAN
- adolescent smoking, influences and prevention methods, 82–1101
- adolescent smoking, sex and peer behavior differences, 82–1116
- adolescents, guide to smoking cessation through behavior modification, 82–1120
- aggressive responses, nicotine dose level effect, 82–1091
- caloric regulation and nutrition, smoking relationship, 82–1212
- health behavior change, home correspondence program for smoking cessation, 82–1127
- illusory psychological benefits from smoking, 82–1092
- low-nicotine cigarettes, physiological and psychological effects, 82–1090
- neuroticism and smoking relationship, 82–1099
- positive lifestyle changes, effect on smoking cessation, 82–1136
- professional women, smoking and addiction in Switzerland, 82–1087
- psychological and physiological factors of smoking, 82–1111
- recruitment technique effectiveness in high school smoking intervention programs, 82–1135
- recruitment techniques, effectiveness in smoking intervention programs, 82–1129
- schoolchildren's smoking habits in Czechoslovakia, 82–1114
- schoolchildren's smoking habits in Denmark, 82–1103
- schoolchildren's smoking habits in Norway, social influences, 82–1086
- self-control in reduction of smoking through delay training, 82–1142
- self-quitting vs. therapeutic smoking cessation, 82–1096
- smoking cessation strategies, review, 82–1134, 82–1143
- smoking habit, an overview, 82–1106
- smoking prevention strategies, need for information, 82–1202
- tobacco dependence, current state of knowledge, 82–1108
- tobacco dependence, influencing factors, 82–1105
- work-related stress, effect on smoking among nurses, 82–1115
- workplace programs for health lifestyle changes, 82–1133

BENZO(*A*)PYRENE
- carcinogenicity and need for environmental standard, 82–0976

passive smoking exposure, lung cancer incidence relationship, 82-1066

BIOASSAY
Salmonella, nitroarenes formation investigation, 82-0943
animal vs. human models in disease research, 82-1076
Doppler sonography in blood vessel damage, smoking effects and validity in stroke detection, 82-1030
Doppler ultrasonography in measuring fetal, placental blood flow, 82-1053
Ecolyzer measurement to corroborate accuracy of self-reported smoking rates, 82-1121
erythrocyte sedimentation rate for predicting cardiovascular diseases in women, smoking effects, 82-1041
models for inhalation exposure dosimetry, 82-0934

BIRTH WEIGHT
growth potential relationship, maternal smoking effect, 82-1052
maternal arterial oxygenation at high altitudes, smoking relationship, 82-1055
polycyclic aromatic hydrocarbons, exposure effects on rat fetuses, 82-1059
reduction, maternal smoking relationship, 82-1054

BLADDER NEOPLASMS
etiological role of tobacco, 82-0974
L-tryptophan excretion in smoking vs. nonsmoking patients vs. controls, 82-0965
occupational exposure and cigarette smoking, combined effects, 82-0981

BLOOD
acetylsalicylic acid, reduction of tobacco smoke effect on platelet aggregate ratio, 82-1027
elastase inhibitory capacity in smokers, 82-0986
lead concentration in smokers and drinkers, 82-0939
lead content and Δ-aminolevulinic acid dehydratase activity, smoking and alcohol effects, 82-1069
white cell invasion in arterial intima of rats, tobacco smoke inhalation relationship, 82-1044

BLOOD CHEMICAL ANALYSIS
plasma nicotine analysis, nicotine yields of cigarettes, relationship, 82-0940

BLOOD CHOLESTEROL
circulatory disorder risk factors, 82-1042
coronary artery disease, risk factor with family history and smoking, 82-1026
high density lipoprotein cholesterol levels in Japanese males, smoking and alcohol effects, 82-1031
high density lipoprotein levels in Japanese populations, smoking relationship, 82-1048
high density lipoprotein levels, smoking and exercise effects, 82-1035

BLOOD CIRCULATION
fetal-placental blood flow, maternal smoking effect, 82-1053

BLOOD COAGULATION
acetylsalicylic acid, reduction of tobacco smoke effect on platelet aggregate ratio, 82-1027
anticoagulants in stroke prevention, sex, smoking, oral contraceptive, and pregnancy effects, 82-1038
atherogenic influence of smoking, 82-1024
thrombotic disorders, smoking as risk factor, 82-1034

BLOOD LIPIDS
higher plasma triglyceride levels associated with smoking, 82-1033

BLOOD PLATELETS
acetylsalicylic acid, reduction of tobacco smoke effect on platelet aggregate ratio, 82-1027
aggregation in rats with tobacco smoke inhalation, 82-1044
monoamine oxidase activity reduced in female smokers, 82-0946

BLOOD PRESSURE
alkaline and acid nicotine in cigarette smoke, effect, 82-1037
elevated in adolescents, smoking relationship, 82-1043

BLOOD SUGAR
atherogenic influence of smoking in heart disease patients, diabetics, and controls, 82-1024

BLOOD VESSELS
cranial blood flow velocity in stroke, smokers vs. nonsmokers, 82-1030
varicose veins and smoking association with arteriosclerosis, 82-1028

BODY WEIGHT
caloric regulation and nutrition, smoking relationship, 82-1212
reduction in smoke-exposed mice, 82-1061

BRAIN
nicotine, sites and mechanisms of action in the rat, 82-0932

BRAZIL
smoking habits of 10- to 17-year-old students, 82-1093

BRONCHI
lignocaine absorption during fibreoptic bronchoscopy, smoking effects, 82-0993
metaplasia in smokers, etretinate treatment effect, 82-1064

SUBJECT INDEX

BRONCHIAL MUCOSA
mucus clearance in smoking vs. former smoking chronic bronchitis patients, 82–0982

BRONCHITIS
incidence among painters and office workers, smokers vs. nonsmokers vs. ex-smokers, 82–1016
mucus clearance, smokers vs. ex-smokers, 82–0982
radon daughters, uranium ore dust and cigarette smoke exposure effect in beagle dogs, 82–0963

CADMIUM
kidney cortex concentration, smoking effect, 82–0947
malignant hypertension relationship, smoking in etiology, 82–1047
tobacco smoke filtration method, 82–1188

CAFFEINE
simulated cigarette with caffeine and a heat-producing compartment, 82–1140

CALIFORNIA
cessation of smoking programs and services, directory 82–1139
Three Community Study for cardiovascular disease intervention, 82–1036

CANADA
chrysotile asbestos mining and milling industry, dust exposure effects in smokers vs. nonsmokers, 82–0991
health professionals' advice to pregnant women on smoking and alcohol consumption, 82–1123

CANNABIS
hashish and tobacco smoking, health effects, 82–1019

CARBON MONOXIDE
acute poisoning via sheesha smoking, 82–0953
ban proposed on high levels in cigarettes in the United Kingdom, 82–1204
carboxyhemoglobin levels in mother and fetus, maternal smoking effect, 82–1051
conversion to carbon dioxide in a filter, 82–1188
increased levels from low-yield cigarettes through compensation, 82–1090
maternal smoking and sudden infant death relationship, 82–1057
national (U.S.) estimates of blood levels by age, race, smoking status, residence, income, and season of year, 82–0948
protective measures recommended for passive smokers when levels exceed 1-2 ppm, 82–1083
reduction of levels by brand switching, 82–1144
workplace exposure, smoking as source, 82–0950

CARBON MONOXIDE CONTENT
chemical analysis of 147 brands, United Kingdom, 82–0931
FTC report of levels in 200 varieties of cigarettes, 82–0923

CARBOXYHEMOGLOBIN LEVELS
elevation in rats associated with arterial intima pathology, 82–1044
mother and fetus, maternal smoking effect, 82–1051
national estimates (U.S.) by age, race, residence, and season of year, 82–0948
sheesha smokers vs. cigarette smokers, 82–0953

CARCINOGENESIS
age vs. duration of asbestos exposure and smoking in risk assessment, 82–0977
cigarette smoke, radon daughters and uranium ore dust, exposure studies in beagle dogs, 82–0963
tobacco-specific nitrosamines, in vitro binding to DNA, 82–0941

CARCINOGENICITY REDUCTION
high-nicotine, low-tar cigarettes recommended, 82–1090

CARCINOGENS
benzo(*a*)pyrene, prevalence and need for control, 82–0976
polonium-210 inhalation from tobacco smoke, possible effects, 82–0970

CARCINOMA, BRONCHOGENIC
alpha-emitters of polonium-210, probable causative agent, 82–0925

CARDIOVASCULAR DISEASES
intervention by lifestyle change, the Three Community Study, 82–1036
mortality relationship to age, smoking habit, and oral contraceptive use, 82–1049
oral contraceptive use, age and smoking as risk factors, 82–1039
predictive value of erythrocyte sedimentation rate in women, smoking effects, 82–1041

CARDIOVASCULAR SYSTEM
alkaline and acid nicotine in cigarette smoke, responses in smokers vs. nonsmokers, 82–1037

CATECHOLAMINES
metoprolol ganglionic blocking effect on nicotine-induced cardiac responses, 82–1045

CELLS
endothelial, elongation with uplifting from the basal membrane following tobacco smoke inhalation in rats, 82–1044

CELLS, EPITHELIAL
laryngeal epithelia of rats, smoke exposure effects, 82–1073

CENTRAL NERVOUS SYSTEM
  sites and mechanisms of nicotine action in rats, 82-0949

CEREBROVASCULAR DISORDERS
  international mortality trends, men vs. women, 82-0955
  physical activity relationship to other risk factors in Finnish men and women, 82-1042

CESSATION OF SMOKING
  adolescents, guide to available programs and strategies, 82-1120
  adolescents, older teenagers interacting with younger ones, 82-1125
  behavioral factors, 82-1108
  biological, biochemical, and physiological factors, 82-0924
  business and industry programs for employees, 82-1133
  California, directory of available programs, 82-1139
  cardiovascular disease intervention, the Three Community Study, 82-1036
  cognitive dissonance, self-labeling as predictors of success, 82-1100
  current strategies, review, 82-1134
  Europe, health congress papers, 82-1095
  group cohesiveness as factor in program success, 82-1141
  lifestyle changes, relationship to success, 82-1136
  lifestyle intervention, quit rates in a 10-year program, 82-1122
  mechanical properties of the lungs, effect, 82-1003
  pregnant women, British counseling strategy, 82-1128
  preoperative recommendation for heavy smokers, 82-1022
  psychological support as success factor in Soviet program, 82-1126
  psychosocial influences, 82-1107
  questionnaires vs. biochemical methods for assessment of consumption, 82-1211
  recruitment techniques for high school intervention programs, 82-1135
  recruitment techniques for smoking intervention programs, 82-1129
  relapse and maintenance of the habit, factors, 82-1106
  self-quitting vs. therapeutic cessation, behavioral analysis, 82-1096
  self-reported data vs. Ecolyzer measurement, accuracy, 82-1121
  two-stage vs. three-stage cessation programs, 3-year abstinence rates, 82-1130

CESSATION OF SMOKING, METHODS
  available quitting procedures, review, 82-1105
  current behavioral strategies, review, 82-1143
  home correspondence program, effectiveness, 82-1127
  microcomputer use in health education at urban clinic, 82-1124
  multistage program of preparation, aversion, and maintenance, 82-1141
  social learning and behavior modification approaches, review, 82-1134

CHEWING TOBACCO
  C_{10}-terpenyl ethers as licorice nuance additive, 82-1152
  1981 production, 82-1192

CHILDREN
  decline in smoking in Denmark, 82-1103
  fourth grade students, parental attitudes toward smoking, 82-1102
  health in smoking family study, mathematical error, 82-1205
  initiation of smoking, influences and prevention methods, 82-1101
  passive smoking effects, 82-1066
  psychosocial determinants of smoking habit and preventive strategies, 82-1108
  resource guide for drug abuse prevention/education in New York elementary schools, 82-1132
  respiratory symptoms, parental smoking as factor, 82-1020
  restrictive tobacco sales legislation proposed in Egypt, 82-1196
  seventh grade students, multicomponent smoking prevention program, 82-1118
  smoking habits of 10- to 17-year-old students in Brazil, 82-1093
  social influences, effect on smoking behavior in Norway, 82-1086
  students in Czechoslovakia, smoking habits, 82-1114

CHINA
  international tobacco industry, economic cooperation, 82-1195

CHRONIC BRONCHOPULMONARY DISEASE
  mortality, smoking relationship, 82-0959
  pulp mill workers, smoking relationship, 82-1013

CIGAR SMOKING
  incidence in men aged 34-61 years in Iceland, 82-1094
  oral leukoplakia relationship, 82-0971
  total mortality compared to nonsmokers and cigarette smokers, 82-0959

CIGAR WRAPPERS
  improved humic acid stained paper, 82-1146
  laser perforation of wrapping material, 82-1181
  magnesium oxide additive to reduce visible sidestream smoke, 82-1145

CIGARETTE PAPER
improved humic acid stained paper, 82–1146
laser beam perforation method and apparatus for regulating air flow in cigarettes, 82–1173
laser perforation of wrapping material, 82–1181
magnesium oxide additive to reduce visible sidestream smoke, 82–1145
spark perforator for a moving web, 82–1170
titanium content in Jordanian cigarettes, 82–0938

CIGARETTE SMOKE
arsenic and bromine concentrations in Jordanian and U.S. brand cigarettes, analysis, 82–0938
biological factors in the smoking habit, 82–0924
dilution and filtration effect on particle size, 82–1178
medical risks and efficacy in aversive therapy, 82–1143
nitration process resulting in nitroarenes, 82–0943
physical and chemical characteristics, determinations, 82–0925

CIGARETTES
apparatus for measuring cigarette characteristics during smoking, 82–1185
FTC report of tar, nicotine, and carbon monoxide in 200 varieties, 82–0923
global production and trade, 1980, 82–1199
mathematical model of evaporation—pyrolysis processes when smoldering, 82–0927
percent of total tobacco products sold in Greece, 82–1197
production and consumption, northern vs. southern hemisphere, 82–1195
1981 production and sales, 82–1192
smoldering ignition of mattresses and upholstery, test method, 82–1209
United Kingdom Government Chemist's report on tar, nicotine, and carbon monoxide content of 147 brands, 82–0931

CIGARETTES, HIGH-NICOTINE
United Kingdom, proposed legislative ban, 82–1204

CIGARETTES, HIGH-TAR
United Kingdom, proposed legislative ban, 82–1204

CIGARETTES, LOW-NICOTINE
plasma nicotine concentration, relationship, 82–0940

CIGARETTES, LOW-YIELD
behavioral response to lower nicotine intake, 82–1105
Egyptian trend, 82–1196
flue-cured consumption, tobacco industry effects, 82–1194
high-nicotine content recommended to counteract physiological and psychological effects, 82–1090
improved filter for reducing tar and nicotine by aeration, 82–1182
sales trends in Greece, 82–1197
smoking-related diseases effect, 82–1207

CIGARETTES, NONTOBACCO
simulated cigarette with caffeine and a heat-producing compartment, 82–1140

CIGARS
filter-tipped large cigar, manufacturing method, 82–1172, 82–1176
sales slump from 1978 to 1980 in Greece, 82–1197

CILIARY ACTIVITY
abnormalities, incidence in asymptomatic smokers, 82–0984

COCARCINOGENS
cigarette smoking and occupational exposures as factors in bladder carcinoma incidence in Italy, 82–0981
lung carcinoma, smoking, and general air pollution interactions, 82–0960

COGNITION
adolescents, need for decisionmaking skills in health choices, 82–1098
concepts of addiction and cognitive dissonance, self-labeling as predictors of success, 82–1100

COLON
ulcerative colitis patients, smoking habits, 82–1065

COLORADO
birth weight at high altitude in smoking vs. nonsmoking mothers, 82–1055

COMBUSTION TEMPERATURE
mathematical model of a smoldering cigarette, with evaporation, 82–0927
smoke composition determinant, 82–0925

CONTRACEPTIVES, ORAL
anticoagulants in ischemic stroke prevention, factor with sex, smoking and pregnancy, 82–1038
cardiovascular disease, estrogen vs. progestogen, age and smoking as risk factors, 82–1039
cardiovascular mortality relationship, 82–1049
thromboembolism incidence in U.S. and German women, smoking relationship, 82–1088

COPPER
kidney cortex concentration, determination, 82–0947

CORONARY DISEASE
age, cholesterol and smoking as risk factors, 82–1050
artery disease, family history, smoking and serum cholesterol as risk factors, 82–1026
cessation of smoking program as prevention, 82–1126
Chicago coronary risk intervention program, smoking cessation rates, 82–1122

ischemic heart disease mortality in Finland, risk factors, 82–1040
ischemic heart disease risk factors in the Soviet Union, 82–1046
Japanese males, smoking and alcohol consumption effects, 82–1031
mortality, smoking relationship, 82–0959
native vs. migrated Polynesians, smoking and other risk factors, 82–0957
risk factors in Norway, 82–0954
smoking risk factor, Sweden, 82–1025

COST BENEFITS ANALYSIS
smoking bans in industry, economic benefits, 82–1084

COUGH
mucus clearance in smoking vs. former smoking chronic bronchitis patients, 82–0982

CYANIDES
smoke content reduction by filter material of ferrate compounds, 82–1186

CZECHOSLOVAKIA
schoolchildren's smoking habits, 82–1114

DEMOGRAPHIC FACTORS
cancer patterns in Thailand, implications for control, 82–0973
carboxyhemoglobin levels of U.S. residents, 82–0948
mortality among native and migrated Polynesians, smoking factor, 82–0957
quitting vs. maintaining smoking, economic and other influences, 82–1107

DENMARK
decline in smoking among children, 82–1103

DENTAL DISEASES
oral carcinoma relationship, comparison with smoking effect, 82–0980

DIABETES
retinopathy and basement membrane thickening, smoking relationship, 82–1071
retinopathy and smoking relationship, 82–1062

DNA
tobacco-specific nitrosamines, in vitro binding, 82–0941

DRUG ABUSE
hashish and tobacco smoking, synergistic effect, 82–1019
multiple drug use as factor in maintaining vs. quitting smoking, 82–1107
prevention, recommendations for Federal regulations, 82–1202
psychiatric comparison of smoking with other drug-using behaviors, 82–1104
tiapride as analgesic in alcoholics and smokers slated for cancer surgery, 82–0937

DRUG METABOLISM
acetylsalicylic acid relationship to tobacco smoke effect on platelet aggregate ratio, 82–1027
lignocaine absorption during fibreoptic bronchoscopy, smoking effects, 82–0993
N-desmethyldiazepam plasma concentrations in smokers vs. nonsmokers, 82–0944
oxazepam clearance, smoking, age, and sex effects, 82–0945
ranitidine treatment for peptic ulcer, age, smoking, and alcohol consumption effects, 82–1082
ranitidine treatment of duodenal ulcer, smoking effect, 82–1074
theophylline clearance in smoking and nonsmoking tuberculosis patients, 82–0996
theophylline plasma levels, age and smoking effect on dosage, 82–1017
theophylline requirements in middle-aged vs. elderly patients, smoking effect, 82–1018

DUST
byssinosis from cotton dust exposure, smoking effect, 82–1010
coal mine dust and smoking, interactive effects in gastric neoplasm etiology, 82–0962
occupational exposure and smoking effect, 82–0991
poly(vinyl chloride) exposure and smoking relationship to pneumoconiosis, 82–1008

DYSPNEA
emphysema in upper radiological zone, influence of smoking on lung function, 82–1002

ECONOMICS
cigarette production and consumption, northern vs. southern hemisphere, 82–1195
cigarette tax to fund health care and research, 82–1210
cost of smoking habit to industry, 82–1084
Egypt, tobacco industry in 1980/1981, 82–1196
export tax incentives, Domestic International Sales Corporation program, 82–1193
global cigarette production and trade, 1980, 82–1199
Greek tobacco industry—production, consumption, imports, taxation, and prices, 82–1197
Hungary, price increase and health awareness effects on tobacco consumption, 82–1110
military store tobacco sales, Department of Defense proposal, 82–1200
pros and cons of tobacco price support system, legislative review, 82–1203
recouping public expenditures made for smokers' health care, 82–1206
1981 tobacco industry trends, 82–1192

United States, pricing policy effect on flue-cured tobacco industry, 82–1194
United States, tobacco data for 1980, 82–1198

EGYPT
tobacco industry 1980/1981, 82–1196

EMBRYO
nicotine administration in pregnant rats, effect, 82–1058

EMPHYSEMA
antitrypsin deficiency evaluation, simple method, 82–0986
lung function, annual rate of change in smokers vs. ex-smokers, 82–1002
lung α_1-proteinase inhibitor in smokers vs. nonsmokers, 82–0988
radon daughters, uranium ore dust and cigarette smoke exposure effect in beagle dogs, 82–0963
serum antitrypsin activity, smoking effect in etiology, 82–0989

ENZYME ACTIVITY
Δ-aminolevulinic acid dehydratase activity, smoking and blood lead effects, 82–1069
aryl hydrocarbon hydroxylase levels in lung cancer patients, 82–0972
elastase inhibitory capacity of human sera, evaluation in smokers, 82–0986
high aryl hydrocarbon hydroxylase inducibility associated with smoking, 82–1060
lung α_1-proteinase inhibitor in smokers vs. nonsmokers, 82–0988
platelet monoamine oxidase, reduced in female smokers, 82–0946
pulmonary aryl hydrocarbon hydroxylase dose—response relationship to cigarette smoke in rat lung, 82–0987
serum antitrypsin activity, smoking effect in emphysema etiology, 82–0989

ENZYMES
Δ-aminolevulinic acid dehydratase, 82–1069
aryl hydrocarbon hydroxylase, 82–0972, 82–0987, 82–1060
elastase, 82–0986
monoamine oxidase, 82–0946

ERYTHROCYTES
Δ-aminolevulinic acid dehydratase activity, smoking and blood lead effects, 82–1069
numbers among smokers in Japan, 82–1011
sedimentation rate in predicting cardiovascular diseases in women, smoking effects, 82–1041

ESOPHAGEAL NEOPLASMS
alcohol/tobacco relationship, 82–0974

ESTERS
alicyclic esters as tobacco-cooling additives, 82–1154
alkyl esters of 1-alkanoyl cycloalkanols as tobacco flavorants, 82–1156

EUROPE
mortality trends in men vs. women, "civilization-associated" diseases, 82–0955
political, preventive, therapeutic aspects of smoking, health congress papers, 82–1095

EX-SMOKERS
men aged 34-61 years in Iceland, 82–1094
percentage among Swiss professional women, 82–1087
serum immunoglobulin E levels compared with smokers and nonsmokers, 82–0952

EYE DISEASES
diabetic retinopathy and basement membrane thickening, smoking relationship, 82–1071
diabetic retinopathy and smoking relationship, 82–1062

FEDERAL REPUBLIC OF GERMANY
lung function, smoking and extroversion vs. introversion effects, 82–1006
Task Group 4 report on psychology, prevention, and therapy of smoking, 82–1089
women's smoking habit, 82–1088

FERTILITY
nicotine effects on sperm generation, motility, 82–1075
toxic environmental agents, potential reproductive effects, 82–1052

FETAL GROWTH
reduction, maternal smoking relationship, 82–1054

FETUS
blood flow, pulse rate, and placental circulation, maternal smoking effects, 82–1053
carboxyhemoglobin levels, maternal smoking relationship, 82–1051
nicotine administration in pregnant rats, effects, 82–1058
nonreactive nonstress test results, smoking relationship, 82–1056

FILTERS
aeration groove filter, 82–1174
cadmium and carbon monoxide removal from tobacco smoke, 82–1188
cigarette holder with replaceable filter material, 82–1189
ferrate compounds use, 82–1186
filter-tipped large cigar, manufacturing process, 82–1172, 82–1176

flow-directing grooves for ventilation in cigarettes, 82-1177
hot melt adhesive for filter elements, 82-1180
improvement for reducing tar and nicotine by aeration, 82-1182
lengthened filter in Egyptian cigarette, 82-1196
manufacturing process for smoking products, 82-1183
material for removing injurious smoke components, 82-1187
open-cell foamed polyolefin, 82-1169
particulate matter, effect on size and distribution, 82-1178
polymer fiber, manufacturing process, 82-1190
tar, nicotine, and carbon monoxide in smoke, effect of, 82-0931

FINLAND
cardiovascular disorders, physical activity and other risk factors relationship, 82-1042
ischemic heart disease mortality, risk factors, 82-1040
North Karelia project, hypertension and smoking association, 82-1047

FIRES
smoldering ignition of mattresses and upholstery, cigarette testing, 82-1209

FRANCE
Paris Prospective Study, varicose veins and smoking association, 82-1028

GAS PHASE, CIGARETTE SMOKE
hydrogen cyanide and nitrogen oxides reduction by filter material of ferrate compounds, 82-1186

GASTROINTESTINAL DISEASES
duodenal ulcer healing rates in smokers vs. nonsmokers, 82-1074, 82-1078

GENETICS, PLANTS
altering tobacco smoke condensate concentrations, 82-0916
cultivar differences in smoke components, 82-0918

GEORGIA
byssinosis, information and recommendations of Georgia Thoracic Society, 82-0997

GREECE
cigarette market—production, consumption, imports, taxation, prices, 82-1197

HEALTH EDUCATION
accuracy of smokers' beliefs about their habit, cessation success predictor, 82-1100
advertising ban and health information, consumption effects, 82-1138
decisionmaking skills concerning smoking, need for, 82-1098
efficacy in cessation of smoking, 82-1143
fourth grade students, parental attitudes toward smoking, 82-1102
industry smoking cessation programs for employees, review, 82-1133
microcomputers, evaluation as tool in low-income clinic setting, 82-1124
pregnant women, advice on smoking and alcohol consumption by health care professionals, 82-1123
psychology, prevention, and therapy of smoking in West Germany, 82-1089
resource guide for drug abuse prevention in New York elementary schools, 82-1132
resource guide for drug use/abuse programs in New York secondary schools, 82-1131

HEALTH PRACTICES
home correspondence program effect on smoking cessation and weight reduction, 82-1127
mortality influence, smoking relationship, 82-0955

HEART DISEASES
international mortality trends, men vs. women, 82-0955
risk factors in Norway, 82-0954

HEART FUNCTION
cardiac allergic reactions to tobacco glycoprotein in guinea pigs and rabbits, 82-1032
metoprolol ganglionic blocking effect on nicotine-induced responses, 82-1045

HEART RATE
alkaline and acid nicotine in cigarette smoke, effect, 82-1037

HEMATOCRIT
values among smokers in Japan, 82-1011

HEMOGLOBINS
high altitude relationship to birth weight in pregnant smokers vs. nonsmokers, 82-1055
values among smokers in Japan, 82-1011

HEREDITY
genetic predisposition to occupational and smoking hazards, 82-0956
genetic variability factor in animal models, 82-1076

HISTAMINE
gastric mucosa levels in ulcer patients, smoking effect, 82-1080

HUNGARY
consumption of tobacco products for 1980, 82-1110

SUBJECT INDEX

HYPERCHOLESTEREMIA
- comparisons with low density lipoprotein cholesterol and hypertriglyceridemia as a coronary artery disease risk factor, 82–1031

HYPERTENSION
- adolescents, smoking as factor in the Soviet Union, 82–1043
- cadmium and smoking in malignant hypertension, 82–1047

HYPNOSIS
- smoking cessation method for adolescents, guidebook, 82–1120

ICELAND
- smoking survey among men aged 34-61 years in the Reykjavik area, 82-1094

IMMUNE SYSTEM
- cardiac allergic reactions to tobacco glycoprotein in guinea pigs and rabbits, 82–1032
- chronic obstructive pulmonary disease, immunology and smoking interaction, 82–1014
- malignant melanoma metastasis, smoking as risk factor, 82–0978
- response to inhaled antigens among smokers vs. nonsmokers, 82–0933
- serum immunoglobulin E levels in smokers, ex-smokers and nonsmokers, 82–0952
- suppressive activity of tobacco, 82–0999

IMMUNITY
- response to inhaled antigens among smokers vs. nonsmokers, 82–0933

IMMUNOGLOBULINS
- formation in lungs in response to tobacco antigens, 82–0999
- immunoglobulin E levels, age, sex, and smoking effect, 82–1079
- immunoglobulin E levels in smokers, ex-smokers and nonsmokers, 82–0952

INFANT MORTALITY
- sudden infant death, maternal smoking relationship, 82–1057

INTESTINAL NEOPLASMS
- metaplasia factors in Japan, 82–0975

INTESTINES
- ulcerative colitis patients, smoking habits, 82–1065

ISCHEMIA
- anticoagulants in stroke prevention, sex, smoking, oral contraceptive, and pregnancy effects, 82–1038
- diagnostic and monitoring techniques, smoking effects, 82–1023
- risk factors for ischemic vascular disease, with smoking, 82–1025
- stroke, cranial blood flow velocity in smokers vs. nonsmokers, 82–1030

ISRAEL
- respiratory symptoms in children of smoking parents, 82–1020

ITALY
- bladder carcinoma incidence in Torino, tobacco smoke and occupational exposures relationship, 82–0981

JAPAN
- coronary artery disease in males, smoking and alcohol consumption effects, 82–1031
- high density lipoprotein—cholesterol levels, smoking relationship, 82–1048
- intestinal metaplasia factors, 82–0975
- mortality trends in men vs. women, "civilization-associated" diseases, 82–0955
- pulmonary function deterioration and polycythemia in smokers, 82–1011

JORDAN
- trace element analysis of domestic vs. U.S. cigarette brands, 82–0938

KENYA
- pesticide poisoning from Aldrin among tobacco farm workers, 82–0919

KETONES
- flavor and aroma additives, 82–1168

KIDNEYS
- trace metal concentration in cortex, determination at autopsy, 82–0947

LARYNGEAL NEOPLASMS
- alcohol/tobacco relationship, 82–0964, 82–0974

LARYNX
- degenerate cells in epithelia of rats, smoke exposure effects, 82–1073

LEAD
- blood concentration in smokers and drinkers, 82–0939
- environmental poisoning, smoking effect, 82–1069
- kidney cortex concentration, determination, 82–0947

LEGISLATION
- advertising ban and health information, consumption effects, 82–1138
- air quality standards for total suspended particles in the United States, relaxation proposal debated, 82–0960
- amendments to Medicines Act 1968 proposed in the United Kingdom, 82–1204

export tax incentives, Domestic International Sales Corporation program, 82–1193
military store tobacco sales, Department of Defense proposal, 82–1200
price support system, pros and cons, 82–1203
regulation of smoking in restaurants and health-care facilities, attitude survey in Massachusetts, 82–1201
smoking control laws proposed in Egypt, 82–1196
television advertising of cigarettes ban in United Kingdom, consumption effect, 82–1137
tobacco availability, drug regulation issues, 82–1202

LEUKOCYTES
numbers among smokers in Japan, 82–1011

LEUKOPLAKIA
aryl hydrocarbon hydroxylase inducibility, associated with smoking, 82–1060
malignant transformation risk by site, smoking relationship, 82–1081
prognostic indicators for malignant change, 82–0971

LIPIDS
high density lipoprotein phospholipids in smoke-exposed pigeons, 82–1029

LIPOPROTEINS
atherogenic influence of smoking, 82–1024
coronary disease relationship, with smoking and age, 82–1050
high density cholesterol levels in Japanese populations, smoking relationship, 82–1048
high density cholesterol levels, smoking and exercise effects, 82–1035
high density phospholipids in smoke-exposed pigeons, 82–1029

LIVER
blood lead concentrations in drinkers and smokers, hepatic dysfunction as cause, 82–0939

LUNG DISEASES
chest complications in asymptomatic smokers vs. nonsmokers, 82–0984

LUNG FUNCTION
ABH antigen secretion, protective effect, 82–0998
abnormal pulmonary perfusion scan defects in smokers, prevalence, 82–1021
adult twins, genetic, environmental, and smoking influences, 82–1001
anesthesia and surgery effects, smoking risk, 82–1022
annual rate of change in smoking vs. ex-smoking emphysema patients, 82–1002
deterioration among smokers in Japan, 82–1011
discriminatory analysis in small airways disease diagnosis, smokers vs. nonsmokers, 82–0995
extroversion vs. introversion relationship in smokers vs. nonsmokers, effects, 82–1006
forced oscillation measurements, smokers vs. nonsmokers, 82–1005
harmonic content of forced expiratory flow, smoking effect, 82–1012
hashish and tobacco smoking, synergistic effect, 82–1019
industrial pollutants exposure and smoking effect, 82–1016
mechanical properties of smokers vs. nonsmokers, 82–1003
pulp mill workers, smokers vs. ex-smokers vs. nonsmokers, 82–1013
respiratory resistance among smoking vs. nonsmoking divers, 82–0990
rural study population, smoking effects, 82–0985
test parameters, smoking effect, 82–0983

LUNG NEOPLASMS
age vs. duration of asbestos exposure and smoking in risk assessment, 82–0977
aryl hydrocarbon hydroxylase levels in patients, 82–0972
benzo(*a*)pyrene, passive smoking effect on incidence, 82–1066
cocarcinogenic interaction of smoking and general air pollution, 82–0960
etiological role of tobacco, 82–0974
increase among the population of Thailand, 82–0973
international mortality trends, men vs. women, 82–0955
mortality, smoking relationship, 82–0959
Navajo uranium miners, radiation vs. smoking relationship, 82–0966
passive smoking as factor in etiology, 82–0979
sheesha smoking risk compared to cigarettes, 82–0953
women, 12-month survival rates in smokers vs. nonsmokers, 82–0968

LUNGS
deposition patterns of smoke components, 82–0925
magnetic measurement of ferrimagnetic particles in relation to occupational exposure to asbestos dust, 82–0991
α_1-proteinase inhibitor in smokers vs. nonsmokers, 82–0988
pulmonary alveolar macrophages, differences in smokers vs. nonsmokers, 82–1015
rats, pulmonary aryl hydrocarbon hydroxylase response to cigarette smoke, 82–0987
uranium miners, alveolar macrophage studies, 82–0992

MACROPHAGES
pulmonary alveolar differences in smokers vs. nonsmokers, 82–1015

MASS MEDIA
- cigarette advertising bans in the United Kingdom, consumption effect, 82–1137

MASSACHUSETTS
- regulation of smoking in restaurants and health-care facilities, attitude survey, 82–1201

MATERNAL SMOKING
- carboxyhemoglobin levels in mother and fetus, 82–1051
- consequences to infant and mother, 82–1088
- fetal blood flow, pulse rate, and placental circulation, effects, 82–1053
- health professionals' advice, 82–1123
- high altitude effect on birth weight, relationship, 82–1055
- polycyclic aromatic hydrocarbons, exposure effects on rat fetuses, 82–1059
- reduction or cessation, British counseling strategy, 82–1128
- respiratory symptoms in children, determinant, 82–1020
- spontaneous abortion and low birth weight, relationship, 82–1054
- sudden infant death relationship, 82–1057

MATHEMATICAL MODELS
- evaporation—pyrolysis process inside a smoldering cigarette, 82–0927
- inhalation exposure dosimetry, 82–0934
- statistical critique of smoking/cancer study, 82–0967

MESOTHELIOMA
- risk assessment, 82–0977

METALS, METAL ISOTOPES
- manganese, cadmium, copper, lead, selenium concentrations in human kidney cortex, smoking relationship, 82–0947

MOISTURE CONTENT
- tobacco in a smoldering cigarette, mathematical model with pyrolysis, 82–0927

MORBIDITY
- cigarette tax for funding epidemiological, environmental studies, 82–1210
- occupational exposure and smoking, 82–0961
- postoperative, higher incidence in patients smoking > 10 cigarettes/day, 82–0984

MORTALITY
- byssinosis among flax workers in Northern Ireland, smoking effect, 82–0994
- cardiovascular disease, age, smoking, and oral contraceptive use relationship, 82–1049
- circulatory disorders, physical activity and other risk factors relationship, 82–1042
- "civilization-associated" diseases, international trends in men vs. women, 82–0955
- coronary heart disease and cancer in Norway, risk factors, 82–0954
- female lung cancer patients, survival rates in smokers vs. nonsmokers, 82–0968
- ischemic heart disease in Finland, risk factors, 82–1040
- lung cancer, bronchopulmonary disease, and coronary disease, smoking relationship, 82–0959
- native vs. migrated Polynesians, smoking factor, 82–0957
- time trends in international cancer mortality, 82–0974

MOTIVATION
- adolescent smoking, influences and prevention methods, 82–1101
- adolescent smoking, sex and peer behavior differences, 82–1116
- decline in smoking among Danish children, 82–1103
- illusory psychological benefits from smoking, 82–1092
- psychological and physiological factors of smoking, 82–1111
- schoolchildren's smoking habits in Czechoslovakia, 82–1114
- self-quitting vs. therapeutic smoking cessation, 82–1096
- social influences, effect on smoking habits of schoolchildren in Norway, 82–1086

MOUTH DISEASES
- cheek pouch epithelial dysplasia in hamsters, induced by betel quid, 82–1067
- increased risk from smoking, 82–0969

MOUTH NEOPLASMS
- aryl hydrocarbon hydroxylase inducibility, smoking, and dental status, relationship, 82–0980
- increased risk from smoking, 82–0969
- leukoplakia as a precursor, prognostic indicators, 82–0971
- leukoplakia malignancy risk by site, smoking relationship, 82–1081

MUTAGENS
- ethyl nitrite, generation from combined smoking and drinking, 82–0942
- nitroarenes, generation from cigarette smoke, 82–0943

MYOCARDIAL INFARCT
- arsenic and bromine concentrations in cigarettes, risk factor, 82–0938
- mortality, smoking relationship, 82–0959
- physical activity relationship to other risk factors, 82–1042
- smoking association, 82–1050

  smoking risk factor, Sweden, 82–1025

NASAL MUCOSA
  lignocaine absorption during fibreoptic bronchoscopy, smoking effects, 82–0993

NEONATE
  carboxyhemoglobin levels, maternal smoking relationship, 82–1051
  nicotine administration in pregnant rats, effects, 82–1058

NEOPLASMS
  blood tests in smokers, health congress papers, 82–1095
  correlation with smoking in Stockholm Prospective Study, 82–1025
  dose—response relationship with cigarette consumption, 82–1207
  metastatic risk in malignant melanoma patients, smoking relationship, 82–0978
  risk factors in Norway, 82–0954
  statistical critique of smoking/cancer study, 82–0967
  tiapride as analgesic in alcoholics and smokers slated for cancer surgery, 82–0937
  time trends in international cancer mortality, 82–0974

NEUROTICISM
  maintaining vs. quitting smoking, 82–1107
  smoking relationship, 82–1092, 82–1099

NEW YORK STATE
  chronic respiratory disease, air pollution and smoking effects in New York City, 82–1004
  resource guide for substance abuse prevention/education in elementary schools, 82–1132
  resource guide for substance abuse prevention/education in secondary schools, 82–1131

NEW ZEALAND
  smoking problems, lack of antismoking efforts, 82–1113

NICOTINE
  acute physiological effects, 82–1037
  addiction, smoking as psychiatric disorder, 82–1104
  addiction, theory of smoking maintenance, 82–1111
  addictive adjustments by smokers, 82–1109
  behavioral effects, reinforcement and tolerance, 82–1097
  component comparisons between tobacco and marihuana, possible toxicological significance, 82–0926
  fetal effects of administration in pregnant rats, 82–1058
  first molar tooth germs of mice, possible effects, 82–1068
  genetic alterations, effect on nicotine:condensate ratio, 82–0916
  human aggression, varying dosage effect, 82–1091
  human sperm generation and motility, effect, 82–1075
  low-yield cigarettes, physiological and psychological effects, 82–1090
  physiological basis for smoking initiation in adolescent females vs. males, 82–1112
  role in dependence, maintenance of smoking habit, 82–1106
  saliva and urine concentrations in smokers and passively exposed nonsmokers, 82–0935
  tobacco dependence, influencing factors, 82–1105
  weight regulation effect, 82–1212

NICOTINE CONTENT
  chemical analysis of 147 brands, United Kingdom, 82–0931
  cultivar differences in smoke components, 82–0918
  FTC report of levels in 200 varieties of cigarettes, 82–0923
  lower yield cigarettes, risk assessment, 82–0940
  stalk position and tobacco variety effect on delivery, 82–0930
  varying tobacco strains cultured in presence of nicotine, effect, 82–0920

NICOTINE METABOLISM
  sites and mechanisms of action in rat brain, 82–0932

NICOTINE PHARMACOLOGY
  central nervous system sites and mechanisms for action in rats, 82–0949
  metoprolol ganglionic blocking effect on induced cardiac responses, 82–1045
  tobacco dependence, influencing factors, 82–1105

NICOTINE REDUCTION
  low-tar and -nicotine brands as alternative to abstinence, 82–1144

NITROGEN COMPOUNDS
  ethyl nitrite generation from combined smoking and drinking, 82–0942

NITROGEN OXIDES
  detection in tobacco smoke, apparatus, 82–0922
  smoke content reduction by ferrate filter material, 82–1186
  tobacco denitration method, 82–1179

NITROSAMINES
  sidestream vs. mainstream smoke, concentration, 82–1066
  tobacco-specific, in vitro binding to DNA and guanosine, 82–0941
  volatile, analysis by glass capillary chromatography, 82–0921

SUBJECT INDEX

NONSMOKERS
- acute cardiopulmonary effects of cigarette smoke compared to smokers, 82–1037
- carboxyhemoglobin levels of U.S. residents, 82–0948
- economic benefits to employers, 82–1084
- humoral immune response to inhaled antigens among cigar plant workers, 82–0933
- lung cancer incidence, passive smoking as factor, 82–0979
- nicotine concentrations in saliva and urine, 82–0935
- passive smoking effects, literature review, 82–1066
- serum immunoglobulin E levels compared with smokers and ex-smokers, 82–0952

NORWAY
- Oslo study, risk factors for coronary heart disease and cancer mortality, 82–0954
- schoolchildren, social influences effect on smoking behavior, 82–1086

NUCLEIC ACIDS
- component comparisons between tobacco and marihuana, possible toxicological significance, 82–0926

OBESITY
- home correspondence program for weight loss and smoking cessation, effectiveness, 82–1127

OBSTRUCTIVE AIRWAY DISEASES
- diagnostic value of closing volume tests in smokers vs. nonsmokers, 82–1007
- immunology and smoking interaction, 82–1014
- plasma levels of theophylline, age and smoking effect on dosage, 82–1017
- pulp mill workers, smoking relationship, 82–1013
- theophylline dosage in middle-aged vs. elderly patients, smoking effect, 82–1018

OCCUPATIONAL DISEASES
- bladder carcinoma with smoking as a cocarcinogen, 82–0981
- byssinosis among flax workers in Northern Ireland, 82–0994
- byssinosis in textile workers, smoking effect, 82–1010
- chronic respiratory disease in pulp mill workers, smoking relationship, 82–1013
- gastric neoplasms in coal miners, interactive effects of coal dust and smoking, 82–0962
- lung cancer in Navajo uranium miners, 82–0966
- mesothelioma in asbestos workers, risk factors, 82–0977
- occupational exposures and smoking, interactive effect, 82–0956
- pneumoconiosis, poly(vinyl chloride) exposure and smoking relationship, 82–1008
- potentiating or additive effects of smoking, 82–0961

OCCUPATIONS
- asbestos workers, 82–0977
- chemical workers, 82–1008
- chrysotile asbestos miners and millers, 82–0991
- cigar plant workers, 82–0933
- coal miners, 82–0962
- cotton textile workers, 82–1010
- deep-sea divers, 82–0990
- dockyard workers, 82–1009
- flax workers, 82–0994
- industrial painters, 82–1016
- nurses, 82–1115
- office workers, 82–1016
- policemen, 82–1028
- pulp mill workers, 82–1013
- tobacco farmers, 82–0919
- uranium miners, 82–0966, 82–0992

ORAL HYGIENE
- health hazards of smoking, 82–0969

OXYGEN TRANSPORT
- high altitude effect on birth weight, smoking relationship, 82–1055
- smoking and Po_2 measures in healing of amputation for vascular insufficiency, 82–1063

PARENTAL SMOKING
- children's health study, mathematical error, 82–1205
- respiratory symptoms in children, determinant, 82–1020
- schoolchildren in Norway, effect on smoking behavior, 82–1086

PASSIVE SMOKING
- acute cardiopulmonary effects of sidestream cigarette smoke, 82–1037
- freedom for smokers vs. health risks, 82–1070
- harmful effects, literature review, 82–1066
- health risk controversy, critique of four studies, 82–1072
- lung cancer relationship, 82–0979
- negative ion concentrations, effect of smoking and stearin candle, 82–1085
- nicotine saliva and urine levels from occupational exposure, 82–0935
- protective measures against high carbon monoxide levels, 82–1083
- sidestream cigarette smoke, measurement of deposition in humans, 82–1000

PATENTS
- β-damascone in tobacco flavor enhancement, 82–1165
- 1-acyl-2, 6, 6-trimethylcyclohexene derivatives, processes and uses, 82–1166
- aeration groove filter, 82–1174
- alicyclic amides as tobacco-cooling additives, 82–1154
- alkyl esters, tobacco flavorant process, 82–1156

apparatus for measuring cigarette characteristics during smoking, 82–1185
apparatus for measuring smoke dilution in vented-filter cigarettes, 82–1171
benzodionones for augmenting or enhancing tobacco taste and aroma, 82–1158
C_{10}-terpenyl ethers as organoleptic agents, 82–1152
caryophyllene alcohol mixture for tobacco flavor and aroma, 82–1153
cigarette holder with replaceable filter material, 82–1189
cis-3-hexenyl derivatives as flavorants, 82–1151
cyclohexadiene derivatives, process of preparation as tobacco flavorants, 82–1148
cyclohexane methanol derivatives as flavorants, 82–1164
felt web of viscose fibers for tobacco substitute, process, 82–1175
filter for cigarettes, flow-directing grooves for ventilation, 82–1177
filter material and manufacturing method, 82–1188
filter material for removing injurious smoke components, 82–1187
filter material of ferrate compounds, 82–1186
filter-tipped large cigar, manufacturing method, 82–1172
filtered large cigars, method and apparatus for manufacture, 82–1176
flavor properties of cyclohexadien compound, 82–1150
flavorant additive, pyrazine-containing polyheterocyclic compound, 82–1155
heterocyclic derivatives as tobacco flavorants, 82–1167
hot melt adhesive for filter elements, 82–1180
humic acid-dyed paper for tobacco products, improved process, 82–1146
improved filter for reducing tar and nicotine by aeration, 82–1182
improved tobacco expansion process using carbon dioxide, 82–1191
ketone derivative as an organoleptic agent, 82–1149
ketones as flavor and aroma enhancers, 82–1168
laser perforation of cigarette and cigar wrapping material, 82–1181
manufacturing process and laser beam apparatus to regulate air flow in cigarettes, 82–1173
manufacturing process for ventilated filter-tipped smoking products, 82–1183
nitric oxide in tobacco smoke, detection apparatus, 82–0922
2-oxabicyclooctane derivative for flavor and aroma enhancement, 82–1160
oxabicyclooctane derivatives as flavor/aroma additives, 82–1162, 82–1163
oxabicyclooctane derivatives as tobacco flavor and aroma additives, 82–1161
polyolefin filter, 82–1169
simulated cigarette with caffeine and a heat-producing compartment, 82–1140
spark perforator for ventilated cigarette, 82–1170
spirane derivatives as flavorants, 82–1157
substituted dimethyl dihydroxy benzene and cyclohexadiene compounds as tobacco flavorants, 82–1147
tobacco denitration method, 82–1179
tobacco polymer fiber filter, manufacturing process, 82–1190
tobacco substitute, 82–1184
tobacco wrapper containing magnesium oxide to reduce visible sidestream smoke, 82–1145
tricyclic ketones, flavorants, 82–1159

PEER GROUPS
adolescent attitude toward smoking, relationship, 82–1116
adolescents, factor in decisionmaking on health choices, 82–1098

PEPTIC ULCER
duodenal healing rates in smokers vs. nonsmokers, 82–1074, 82–1078
gastric mucosa histamine levels, smoking effect, 82–1080
ranitidine treatment resistance, age, smoking, and alcohol consumption effect, 82–1082

PERIODONTAL DISEASES
snuff-dipping and smoking effect among Swedish schoolchildren, 82–1077

PERSONALITY
extroversion vs. introversion effects on lung function in smokers vs. nonsmokers, 82–1006
maintenance and cessation of smoking, psychosocial influences, 82–1107
platelet monoamine activity and smoking relationship, 82–0946
smoking motives relationship, 82–1092

PESTICIDES
Aldrin in poisoning of Kenyan tobacco farmers, 82–0919
methyl bromide residues in Jordanian tobacco, 82–0938
toxic environmental agents, potential reproductive effects, 82–1052

PHENOL CONTENT
catechol production by pyrolysis, 82–0929

PHYSICAL ACTIVITY
factor in circulatory disorders, 82–1042
high density lipoprotein cholesterol levels among university students, 82–1035

SUBJECT INDEX

PHYSICAL FITNESS
endurance fitness of middle-aged executives, smoking effects, 82–1033

PIPE SMOKING
incidence in men aged 34-61 years in Iceland, 82–1094
oral leukoplakia relationship, 82–0971
total mortality compared to nonsmokers and cigarette smokers, 82–0959

PIPE TOBACCO
sales slump from 1978 to 1980 in Greece, 82–1197

PLACENTA
blood flow, maternal smoking effect, 82–1053
carbon monoxide effects, sudden infant death relationship, 82–1057
nicotine administration in pregnant rats, effect, 82–1058

PNEUMOCONIOSIS
byssinosis among flax workers in Northern Ireland, 82–0994
byssinosis in textile workers, smoking effect, 82–1010
byssinosis, information and compensation recommendations, 82–0997
poly(vinyl chloride) exposure and smoking relationship, 82–1008

POLAND
residues of six herbicides on tobacco leaves, 82–0917

POLONIUM-210
alpha-emitter activity in the lung, 82–0925
inhalation from tobacco smoke, possible cancer effects, 82–0970

POLYNESIA
mortality in native communities and among migrated natives, 82–0957

PRECANCEROUS CONDITIONS
intestinal metaplasia in Japan, factors, 82–0975
metaplasia in smokers, etretinate treatment effect, 82–1064
oral leukoplakia, prognostic indicators for malignant change, 82–0971

PREGNANCY
anticoagulants in ischemic stroke prevention, factor with sex, smoking and oral contraceptives, 82–1038
fetal blood flow, pulse rate, and placental circulation, maternal smoking effects, 82–1053
health professionals' advice on smoking and alcohol consumption, 82–1123
high altitude relationship to birth weight in smokers vs. nonsmokers, 82–1055
nicotine administration in rats, fetal effects, 82–1058
nonreactive nonstress test results, smoking relationship, 82–1056
smoking risks, health congress papers, 82–1095
toxic environmental agents, potential reproductive effects, 82–1052

PREVENTION OF SMOKING
advertising ban and tobacco tax, suggestions, 82–1208
children and adolescents, methods, 82–1101
current strategies, review, 82–1134
fourth grade students, parental attitudes, 82–1102
multicomponent program for seventh grade students, 82–1118
national policy, recommendations for, 82–1202
recommended measures in West Germany, 82–1089
resource guide for New York programs, elementary schools, 82–1132
resource guide for New York programs, secondary schools, 82–1131

PROGRAMS AND POLICIES
Department of Health and Human Services, goals for the 1980s, 82–1202

PROTEIN CONTENT
tobacco vs. marihuana smoke condensates, analysis, 82–0926

PSYCHIATRIC DISORDERS
mental health implications of smoking, 82–1104
oxazepam clearance, smoking, age, and sex effects, 82–0945

PULMONARY ALVEOLAR MACROPHAGES
structural differences in smokers vs. nonsmokers, 82–1015
uranium miners, smokers and nonsmokers, 82–0992

PULMONARY CLEARANCE
chronic bronchitis patients, smokers vs. ex-smokers, 82–0982

PULMONARY FIBROSIS
parenchymal fibrosis in asbestos-exposed dockyard workers, 82–1009
radon daughters, uranium ore dust and cigarette smoke exposure effect in beagle dogs, 82–0963

RADIATION
causation of lung cancer among Navajo uranium miners, smoking comparison, 82–0966
polonium-210 inhalation from tobacco smoke, possible cancer effects, 82–0970
pulmonary tract tumor induction in beagle dogs, radon daughters, cigarette smoke and uranium ore dust effect, 82–0963

RECIDIVISM
- booster session effect in multistage cessation program, 82–1141
- positive lifestyle changes, effect on maintenance, 82–1136
- two-stage vs. three-stage cessation programs, 3-year abstinence rates, 82–1130
- 10-year coronary intervention program, rates among quitters, 82–1122

REDUCTION OF SMOKING
- cardiovascular disease intervention, the Three Community Study, 82–1036
- current behavioral strategies, review, 82–1143
- methods, progressive tar and nicotine reduction, 82–1144
- older teenagers interacting with younger ones, 82–1125
- pregnant women, British counseling strategy, 82–1128
- rapid smoking vs. increasing delay training, 82–1142
- self-reported rates, accuracy confirmed by Ecolyzer testing, 82–1121

RESPIRATORY FUNCTION TESTS
- ABH antigen secretion, effect in smokers and nonsmokers, 82–0998
- abnormal pulmonary perfusion scan defects in smokers, prevalence, 82–1021
- adult twins, genetic, environmental, and smoking influences, 82–1001
- annual rate of change in smoking vs. ex-smoking emphysema patients, 82–1002
- closing volume test, diagnostic value in obstructive airways disease, smokers vs. nonsmokers, 82–1007
- deterioration among smokers in Japan, 82–1011
- discriminatory analysis in small airways disease diagnosis, smokers vs. nonsmokers, 82–0995
- forced oscillation technique, smokers vs. nonsmokers, 82–1005
- hashish and tobacco smoking, synergistic effect, 82–1019
- lung resistance among smoking vs. nonsmoking divers, 82–0990
- mechanical properties of smokers vs. nonsmokers, 82–1003
- preoperative counseling of heavy smokers, 82–1022
- pulp mill workers, smokers vs. ex-smokers vs. nonsmokers, 82–1013
- rural study population, smoking effects, 82–0985
- spirometric measurements, minimal frequency response requirements, 82–1012
- test parameters, smoking effect, 82–0983
- uranium miners, alveolar macrophage studies, 82–0992

RESPIRATORY SYMPTOMS
- atmospheric sulphur and particulates, smoking role in health effects, 82–0958
- chest x-ray alterations, poly(vinyl chloride) exposure and smoking relationship, 82–1008
- children of smoking parents, 82–1020
- hashish and tobacco smokers, 82–0996, 82–1019
- pulp mill workers, smokers vs. ex-smokers vs. nonsmokers, 82–1013
- rural study population, smoking effect, 82–0985
- uranium miners, alveolar macrophage studies, 82–0992

RESPIRATORY SYSTEM
- alkaline and acid nicotine in cigarette smoke, responses in smokers vs. nonsmokers, 82–1037
- lung function test parameters, smoking effect, 82–0983
- sidestream cigarette smoke deposition, measurement, 82–1000

RESPIRATORY TRACT DISEASES
- air pollution and smoking effect, 82–1004
- parenchymal fibrosis in asbestos-exposed dockyard workers, 82–1009

RESPIRATORY TRACT NEOPLASMS
- radon daughters, uranium ore dust and cigarette smoke, carcinogenic effect in beagle dogs, 82–0963

SALIVA
- nicotine levels in smokers and passively exposed nonsmokers, 82–0935

SELENIUM
- kidney cortex concentration, determination, 82–0947

SEX BEHAVIOR
- adolescent attitude toward smoking, relationship, 82–1116
- characteristics, attitudes of smoking women in United States and Germany, 82–1088

SEX RATIO
- blood lead content and Δ-aminolevulinic acid dehydratase activity, smoking and alcohol effect, 82–1069
- Brazilian youth's smoking habits, 82–1093
- Danish schoolchildren's smoking habits, 82–1103
- immunoglobulin E levels, smoking effect, 82–1079
- professional women vs. professional men, smoking habits in Switzerland, 82–1087
- schoolchildren's smoking habits in Czechoslovakia, 82–1114

SKIN DISEASES
- urticarial vasculitis and obstructive airways disease, immunology and smoking interaction, 82–1014

SMALL AIRWAYS DISEASE
asbestos dust exposure effect, smoking relationship, 82–0991
asymptomatic smokers, incidence, 82–0984
discriminatory analysis in diagnosis, smokers vs. nonsmokers, 82–0995
occurrence among heavy smokers, 82–1022

SMOKE INHALATION, ANIMAL
arterial intima, pathologic effect in rats, 82–1044
growth, survival, and spermatogenic effects in mice, 82–1061
high density lipoprotein phospholipids in pigeons, 82–1029
interactions of smoking machines and proband, effect on bioassay results, 82–0936
laryngeal epithelia in rats, 82–1073
methodology of various systems, description, 82–0951
models for inhalation exposure dosimetry, 82–0934

SMOKE STREAMS
N-nitrosamine concentrations in sidestream vs. mainstream smoke, 82–1066
aerodynamic properties, factors in inhalation assessment, 82–0928
alkaline and acid nicotine, acute physiological effects in smokers vs. nonsmokers, 82–1037
filter material for removing injurious components, 82–1187
mainstream smoke, physical and chemical characteristics, determinations, 82–0925
oxabicyclooctane derivatives as flavor/aroma additives, 82–1162, 82–1163
sidestream cigarette smoke deposition in humans, measurement, 82–1000
sidestream smoke reduction with wrappers containing magnesium oxide, 82–1145
smoke dilution measurement in vented-filter cigarettes, apparatus, 82–1171

SMOKING
monetary cost of related health care to public, proposed "safety tax", 82–1206

SMOKING ARTICLES
cigarette holder with replaceable filter material, 82–1189
improved humic acid stained paper, 82–1146
sheesha smoking, health risks, 82–0953
simulated cigarette with caffeine and a heat-producing compartment, 82–1140

SMOKING CHARACTERISTICS
inhalers among Icelandic men aged 34-61 years, 82–1094
nicotine craving alleviation, methods, 82–1109
number consumed, nicotine content of cigarettes, effect on saliva and urine nicotine levels, 82–0935

SMOKING CONTROL PROGRAMS
business and industry programs for employees, 82–1133
current strategies, review, 82–1134
health congress papers, 82–1095
national programs and policies, recommendations, 82–1202
passive smoking issue, smokers' freedom vs. health risks, 82–1070
resource guide, New York elementary schools, 82–1132
resource guide, New York secondary schools, 82–1131
suggested advertising ban, tobacco tax, 82–1208
United Kingdom, public restrictions and health education effects on consumption, 82–1137
West Germany, psychology, prevention, and therapy of smoking, 82–1089

SMOKING HABIT
adolescents, decisionmaking skills as factor in choice, 82–1098
antismoking activities in the United Kingdom, consumption effect, 82–1117, 82–1137
behavioral aspects, 82–1108
biological, biochemical, and physiological factors, 82–0924
cancer mortality rates relationship, 82–0974
children, ages 12-15, in Norway, 82–1086
children, decline in smoking in Denmark, 82–1103
declining rates in New Zealand, 1976-1981, 82–1113
economic cost to industry, 82–1084
males 34-61 years in Iceland, 82–1094
nicotine, anger, and social factors, role in maintenance, 82–1106
nurses, effect of work-related stress, 82–1115
premature mortality from various causes, relationship, 82–0959
psychological and physiological factors, 82–1111
questionnaires vs. biochemical methods for assessment of consumption, 82–1211
schoolchildren in Czechoslovakia, 82–1114
schoolchildren in Sweden, oral health relationship, 82–1077
students in Brazil, 10- to 17-year-old, 82–1093
tobacco consumption in Hungary, 1975 to 1980, 82–1110
tobacco dependence, influencing factors, 82–1105
ulcerative colitis patients, 82–1065
women, United States and Germany, 82–1088
women vs. men, professionals in Switzerland, 82–1087

SMOKING MACHINES
inhalation system for dose—response studies, 82–0987
interactions of animals and proband, effect on bioassay results, 82–0936
passive inhalation exposures in animals, 82–0951

SMOKING SURVEYS
  Brazil, 10- to 17-year-old students, 82–1093
  Czechoslovakian schoolchildren, 82–1114
  Denmark, decline in smoking among children, 82–1103
  Iceland, men aged 34-61 years, 82–1094
  New Zealand, 1976-1981, 82–1113
  Norwegian children ages 12-15, 82–1086
  Switzerland, professional women vs. men, 82–1087
  ulcerative colitis patients, 82–1065
  United States and Germany, women, 82–1088
  University of Michigan Hospital nurses, 82–1115

SNUFF
  1981 production, 82–1192

SNUFF-DIPPING
  oral health in Swedish schoolchildren, relationship, 82–1077

SOCIOECONOMIC STATUS
  cigarette production and consumption, relationship, 82–1195
  factor in smoking habits of Brazilian youth, 82–1093

SPERMATOZOA
  inhibited formation among smoke-exposed mice, 82–1061
  nicotine effects on generation, motility, 82–1075

SPUTUM
  mucus clearance in smoking vs. former smoking chronic bronchitis patients, 82–0982

STATISTICAL ANALYSIS
  critique of methods used in smoking/cancer study, 82–0967
  health information and advertising ban, consumption effects, 82–1138
  mathematical error in study of children's health in smoking families, 82–1205
  mortality and risk factor analysis in Stockholm Prospective Study, 82–1025

STOMACH NEOPLASMS
  coal miners, interactive effects of coal dust and smoking, 82–0962

STRESS
  nurses, effect on smoking behavior, 82–1115

STUDENTS
  college, high density lipoprotein cholesterol levels, smoking and exercise effects, 82–1035
  elementary schools, resource guide for New York drug abuse prevention/education programs, 82–1132
  high school, recruitment techniques for smoking intervention programs, 82–1135
  secondary schools, resource guide for New York drug use/abuse programs, 82–1131
  seventh graders, multicomponent smoking prevention program, 82–1118
  smoking habits of 10- to 17-year-olds in Brazil, 82–1093

SULFUR COMPOUNDS
  atmospheric sulphur and particulates, smoking role in health effects, 82–0958

SWEDEN
  coronary disease and smoking, the Stockholm Prospective Study, 82–1025
  oral carcinoma, aryl hydrocarbon hydroxylase, smoking, and dental status relationship, 82–0980
  periodontal diseases in schoolchildren, snuff-dipping and smoking effect, 82–1077

SWITZERLAND
  professional women vs. men, smoking habits, 82–1087

TAR CONTENT
  chemical analysis of 147 brands, United Kingdom, 82–0931
  FTC report of levels in 200 varieties of cigarettes, 82–0923

TAR REDUCTION
  improved tobacco expansion process, 82–1191
  low-tar and -nicotine brands as alternative to abstinence, 82–1144

TARS, CIGARETTE
  biological testing in rat pulmonary enzyme system, 82–0987
  dilution and filtration effect on size distribution, 82–1178
  N-nitrosamines, analysis by glass capillary chromatography, 82–0921
  nitration to mutagenic substances, 82–0943
  particle size in tobacco smoke, factors and effects, 82–0928

TARS, TOBACCO
  Burley variety and stalk position effect, 82–0930
  cultivar differences in smoke components, 82–0918
  genetically altering smoke condensate concentrations, 82–0916

TAXATION
  binomial system proposed in Greece with resultant rise in prices, 82–1197
  cigarette tax to fund health care and research, 82–1210
  export incentives, Domestic International Sales Corporation program, 82–1193
  proposed health tax on tobacco, 82–1208
  "safety tax" to pay public expenses made for smokers' health care, 82–1206

  U.S. economic data for 1980, 82–1198
  women's tobacco consumption, effect in Great Britain, 82–1117

TEETH
  nicotine treatment of first molar germs of mice, 82–1068

THAILAND
  cancer patterns, implications for control, 82–0973

THIOCYANATES
  reduction of levels by brand switching, 82–1144

THROMBOEMBOLISM
  oral contraceptives and smoking relationship in U.S. and German women, 82–1088

THROMBOSIS
  clotting factors and thrombotic disorders, smoking risk factor, 82–1034

TOBACCO ADDITIVES
  β-damascone in tobacco flavor enhancement, 82–1165
  1-acyl-2, 6, 6-trimethylcyclohexene derivatives, 82–1166
  alicyclic amides for cooling effect, 82–1154
  alkyl esters of 1-alkanoyl cycloalkanols as flavorants, 82–1156
  benzodionones for taste and aroma enhancement, 82–1158
  C_{10}-terpenyl ethers for chewing tobacco, 82–1152
  caryophyllene alcohol mixture for tobacco flavor and aroma, 82–1153
  cis-3-hexenyl derivatives as flavorants, 82–1151
  cycloheradiene derivatives as flavorants, 82–1148
  cyclohexane methanol derivatives as flavorants, 82–1164
  flavor properties of cyclohexadien compound, 82–1150
  heterocyclic derivatives as flavorants, 82–1167
  ketone derivative as an organoleptic agent, 82–1149
  ketones for flavor and aroma enhancement, 82–1168
  2-oxabicyclooctane derivative, 82–1160
  oxabicyclooctane derivatives as flavor and aroma agents, 82–1161
  oxabicyclooctane derivatives as flavor/aroma agents, 82–1162, 82–1163
  spirane derivatives as flavorants, 82–1157
  substituted dimethyl dihydroxy benzene and cyclohexadiene compounds as additives, 82–1147
  tricyclic ketones, flavorants, 82–1159

TOBACCO AROMA
  1-acyl-2, 6, 6-trimethylcyclohexene derivatives, 82–1166
  alkyl esters of 1-alkanoyl cycloalkanols as additives, 82–1156
  benzodionones as enhancers, 82–1158
  C_{10}-terpenyl ethers for chewing tobacco, 82–1152
  caryophyllene alcohol additive, 82–1153
  cis-3-hexenyl derivatives as flavorants, 82–1151
  cyclohexadiene derivatives, 82–1148
  cyclohexane methanol derivatives as flavorants, 82–1164
  heterocyclic derivatives as additives, 82–1167
  ketone derivative as an organoleptic agent, 82–1149
  ketones as enhancers, 82–1168, 82–1168
  2-oxabicyclooctane derivative, 82–1160
  oxabicyclooctane derivatives, 82–1161, 82–1162, 82–1163
  spirane derivatives as additives, 82–1157
  substituted dimethyl dihydroxy benzene and cyclohexadiene compounds as additives, 82–1147
  tricyclic ketones, 82–1159

TOBACCO CHEWING
  cheek pouch epithelial dysplasia in hamsters, induced by betel quid, 82–1067

TOBACCO CONTAMINANTS
  bromine, strontium, arsenic, and titanium in Jordanian vs. U.S. cigarettes, 82–0938
  residues of six herbicides on tobacco leaves, 82–0917

TOBACCO FLAVOR
  β-damascone for flavor enhancement, 82–1165
  1-acyl-2, 6, 6-trimethylcyclohexene derivatives, processes and uses, 82–1166
  alkyl esters of 1-alkanoyl cycloalkanols as additives, 82–1156
  benzodionones as enhancers, 82–1158
  C_{10}-terpenyl ethers for chewing tobacco, 82–1152
  caryophyllene alcohol additive, 82–1153
  cis-3-hexenyl derivatives as flavorants, 82–1151
  cyclohexadiene derivatives, 82–1148
  cyclohexane methanol derivatives as flavorants, 82–1164
  heterocyclic derivatives as additives, 82–1167
  ketone derivative as an organoleptic agent, 82–1149
  ketones as enhancers, 82–1168
  2-oxabicyclooctane derivative, 82–1160
  oxabicyclooctane derivatives, 82–1161, 82–1162, 82–1163
  properties of cyclohexadien compound, 82–1150
  pyrazine-containing polyheterocyclic compound additive, patent, 82–1155
  spirane derivatives as flavorants, 82–1157
  substituted dimethyl dihydroxy benzene and cyclohexadiene compounds as additives, 82–1147
  tricyclic ketones, flavorants, 82–1159

TOBACCO INDUSTRY
  economic assistance needed in emerging countries, 82–1195
  Egypt, production, sales, consumption, prices, legislation, 82–1196

- export tax incentives, Domestic International Sales Corporation program, 82–1193
- global cigarette production and trade, 1980, 82–1199
- Greece—production, consumption, imports, taxation, and prices, 82–1197
- pros and cons of proposed price support legislation, 82–1203
- United States, consumption patterns and pricing policies, effect on flue-cured market, 82–1194
- United States, economic data for 1980, 82–1198
- upward trends for 1981, 82–1192

TOBACCO LEAF COMPONENTS
- catechols produced by pyrolysis, 82–0929
- denitration process, 82–1179
- genetically altering smoke condensate concentrations, 82–0916
- variety and stalk position effect, 82–0930

TOBACCO PROCESSING
- apparatus for measuring cigarette characteristics during smoking, 82–1185
- denitration process, 82–1179
- expansion process improvement using carbon dioxide, 82–1191

TOBACCO PYROLYSIS PRODUCTS
- Burley variety and stalk position effect, 82–0930
- catechol formation, 82–0929
- genetically altering smoke condensate concentrations, 82–0916

TOBACCO SMOKE
- biological factors in the smoking habit, 82–0924
- Burley variety and stalk position effect on constituents, 82–0930
- chlorophyll content effect, 82–0918
- filter material for removing injurious components, 82–1187
- filtration method for cadmium and carbon monoxide, 82–1188
- genetically altering condensate concentrations, 82–0916
- interactions of smoking machines and animals, effect on bioassay results, 82–0936
- marihuana smoke condensate comparison via capillary gas chromatography/mass spectrometry, 82–0926
- models for inhalation exposure dosimetry, 82–0934
- nitric oxide detection apparatus, 82–0922
- pyrolytic production of catechols, 82–0929

TOBACCO SUBSTITUTES
- felt web of viscose fibers, production method, 82–1175
- nicotine chewing gum, 82–1109
- synthetic film with incorporated blowing agent, 82–1184

TOBACCO VARIETIES
- Burley, cultivar and stalk position effect on smoke components, 82–0930
- catechol production by pyrolysis, 82–0929
- genetically altering smoke condensate concentrations, 82–0916
- nicotine uptake by Burley 21, Robinson Medium Broadleaf in suspension culture, 82–0920

TONGUE DISEASES
- malignant transformation risk of sublingual keratosis, smoking relationship, 82–1081

TRIGLYCERIDES
- higher plasma levels associated with smoking, 82–1033
- levels in Japanese males, coronary artery disease relationship, 82–1031

TRYPTOPHAN
- excretion in smoking vs. nonsmoking bladder cancer patients vs. controls, 82–0965

TUBERCULOSIS
- theophylline clearance in smoking and nonsmoking patients, 82–0996

TWINS
- genetic and environmental influences on pulmonary function, 82–1001

UNION OF SOVIET SOCIALIST REPUBLICS
- adolescent elevated blood pressure, smoking relationship, 82–1043
- international tobacco industry, economic cooperation, 82–1195
- ischemic heart disease risk factors, 82–1046
- psychological support as success factor in smoking cessation program, 82–1126

UNITED KINGDOM
- antismoking activities effect on tobacco consumption, 82–1117, 82–1137
- byssinosis among flax workers in Northern Ireland, 82–0994
- Government Chemist's report on tar, nicotine, carbon monoxide content of 147 brands, 82–0931
- legal recommendations to counter health hazards of smoking, 82–1204
- pregnant women, counseling strategy to reduce cigarette consumption, 82–1128
- respiratory symptoms in children of smoking parents, 82–1020

UNITED STATES
- air quality standards for total suspended particles, pros and cons of relaxation, 82–0960
- mortality trends in men vs. women, "civilization-associated" diseases, 82–0955

respiratory symptoms in children of smoking parents, 82–1020
women's smoking habit, 82–1088

URANIUM
pulmonary tract tumor induction in beagle dogs, with cigarette smoke and radon daughters, 82–0963

URINE
nicotine levels in smokers and passively exposed nonsmokers, 82–0935
pH level, possible influence on nicotine intake, 82–1112
tryptophan levels in smoking vs. nonsmoking bladder cancer patients vs. controls, 82–0965

VASCULAR DISEASES
anticoagulants in ischemic stroke prevention, smoking effect, 82–1038
healing of amputation for vascular insufficiency, smoking relationship, 82–1063
thrombosis and clotting factors, smoking effect, 82–1034
varicose veins and smoking association with arteriosclerosis, 82–1028

VITAMIN A
retinoid treatment for metaplasia in heavy smokers, 82–1064

WITHDRAWAL SYMPTOMS
coping strategies for adolescents who quit smoking, 82–1120
description, 82–1105

WOMEN
acid urine influence on nicotine intake, 82–1112
advice on smoking and alcohol consumption given during pregnancy, 82–1123
cardiovascular disease, relationship to age, smoking, and oral contraceptive use, 82–1039, 82–1049
erythrocyte sedimentation rate in predicting cardiovascular diseases, smoking effects, 82–1041
lung cancer 12-month survival rates in smokers vs. nonsmokers, 82–0968
platelet monoamine oxidase activity in smokers vs. nonsmokers, 82–0946
professionals in Switzerland, smoking habits, 82–1087
smoking characteristics and consequences to offspring, United States and Germany, 82–1088
total mortality and smoking relationship, 82–0959
work-related stress and smoking among nurses, 82–1115

☆U.S. GOVERNMENT PRINTING OFFICE: 1982-361-166:356

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

SMOKING AND HEALTH LITERATURE SEARCH REQUEST

Individual requesting search	Title or position
Individual other than requester completing form	Telephone number
Organization	Date of request
Address including zip code	**Purpose of search** ☐ Research ☐ Paper presentation ☐ Writing article ☐ Lecture ☐ Other __________

SEARCH REQUIREMENTS Give below a detailed statement of information requirements together with any necessary background data to meet your needs. Be as specific as possible to help formulate search strategy.

If topic has been reviewed or cited in past issues of the *Smoking and Health Bulletin* or *Bibliography on Smoking and Health* give pertinent accession number(s) or citation of articles.

Give below other articles which may serve as a guide on subject area (list full bibliographic citation if known)

List relevant key words or index terms appropriate to search.

If a search has been performed on subject utilizing other data sources, please list sources, results of search and search strategy if known.

SEARCH RESTRICTIONS Check appropriate search out-put desired.

☐ Full range (no restrictions)

☐ Year(s)_______________only

☐ Citation only (no abstract)

☐ English language only

☐ Other language _______________________

____

AGENCY USE ONLY. Do Not Write Below This Line.

Search statement or criteria	Date search received Date search released
	Number of documents retrieved

NOTICE

If you no longer need this publication, check here ____, return this sheet to the address below, and your name will be dropped from the mailing list.

If you have a change in name or address, please write the change on this sheet and return it to the address below.

____

____

____

WHEN REQUESTING A CHANGE PLEASE BE SURE YOUR FORMER LISTING ALSO APPEARS ON THIS SHEET.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health
Rockville, Maryland 20857

U.S. MAIL

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS
BOOK

82/5

SEPTEMBER-OCTOBER 1982
Abstracts 82-1213-82-1533

Smoking and Health Bulletin

NEWARK PUBLIC LIBRARY
NOV 23 1982
U. S. GOVT. PUBS.
DEPOSITORY 376

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

HE 20.7211:

INTRODUCTION

All items appearing in the Smoking and Health Bulletin become part of the permanent document collection of the Technical Information Center (TIC) of the Office on Smoking and Health and can be referred to by their accession number.

In the past, at the end of each calender year, it has been customary to publish the Bibliography on Smoking and Health which çontained a cumulative author and subject index based on the individual Bulletins issued that year. However, this year, due to fiscal and other constraints, it may not be possible to issue the index in the form of the Bibliography. All readers are therefore advised to keep the individual Bulletin issues for both 1981 and 1982 until further notice.

The TIC offers bibliographic search and retrieval services for information on smoking, tobacco, and tobacco use through its Automated Search and Retrieval System. Readers are encouraged to submit searches on the search request form found at the end of each Bulletin issue. Multiple copies of this form can be ordered from the TIC. Currently the automated file contains approximately 25,000 records, the majority of which were published and processed into the TIC collection since 1970. The TIC collection totals more than 35,000 items.

Bibliographic and reference services continue to be offered. Readers can take advantage of these services by writing or calling the TIC at the number listed below. Staff limitations currently exists for photocopying services, therefore your cooperation is sought when requesting such services. Until further notice no more than three items per Bulletin per request can be provided. Requests for services above this three document limit cannot be honored. In order that we may continue to provide photocopying services to the largest number of readers, we ask before requesting such items from the TIC collection, to first check your local resources regarding their availability. Visitors may use the TIC library during regular office hours, 8:30 a.m. to 5:00 p.m. weekdays. Advance arrrangements of any planned visits are suggested.

We welcome suggestions or comments from our readership regarding our publications and services.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Telephone: 301-443-1690

ISSN: 0081-0363
September–October 1982
Abstracts 82-1213–82-1533

Smoking and Health Bulletin

CONTENTS

CHEMISTRY 281
PHARMACOLOGY AND TOXICOLOGY 283
MORTALITY AND MORBIDITY 289
NEOPLASTIC DISEASES 290
NON-NEOPLASTIC RESPIRATORY DISEASES 301
CARDIOVASCULAR DISEASES 309
PREGNANCY AND INFANT HEALTH 317
OTHER DISEASES AND CONDITIONS 320
BEHAVIORAL AND PSYCHOLOGICAL ASPECTS 325
SMOKING PREVENTION AND INTERVENTION 335
SMOKING CESSATION METHODS 340
TOBACCO PRODUCT ADDITIVES 341
TOBACCO MANUFACTURING AND PROCESSING 343
TOBACCO ECONOMICS 346
LEGISLATION 348
GENERAL 349
AUTHOR AND ORGANIZATIONAL INDEX i
SUBJECT INDEX vii

CHEMISTRY

82–1213. Arany-Fuzessery, K.; Fugedi-Nemeti, M. **Determination of Benzo/a/pyrene in Cigarette Smoke.** *Acta Alimentaria* 10(4): 329-336, 1981.

A spectrophotometric method for the determination of benzo(*a*)pyrene (BaP) based on ultraviolet absorption is described. A difficulty in the determination of this compound lies in the fact that it is present in smoke only in minute amounts (some ng per cigarette) and it is essential to separate it from the great number of other compounds present in cigarette smoke. A multistep purification and separation procedure, based on a combination of steps of the methods found in related literature, was developed. This method is suitable, within the reproducibility and recovery limits, for the quantitative determination of BaP in the smoke of different cigarettes, and for establishing the efficiency of filters of various types aimed at reducing the harmful components of cigarette smoke. (Auth. Abs. Mod.)

82–1214. Blum, J. K.; Kaupass, R. **The Origin of Manganese in Cigarette Smoke and Ash.** *Naturwissenschaften* 69(2): 93-94, 1982.

Whole cigarettes, and the paper and tobacco separately, were investigated for the source of manganese. Ten popular brands were subjected to x-band spectrometry. The E.P.R. spectra indicate that the manganese ions originate in manganese-containing calcite powder which is used as an additive in the manufacturing of both the paper and the tobacco.

82–1215. Gyuzelev, L.; Poeva, S.; Biryukova, N. E.; Mokhnachev, I. G. **Spektrofotometrichen Analiz na Tyutyunevitya Dim. [Spectrophotometric Analysis of Tobacco Smoke.]** *Bulgarski Tyutyun* 27(2): 37-40, 1982, Bulgarian.

A relatively simple and accurate method is introduced for determining the efficiency of a cigarette filter by total spectrophotometric analysis of the yielded tobacco smoke. The method consists of collecting cigarette smoke in ethanol and comparing ultraviolet (UV) and visible absorption spectra of the smoke solutions. Determination of the filter efficiency is based on increases in the extinction coefficient at decreasing wavelength and the identical sequence of both UV and visible spectra of different tobacco samples. The smoke retention capacity of a filter is calculated from the difference between the area below the smoke absorption curve from the filtered vs. nonfiltered cigarette. The effect of smoke composition on retention capacity of a cigarette filter is shown by comparing absorption selectivity of smoke from trapezund 4th class tobacco variety with that from Dyubek 2nd class variety. Different smoke compositions have different retention patterns. Retention capacity of a filter is correlated with spectral wavelength and, at equal wavelengths, increases with increasing filter length.

82–1216. Lowe, R. H.; Sheen, S. J. **Accumulation of Soluble Proteins and Nitrogenous Compounds in the Leaf of Bright and Burley Tobaccos During the Growing Season.** *Beitraege zur Tabakforschung International* 11(3): 161-169, January 1982.

In studying tobacco as a protein crop, it was found that Fraction I protein (FI protein) and Fraction II protein (FII protein) in bright (NC95) and Burley (Ky14) tobaccos were similar. Their quantities reached maximal levels around flowering, followed by a gradual decline. However, the loss of FI protein was much more rapid than that of FII protein during leaf maturation. The 2-year results revealed that Ky14 contains significantly more FI protein, insoluble proteins, total proteins, free amino acids, total nitrogen, nitrate nitrogen, and total alkaloids than NC95, if averaged over the growing season. The latter showed a greater amount of chlorophyll only. There was no difference in FII protein concentration between the two cultivars. The estimated yields of soluble proteins in Ky14 at the immature plant stage, about 1 week before topping, and by harvest time are 888, 470, and 281 kg/ha, respectively. When including both soluble and insoluble proteins, the immature plants of NC95 could yield 1,387 kg/ha of protein, in comparison to 1,308 kg/ha for Ky14. Multiple harvests of regrowth can further the protein yield per ha. (Auth. Abs. Mod.)

82–1217. McCoy, G. D.; Hecht, S. S.; Katayama, S.; Wynder, E. L. **Differential Effect of Chronic Ethanol Consumption on the Carcinogenicity of** *N*-Nitrosopyrrolidine and *N'*-Nitrosonornicotine in Male Syrian Golden Hamsters. *Cancer Research* 41: 2849-2854, July 1981.

The carcinogenicity of *N*-nitrosopyrrolidine (NPYR) was greatly enhanced in chronic ethanol-consuming Syrian golden hamsters. This is suggested by the results of an investigation of the effect of chronic ethanol consumption on the carcinogenicity of NPYR and *N'*-nitrosonornicotine (NNN) in male Syrian golden hamsters. Groups of hamsters were maintained on either ethanol-containing or control liquid diets for 4 weeks prior to and during carcinogen treatment. NPYR or NNN was administered to ethanol-consuming or control hamsters by intraperitoneal (i.p.) injection over a 25-week period in total doses of either 1 or 2 mmol. In the group treated with 1 mmol of NPYR and maintained on a control diet, 1 out of 20 hamsters developed a nasal cavity tumor and 4 out of 20 hamsters developed tracheal tumors. In the group treated with 1 mmol of NPYR and maintained on the ethanol-containing diet, 8 out of 18 hamsters developed nasal cavity tumors and 9 out of 18 hamsters developed tracheal tumors. The corresponding results in hamsters given 2 mmol of NPYR were: nasal cavity tumors, 14 out of 21 (control) and 16 out of 17 (ethanol); tracheal tumors, 8 out of 21 (control) and 11 out of 17 (ethanol). The results demonstrate that the carcinogenicity of

NPYR is enhanced in ethanol-treated Syrian golden hamsters, particularly at the lower dose. In the groups treated with 1 mmol of NNN and a control diet, 1 out of 21 hamsters developed a nasal cavity tumor and 4 out of 21 developed tracheal tumors. In the corresponding ethanol-treated groups, 1 out of 17 hamsters had a nasal cavity tumor and 5 out of 17 developed tracheal tumors. In the hamsters given 2 mmol of NNN, the results were: nasal cavity tumors, 5 out of 21 (control) and 4 out of 21 (ethanol); tracheal tumors, 9 out of 21 (control) and 7 out of 21 (ethanol). The carcinogenicity of NNN in the Syrian golden hamster was not affected by ethanol treatment; however, the metabolic activation of NPYR, but not that of NNN, may be enhanced by chronic ethanol consumption in the Syrian golden hamster. (Auth. Abs. Mod.)

82–1218. McCusker, K.; Hiller, F. C.; Wilson, J. D.; McLeod, P.; Sims, R.; Bone, R. C. **Dilution of Cigarette Smoke for Real Time Aerodynamic Sizing With a SPART Analyzer.** *Journal of Aerosol Science* 13(2): 103-110, 1982.

A three-stage dilution system has been constructed which mixes a puff of smoke immediately after generation with filtered air without apparent coagulation. Aerosol particles are then serially aspirated and mixed with clean air to achieve dilution of 126,000:1. Using this dilution system, it was possible to analyze the aerodynamic size distribution of cigarette smoke with a single particle aerodynamic relaxation time (SPART) analyzer with minimum coincidence loss. The cigarette smoke leaving the butt end of a cigarette was found to have a particle concentration of 4.2 x 10^9 particles/cm^3, with mass median aerodynamic diameter of 0.43 μm. The experimental design and calibration of the dilution are briefly described. (Auth. Abs.)

82–1219. Sheen, S. J.; Lowe, R. H.; Burton, H. R. **Leaf Proteins and Chemical Constituents in Tobacco Chlorophyll Genotypes.** *Beitraege zur Tabakforschung International* 11(3): 170-179, January 1982.

Tobacco can be grown for the production of both water-soluble and insoluble proteins as main products and deproteinized fibers as a byproduct. In this study, bright and Burley tobacco breeding lines carrying the chlorophyll-deficiency genes *Py* and *yg* were compared with normal genotypes, dark fire-cured, and Maryland tobacco types for the quantity of proteins and other constituents. Varietal difference in soluble leaf protein content was not observed in the immature plant stage but became evident at leaf maturation. Fraction I (FI) protein accumulated to a maximum level of about 130 mg/g dry weight in all tobaccos analyzed before topping. The greatest quantity of Fraction II (FII) protein, ranging from 100 to 160 mg/g dry weight, occurred 1 to 2 weeks after maximum FI protein accumulation. FI protein concentration decreased at a faster rate than FII protein during leaf maturation and senescence. Among the chlorophyll genotypes, Ky14 and Burley 21-*yg* had the highest amounts of soluble leaf proteins and the lowest levels of plastid pigments and/or chlorogenic acid. (Auth. Abs. Mod.)

82–1220. Sterling, T. D.; Dimich, H.; Kobayashi, D. **Indoor Byproduct Levels of Tobacco Smoke: A Critical Review of the Literature.** *Journal of the Air Pollution Control Association* 32(3): 250-259, March 1982.

The contribution of cigarette smoke to indoor air pollution is difficult to assess: presently available data and measuring practices are useful, but serious methodological problems persist. Test conditions which must be accommodated in determining sidestream smoke exposure are summarized, including ventilation, size and type of premises, monitoring conditions, number of smokers, and rate of smoking. Current methodology and major findings are critically evaluated and reviewed. Tables are given of common or average levels of cigarette smoke components, such as acrolein, aldehydes, aromatic hydrocarbons, carbon monoxide, nicotine, nitrogen oxides, nitrosamines, particulates.

82–1221. U.S. Department of Health and Human Services, National Toxicology Program. **Second Annual Report on Carcinogens.** U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Publication No. NTP 81-43, December 1981, 447 pp.

All substances, either known or suspected to be carcinogens and to which a significant number of persons residing in the United States are exposed, are listed along with summarized information on the nature and degree of exposures, and the extent to which Federal regulations are effective in reducing the risk to the public health. Many compounds (e.g., acridenes, aromatic hydrocarbons, arsenic, asbestos, benz(*a*)anthracene, benzo(*a*)pyrene, beryllium, cadmium, carbazoles, chromium, dibenzanthracenes, and nitrosonornicotine) found in cigarette smoke or synergistic with it appear among the 88 chemicals listed. Requests to the Department of Health and Human Services for chemical testing and information, as mandated by law, are tabulated. The appendices include an alphabetical list of substances discussed and their synonyms, a glossary, an acronyms listing, and measurements commonly used. Two precautions with respect to the summaries are issued: (1) science and society have not arrived at a final consensus on the definition of a carcinogen; and (2) regulatory expression, in all but one case on food additives and colors, is the judgmental interpretation by the regulator of its Congressional mandates.

PHARMACOLOGY AND TOXICOLOGY

82-1222. Badawy, A.A.-B.; Evans, M.; Punjani, N. F. **Reversal by Naloxone of the Effects of Chronic Administration of Drugs of Dependence on Rat Liver and Brain Tryptophan Metabolism.** *British Journal of Pharmacology* 74(2): 489-494, October 1981.

Chronic administration of ethanol, morphine, nicotine, or phenobarbitone has previously been shown to enhance rat brain 5-hydroxytryptamine (5-HT) synthesis. This effect is due to the NADPH-mediated inhibition of liver tryptophan pyrrolase activity, thus increasing the availability of circulating tryptophan to the brain. The opiate antagonist, naloxone, reverses the above enhancement of 5-HT synthesis, the accompanying increase in tryptophan availability to the brain, and inhibition of liver tryptophan pyrrolase activity. It is suggested that naloxone exerts these effects by antagonizing the chronic drug-induced increase in liver (NADPH). Naloxone increases serum corticosterone concentration in rats chronically treated with the above four drugs of dependence. Possible explanations of this effect are discussed. (Auth. Abs. Mod.)

82-1223. Dehnen, W.; Verbuecheln, J. **Untersuchungen ueber die Induktion der Benzo(*a*)pyren Hydroxylase und die Bindung von Benzo(*a*)pyren an die DNA der Lunge in vivo unter der Einwirkung von Schwebstoffextrakt nach pertrachealer Applikation. [Studies on the Induction by Extracts From Airborne Particulates of Benzo(*a*)pyrene Hydroxylase and the Binding of Benzo(*a*)pyrene to DNA in the Rat Lung In Vivo After Endotracheal Administration.]** *Zentralblatt fur Bakteriologie Parasitenkunde, Infectionskrankheiten und Hygiene; Erste Abteilung; Originale, Reihe B.* 174(3): 260-273, 1981, German.

A method is described for the administration of suspended material into the rat lung. The rat is narcotized by CO$_2$/air, the trachea is punctured, and the suspension is sucked into the lung by the inhaled air (endotracheal administration, p.t.a.). Benzo(*a*)pyrene (BaP) administered by this method is eliminated from the lung rapidly, while metabolites persist for a longer period. The radioactivity detected in the blood can be attributed predominantly to metabolites; a small fraction is unmetabolized BaP. The activity of BaP hydroxylase is induced in the lung after p.t.a. of extracts from airborne particulate matter. The activity increases depending on the dose until a plateau of activity is reached at high doses. Endotracheally administered BaP is bound to DNA of the lung. Pretreatment with extracts p.t.a. enhances the binding. After enzymatic digestion of DNA, BaP-nucleoside adducts are detected by high power liquid chromatography. According to published data the adducts are supposed to be derived from BaP-phenols and BaP-diolepoxids. It is concluded that the components of airborne particulates can modify the activity of metabolizing enzymes and increase the rate of formation of adducts in the lung in vivo. (Auth. Abs.)

82-1224. Fuder, H.; Siebenborn, R.; Muscholl, E. **Nicotine Receptors Do Not Modulate the ^{3}H-Noradrenaline Release From the Isolated Rat Heart Evoked by Sympathetic Nerve Stimulation.** *Archives of Pharmacology* 318(4): 301-307, March 1, 1982.

Evidence is given for the presence of nicotine receptors in the rat heart, stimulation of which by nicotine and PAPETA (p-aminophenethyltrimethylammonium) caused a small increase in ^{3}H-noradrenaline overflow. Isolated rat hearts with the right sympathetic nerves attached were perfused with Tyrode's solution. (—)-^{3}H-Noradrenaline was then infused to label the noradrenaline stores. After washout the sympathetic nerves were stimulated electrically. In the absence of stimulation, nicotine and PAPETA increased the output of ^{3}H-noradrenaline by activation of nicotine receptors. The amount of transmitter released, however, was small. Neither drug affected ^{3}H-noradrenaline output during electrical nerve stimulation. The role of cocaine in inhibiting the neuronal uptake of PAPETA is also examined.

82-1225. Greenblatt, D. J.; Sellers, E. M.; Shader, R. I. **Drug Therapy: Drug Disposition in Old Age.** *New England Journal of Medicine* 306(18): 1081-1088, May 6, 1982.

Current knowledge of changes in drug absorption, distribution, and clearance with advancing age is reviewed. The physiological factors involved in aging, as well as sex, alcohol consumption, and smoking effects on drug metabolism, are considered. Cigarette smoking is known to stimulate hepatic metabolizing capacity for a number of drugs, increasing total metabolic clearance. The effect may be greater in the young than in the elderly, making data from pharmacogeriatric studies difficult to interpret. Smoking is more likely to be prevalent in the young population, since early mortality tends to select against heavy smokers in the geriatric population. However, even if smoking were equally prevalent in young and elderly groups, the reduced inducibility in the geriatric population would mean that the effects of smoking on drug clearance would tend to magnify age-related differences. (Auth. Abs. Mod.)

82-1226. Gupta, M.; Chatterjee, T. **Effects of Citrinin on Cigarette Smoke Inhaled and Nicotine Treated Mice.** *Indian Drugs* 18(9): 309-313, June 1981.

The mortality rate due to smoke inhalation was significantly reduced in mice injected with large doses of citrinin. Doses of 5.00, 6.25, or 7.50 ml/kg of body weight were administered 30 minutes prior to either smoke or nicotine exposure. Excitation, tremor, and convulsion due to tobacco exposure were significantly decreased. With higher doses of citrinin (25.0 and 30.0 mg/kg), mortality was completely blocked. In the nicotine-treated mice, polyglycol (the solvent vehicle) alone also prevented mortality from exposure. The exact mechanism of citrinin's antagonistic action with nicotine is not known. Possibilities include its central nervous system depressant activities, change in acetylcholine concentrations in the brain, production of hyperglycemia, or sedative mechanisms.

82–1227. Hibberd, A. R.; Gorrod, J. W. **Nicotine Δ1'(5)' Iminium Ion: A Reactive Intermediate in Nicotine Metabolism.** *Advances in Experimental Medicine and Biology* 136B: 1121-1131, 1982.

In studying nicotine's metabolism to cotinine, nicotine Δ1'(5)' iminium ion, a metabolic intermediate, was reacted with glutathione (GSH) and with cyanide. The reaction results were analyzed via chromatography, mass spectrometry, melting point determination, and nuclear magnetic resonance and ultraviolet spectroscopy. The spectroscopy characteristics of the compound formed by cyanide and the ion were in close agreement with the literature values for 5' cyanonicotine. Data from metabolic experiments with rabbit and guinea pig tissues suggest that a pathway to cotinine, mediated by an hepatic soluble enzyme, exists via 5' cyanonicotine. The presence of both GSH and nicotinyl fragments in the mass spectra indicate a nicotinyl-GSH adduct from the ion-GSH reaction. The production of the cyano and GSH iminium ion adducts by chemical reaction in vitro show that covalent bonding with tissue nucleophiles might occur during nicotine metabolism, thus playing a role in toxic reactions and carcinogenesis.

82–1228. Husum, B.; Wulf, H. C.; Niebuhr, E. **Increased Sister Chromatid Exchange Frequency in Lymphocytes in Healthy Cigarette Smokers.** *Hereditas* 96: 85-88, 1982.

Cigarette smokers have increased rates of sister-chromatid exchanges (SCE) in peripheral lymphocytes. In a group of 149 healthy, unmedicated persons, 60 nonsmokers had a mean of 8.25 SCE per cell (standard deviation=0.13) and 89 smokers had 9.23 (0.14) per cell. The sum of SCE in 30 metaphases in each subject was found to be significantly increased with increasing cigarette consumption. An increased SCE rate reflects the influence of mutagens, and lung cancer has been associated with both normal and increased SCEs. This association of SCE rate and cigarette smoking should be considered when using the SCE test system to evaluate in vivo exposure to environmental mutagens. (Auth. Abs. Mod.)

82–1229. Jaffe, J. H.; Kanzler, M.; Friedman, L.; Kaplan, T. **Money and Health Messages as Incentives for Smoking Low Tar/Nicotine Cigarettes: Changes in Consumption and Exhaled Carbon Monoxide.** *British Journal of Addiction* 77(1): 21-34, March 1982.

Cigarette brands advertised as "low" tar are increasing sales yearly, undoubtedly because smokers hope they are less hazardous than "high" tar/nicotine cigarettes. However, it is questionable how much switchers to "low" tar/nicotine brands actually reduce their exposure to toxic components in smoke. A switching project, which examined the cigarette purchases over 15 weeks of 121 paid volunteers in four experimental and one control group (separately within sex), is reported. Experimental groups received differing combinations of money and/or health message incentives to encourage switching to brands with lower tar/nicotine delivery. Subjects in all but one experimental group significantly decreased tar/nicotine exposure; controls did not. Money incentives produced the greatest decrease. The effect of health messages was unclear. Switchers' exhaled carbon monoxide (CO) remained higher than would be expected from the theoretically lower CO delivery of their brands. Small increases in number of cigarettes smoked per day did not account adequately for the persistently high exhaled CO. However, the data on CO indicates that there were substantial changes in puffing patterns. The observations thus support previous reports that changes in puffing patterns rather than number of cigarettes smoked per day is the major mechanism of compensation for reduced nicotine delivery in cigarettes. (Auth. Abs. Mod.)

82–1230. Khan, M. T.; Furchgott, R. F. **Interactions of Phenoxybenzamine and Nicotinic Agonists at the Nicotinic Receptor in the Cat Adrenal Medulla.** *Journal of Pharmacology and Experimental Therapeutics* 221(1): 117-122, April 1982.

An investigation into whether nicotine (NIC) or acetylcholine (ACh) facilitate the blockage of the nicotinic receptor mechanism by phenoxybenzamine (PBZ) in a postsynaptic receptor—effector system is described. The system used was the perfused cat adrenal gland, in which stimulation of the nicotinic receptors of medullary cells by agonists results in release of catecholamines (CA). The inhibition of CA release by PBZ and lack of recovery of release indicate that a 30 min exposure to this agent may produce an essentially irreversible blockade of the nicotinic receptor—effector mechanism. Also, the concurrent presence of either NIC or ACh with PBZ facilitates the capacity of PBZ to produce an irreversible blockade of the nicotinic receptor—effector mechanism. However, the block produced by exposure to propranolol, unlike that by exposure to PBZ, is reversible. Crossover of facilitated block was demonstrated among NIC, ACh, and splanchnic nerve stimulation, which is consistent with the concept that NIC and ACh, and the endogenous neurotransmitter (ACh), are acting on the same set of nicotinic receptors. (Auth. Abs. Mod.)

82–1231. Koek, W.; Slangen, J. L. **The Role of Fentanyl Training Dose and of the Alternative Stimulus Condition in Drug Generalization.** *Psychopharmacology* 76(2): 149-156, February 1982.

Groups of rats were trained to discriminate differing doses of the narcotic fentanyl (F) (0.03, 0.02, or 0.01 mg/kg) from saline or to discriminate 0.03 mg/kg fentanyl (F) from alternative stimulus conditions (saline, 0.15 mg/kg nicotine, or 0.01 mg/kg F). Nicotine and amphetamine revealed noninteraction effects with F; all animals responded almost exclusively on the non-F lever. Morphine, however, showed a significant interaction effect with F. The interaction effect between morphine and nicotine and amphetamine was not significant. Training dose and alternative stimulus condition affected the generalization to morphine. Under none of the conditions did generalization occur to amphetamine or nicotine. The results are discussed in terms of the relative nature of drug generalization. (Auth. Abs. Mod.)

82–1232. Martin, B. R.; Aceto, M. D. **Nicotine Binding Sites and Their Localization in the Central Nervous System.** *Neuroscience and Biobehavioral Reviews* 5(4): 473-478, 1981.

The resolution of racemic nicotine to provide optically pure (+)-nicotine and the synthesis of radiolabeled nicotine with high specific activity have facilitated the study of nicotine binding in brain. The actions of the stereoisomers of nicotine on the central nervous system are qualitatively similar in most tests, but (-)-nicotine is more potent than the unnatural (+)-isomer by 10-fold or greater. Binding of radiolabeled nicotine to brain has both saturable and nonsaturable components. Only saturable binding is affected by incubation conditions such as time, temperature, pH, and ion concentration. Excess concentrations of the stereoisomers are equally effective in displacing (-)-(^{3}H)-nicotine from brain homogenates. Nevertheless, a direct comparison of (+)-(^{3}H)-nicotine and (-)-(^{3}H)-nicotine binding shows that the latter has a K_D three times lower than the former. (-)-(^{3}H)-Nicotine is bound to the greatest degree in hypothalamus and hippocampus, areas that also exhibited the most stereoselectivity for nicotine. However, differences in the binding affinities of the two isomers were far less than the pharmacological stereospecificity observed. (Auth. Abs.)

82–1233. May, D. C.; Jarboe, C. H.; VanBakel, A. B.; Williams, W. M. **Effects of Cimetidine on Caffeine Disposition in Smokers and Nonsmokers.** *Clinical Pharmacology and Therapeutics* 31(5): 656-661, May 1982.

Caffeine elimination is stimulated by cigarette smoking and inhibited by cimetidine. The absorption, distribution, and elimination of caffeine were evaluated in six smokers and six nonsmokers before and on the fourth day of administration of cimetidine. Caffeine absorption, assessed by the maximal serum caffeine concentration (C_{max}) and the time to reach C_{max}(t_{max}), was very rapid relative to elimination. The total body clearance (TBC) of caffeine was higher and the elimination half-life (t1/2) shorter in smokers than nonsmokers, but C_{max}, t_{max}, and the apparent volume of distribution (Vd,app) did not differ. Cimetidine decreased the TBC of caffeine by 31 percent in smokers and by 42 percent in nonsmokers. The increases in t1/2 were 45 percent and 96 percent. C_{max}, t_{max}, and Vd,app were unaffected by cimetidine. Caffeine induced similar slight increases in blood pressure and pulse rate in smokers and nonsmokers both before and during cimetidine dosing. (Auth. Abs. Mod.)

82–1234. McCarty, M. F. **Nutritional Support of Central Catecholaminergic Tone May Aid Smoking Withdrawal.** *Medical Hypotheses* 8(1): 95-102, January 1982.

Nicotine increases the release and turnover of catecholamines in the brain, and many features of the tobacco withdrawal syndrome—such as drug craving, poor concentration, impaired motor performance, drowsiness, fatigue, increased appetite with hyperphagia—may reflect diminished central catecholaminergic tone. Support of central catecholamine synthesis with the nutrients tyrosine and glucose tolerance factor (which enhance brain tyrosine levels) may lessen tobacco withdrawal symptoms and may increase the chance of success in smoking cessation programs. Oral administration of 100 mg/kg of tyrosine in 3 doses increased the 24-hour urinary excretion of catecholamines by about 25 percent in healthy young males, but no effect on blood pressure or heart rate was observed. In rats, however, tyrosine doses produced a reduction in blood pressure. Nutritional measures of this type would probably be safer and more appropriate for long-term use than central-stimulant drugs. (Auth. Abs. Mod.)

82–1235. New Scientist. **Tobacco Company Stops Supplying Pesticide.** *New Scientist* 94(1310): 763, June 17, 1982.

British American Tobacco (BAT) announced it will stop supplying farmers in Kenya with the controversial insecticide, aldrin, for spraying tobacco crops. BAT acknowledged there was considerable evidence regarding aldrin's dangers. Orthene, with acephate as its active ingredient, will be substituted. However, the wearing of protective clothing and frequent washing with soap and water are recommended when using orthene, and since such clothing and soap are in short supply in Kenya, farmers will still be running risks producing tobacco. In responding to questions about the actual tar delivery of *Barclay* cigarettes, BAT spokesmen expressed total confidence in the integrity of the product. However, they refused to outline BAT's policy about encouraging the expansion of cigarette smoking in the Third World.

82–1236. Obe, G.; Vogt, H.-J.; Madle, S.; Fahning, A.; Heller, W. D. **Double-Blind Study on the Effect of Cigarette Smoking on the Chromosomes of Human Peripheral Blood Lymphocytes in Vivo.** *Mutation Research* 92: 309-319, 1982.

A double-blind study with 170 smokers and 124 nonsmokers revealed a doubling of the frequencies of exchange-type aberrations in the peripheral blood lymphocytes of smokers as compared with nonsmokers. Smokers (n=24) had 1 sister-chromatid exchange (SCE) more per metaphase than nonsmokers (n=20), an effect that is significant but low when compared with the effect on structural chromosomal aberrations. These results show that structural chromosomal aberrations are better indicators of the genetical effects of low chronic exposures to mutagens in man than SCEs. The frequencies of micronuclei in 3-day lymphocyte cultures showed no differences in smokers (n=95) and nonsmokers (n=39). Nonsmokers (n=99) had significantly higher frequencies of 2nd metaphases in 48-hour cultures in vitro than smokers (n=141), indicating an effect of smoking on lymphocytes in vitro. (Auth. Abs.)

82-1237. Pantuck, E. J.; Pantuck, C. B.; Anderson, K. E.; Conney, A. H.; Kappas, A. **Cigarette Smoking and Chlorpromazine Disposition and Actions.** *Clinical Pharmacology and Therapeutics* 31(4): 533-538, April 1982.

Cigarette smoking was found to decrease drowsiness and orthostatic hypotension due to chlorpromazine, but not by influencing its metabolism. Chlorpromazine, 75 mg, was taken by eight cigarette smokers and nine nonsmokers. Mean maximum percent decrease in mean blood pressure with change in position from lying to standing during the first 10 hours after chlorpromazine was 46 percent greater in nonsmokers than in smokers. Of the eight smokers, six did not become sleepy after chlorpromazine, one became slightly sleepy, and one fell asleep. Of the nine nonsmokers, three became slightly sleepy, one moderately sleepy, and five fell asleep. Mean peak plasma concentration of chlorpromazine was 24 percent lower and mean area under the plasma concentration-time curve was 36 percent lower in smokers than in nonsmokers. There was no correlation between these plasma level parameters and either the degree of sleepiness or the degree of orthostatic hypotension in the subjects. The relatively small differences between cigarette smokers and nonsmokers make it unlikely that cigarette smoking influenced these effects of chlorpromazine by enhancing its overall metabolism. (Auth. Abs. Mod.)

82-1238. Park, B. K.; Breckenridge, A. M. **Clinical Implications of Enzyme Induction and Enzyme Inhibition.** *Clinical Pharmacokinetics* 6(1): 1-24, 1981.

The pharmacological effect of a drug is partly dependent upon its concentration at its site of action, which in turn is partly dependent upon its rate of elimination. The rate of elimination of many lipophilic drugs is governed by the activity of the hepatic microsomal mixed-function oxidases. Consequently, any alteration in the activity of these enzymes may result in a modification of drug action. A wide range of chemically unrelated substances, such as barbiturates, rifampicin, and phenytoin, may stimulate the activity of the mixed-function oxidases by enzyme induction. Enhancement of drug metabolism by ethanol, tobacco smoking, and diet may also involve enzyme induction. Although the effect of smoking is apparently a selective process, at present it is not possible to predict which drugs will be affected. For instance, the metabolic rates of nicotine, antipyrine, theophylline, imipramine, and pentazocine are increased while the metabolism of diazepam, pethidine, phenytoin, nortriptyline, warfarin, and ethanol remain unchanged. Nevertheless, it is now thought that the smoking habit is one of the primary sources of drug interactions in man. Of the 3,000 chemicals so far identified in cigarette smoke, the polynuclear aromatic hydrocarbons are probably the agents responsible for hepatic enzyme induction. (Auth. Abs. Mod.)

82-1239. Pryor, W. A.; Church, D. F.; Govindan, C. K.; Crank, G. **Oxidation of Thiols by Nitric Oxide and Nitrogen Dioxide: Synthetic Utility and Toxicological Implications.** *Journal of Organic Chemistry* 47(1): 156-158, 1982.

It is suggested that the enzyme-inhibitory effects of cigarette smoke are due to nitric oxide (NO) and nitrogen dioxide (NO_2). Thiols are readily oxidized to disulfides by either NO or NO_2; thus these species are potent inhibitors of thiol-dependent enzymes. This may also explain how cysteine and glutathione protect macrophages against cigarette smoke—the external thiols are sacrificially oxidized, taking up the NO or NO_2 in the cigarette smoke. Other reactions involving NO and NO_2 and their toxicological significance are discussed.

82-1240. Richardson, D.; Martin, W. **Effects of Chronic Tobacco Smoke and Nicotine Administration on Capillary Supply to Rat Skeletal Muscle.** *Microcirculation* 1(1): 17-36, 1981.

Indications are that long-term administration of nicotine alone elicits capillary proliferation in oxidative types of muscle fibers. However, when nicotine is consumed via tobacco smoke this effect is blocked by other substances in the smoke. A study compared the effects of long-term tobacco smoke and nicotine administration on the capillary supply to skeletal muscle. Samples of gracilis muscle were obtained from male Sprague-Dawley rats that had been either administered tobacco smoke daily for a period of 4 to 6 months, given nicotine in their drinking water for the same period of time, or left untreated. The amounts of nicotine consumed by the two modes of treatment were equivalent. The muscle samples were processed by an ATPase method for visualization of the capillaries and differentiation of the slow oxidative (SO), fast oxidative glycolytic (FOG), and fast glycolytic (FG) fibers. The number of capillaries around single fibers (CAF) were counted, and the distance around each fiber's perimeter (FP) was measured. The nicotine-treated group had a significantly higher CAF in the SO and FOG fibers compared to the untreated animals, but there were no differences in FP. The FG fibers in the nicotine group were the same as in the untreated group. By contrast, the FG fibers in the tobacco smoke-treated animals showed a significantly smaller CAF and FP compared to the untreated animals, while the SO and FOG fibers were the same in the smoke-treated and untreated rats. (Auth. Abs.)

82-1241. Riebe, M.; Westphal, K.; Fortnagel, P. **Mutagenicity Testing, in Bacterial Test Systems, of Some Constituents of Tobacco.** *Mutation Research* 101(1): 39-43, March 1982.

Nicotine, minor alkaloids, several degradation products, and some of their heterocyclic components that occur in tobacco and in minor amounts in cigarette smoke condensates were tested for their mutagenic capacity. Two bacterial tests were used: an *Escherichia coli* strains assay and the *Salmonelli typhimurium*/microsome assay (Ames test). Of 12 constituents tested, none showed mutagenic activity in the Ames test. The substances pyrrolidine, nicotine, anatabine, myosmine, 2,3'-dipyridyle and nicotyrine induced repairable DNA damage in the *E. coli* test. The reason for this discrepancy may be the lack of appropriate genetic endpoints in the mutagenicity tests or, alternatively, an effect in the *E. coli* test system which does not lead to mutagenic alterations. Further experiments are required to elucidate this phenomenon. (Auth. Abs. Mod.)

82–1242. Roth, K. A.; McIntire, S. L.; Barchas, J. D. **Nicotinic-Catecholaminergic Interactions in Rat Brain: Evidence for Cholinergic Nicotinic and Muscarinic Interactions With Hypothalamic Epinephrine.** *Journal of Pharmacology and Experimental Therapeutics* 221(2): 416-420, May 1982.

Intraperitoneal injections of nicotine produced several changes in regional catecholamine concentrations in rat brain. These changes were blocked by the centrally active nicotinic antagonist mecamylamine, but not by the quaternary nicotinic antagonist hexamethonium. An examination of the effects of various cholinergic agents on hypothalamic epinephrine concentrations revealed several findings. Central muscarinic antagonism or peripheral muscarinic agonism decreased hypothalamic epinephrine concentrations. The anticholinesterase physostigmine decreased hypothalamic epinephrine concentrations; and this effect was blocked by the centrally acting nicotinic antagonist mecamylamine, but not by hexamethonium, scopolamine, or methscopolamine. The findings indicate an interaction of cholinergic receptors, both nicotinic and muscarinic, with hypothalamic epinephrine. (Auth. Abs.)

82–1243. Sorsa, M.; Norppa, H.; Leppanen, A.; Rimpela, M. **Induction of Sister-Chromatid Exchange in Human Lymphocytes by Smoke Condensates From Different Brands of Cigarette.** *Mutation Research* 103(2): 149-153, February 1982.

Smoke condensates acquired using six commercially available cigarette brands were each found to induce sister-chromatid exchange (SCE) in human lymphocyte samples. The condensates were made by using the Filtrona 302 smoking machine. Some of the brands used had filters while others did not. Some brands were high in tar, while other types were "light." For all condensates, a dose-related effect was found for SCE induction. The data appeared to suggest that the quantity of tar in the cigarettes and also the number of cigarettes used to make a condensate might influence the mutagenic activity in the condensate.

82–1244. Takano, T.; Maeda, H. **Exposure of Firefighters to Carbon Monoxide.** *Journal of Combustion Toxicology* 8: 89-95, May 1981.

Exposure of firefighters to carbon monoxide during firefighting can be a significant health hazard, especially in those who smoke. An investigation of Japanese firefighters 30 to 60 minutes after firefighting found that the calculated mean blood carboxyhemoglobin (COHb) levels averaged 3.4 percent for nonsmokers and 5.8 percent for smokers. Levels of over 8 percent were found in 4 percent of nonsmokers and 14 percent of smokers, a level which can increase oxygen debt during exercise, impair cerebral functions, affect the visual threshold, and impose a burden on the cardiovascular system. Since real COHb levels during actual firefighting could be expected to be even higher, and energy consumption would also be at a high level, it is concluded that such exposure is harmful, especially for those who smoke.

82–1245. Tripathi, H. L.; Martin, B. R.; Aceto, M. D. **Nicotine-Induced Antinociception in Rats and Mice: Correlation With Nicotine Brain Levels.** *Journal of Pharmacology and Experimental Therapeutics* 221(1): 91-96, April 1982.

Nicotine was found to be potent in producing antinociception in mice and rats as measured by tail-flick latency, but its action was of short duration. Nicotine's ED_{50} values (confidence limits) were 0.7 (0.4-1.1) and 2.0 (1.2-3.4) mg/kg in rats and mice, respectively, at the time of maximum effect. Brain levels of nicotine reached a maximum at 10 min, whereas antinociception was maximal within 2 min in rats. However, there was a good correlation between the time courses of antinociception and brain levels of nicotine in mice with both attaining maximal levels at 5 min. It was found that tachyphylaxis developed to the antinociception in rats within 10 min and lasted for up to 14 hours, but tachyphylaxis did not develop to nicotine-induced antinociception in mice. The effect of nicotine appeared to be central in both rats and mice inasmuch as mecamylamine antagonized completely but hexamethonium (5 mg/kg) antagonized partially. Nicotine-induced antinociception was not blocked in either rats or mice by atropine in doses up to 10 mg/kg. Naloxone did not block nicotine in rats but did antagonize the antinociception in mice. In addition, yohimbine, a selective α-2 antagonist, blocked the antinociception in both species. These data implicate several different mechanisms in the antinociceptive action of nicotine. (Auth. Abs.)

82–1246. Vandeputte-Van Messom, G. **Effect of Intracerebroventricular Injection of Nicotine and Acetylcholine on Renal Water and Salt Excretion in Conscious Goats.** *Archives Internationales de Pharmacodynamie et de Therapie* 253(1): 52-67, September 1981.

In hydrated, conscious goats nicotine was administered into the 3rd ventricle and its effects on water and electrolyte excretion were studied. Acetylcholine (ACh) in combination with eserine (Es) was injected by the same route in goats pretreated with atropine. Nicotine as well as large doses of ACh + Es induced an antidiuresis of the pituitary type. The responses to ACh were dose—dependent. Intracerebroventricular (icv) pretreatment with hexamethonium blocked the responses to both nicotine and ACh, whereas atropine and propranolol were ineffective. It appears likely that nicotine receptors are involved in the ACh-induced release of antidiuretic hormone. Phentolamine (icv) completely blocked the effects of ACh + Es and partly those of nicotine, indicating that catecholamines might be involved to some extent. (Auth. Abs.)

82-1247. Vesey, C. J.; Saloojee, Y.; Cole, P. V.; Russell, M.A.H. **Blood Carboxyhaemoglobin, Plasma Thiocyanate, and Cigarette Consumption: Implications for Epidemiological Studies in Smokers.** *British Medical Journal* 284(6328): 1516-1518, May 22, 1982.

Carboxyhemoglobin and plasma thiocyanate concentrations were found to be significantly correlated with self-reported daily cigarette consumption in 360 smokers ($r = 0.416$ and 0.412, respectively; $p < 0.001$). The extent to which inhalation patterns affected the intake of cigarette smoke constituents was determined from the partial correlation between carboxyhemoglobin and plasma thiocyanate concentrations after the number of cigarettes smoked/day had been allowed for ($r = 0.48$). Thus, 23 percent of the variation in carboxyhemoglobin and thiocyanate concentrations was accounted for by the way a cigarette was smoked, and 21 percent by the number smoked/day. Furthermore, the relationship between carboxyhemoglobin or plasma thiocyanate and daily cigarette consumption was not linear but reached an asymptote at consumption rates above 25 cigarettes/day. These results suggest that daily self-reported cigarette consumption alone will not identify those smokers most at risk and will also underestimate the dose—response relationship between smoking and selected diseases. Measurement of either carboxyhemoglobin or plasma thiocyanate concentrations is the superior method to determine the index of exposure to tobacco smoke. (Auth. Abs. Mod.)

82-1248. Vijayalaxmi; Evans, H. J. **In Vivo and In Vitro Effects of Cigarette Smoke on Chromosomal Damage and Sister-Chromatid Exchange in Human Peripheral Blood Lymphocytes.** *Mutation Research* 92: 321-332, 1982.

The frequencies of chromosomal aberrations in blood lymphocytes of a group of moderate cigarette smokers are significantly greater than in a matched group of nonsmoking controls; the incidence of unambiguous exchange aberrations in the smokers was some four times that of controls. The incidence of sister-chromatid exchanges (SCE) in blood lymphocytes of heavy cigarette smokers is approximately 50 percent higher than in nonsmokers. Lymphocytes from both smokers and nonsmokers exposed to cigarette smoke condensate (CSC) in vitro showed dose-dependent increases in SCE frequency, with the yields from smokers being greater than those from nonsmokers. Some evidence was obtained for an increased level of unscheduled DNA synthesis in cells of smokers relative to nonsmokers exposed to CSC in vitro. When lymphocytes from smokers and nonsmokers are exposed to mitomycin C, or to ethyl methane sulphonate in vitro, the yields of SCE at all dose levels are significantly greater in cells from smokers. This difference between smokers and nonsmokers, however, simply reflects the initial basal differences in SCE frequencies between the groups: smokers' cells are not intrinsically more sensitive to exposure to mutagens. (Auth. Abs. Mod.)

82-1249. Wilson, S. P.; Slepetis, R.; Chang, K.-J.; Kirshner, N.; Viveros, O. H. **Differential Secretion of Opioid Peptides and Catecholamines From Cultured Cells of a Human Pheochromocytoma Tumor.** *Life Sciences* 29(22): 2257-2264, 1981.

Dissociated cells from a human pheochromocytoma tumor were maintained in culture, and the secretion of opioid peptides (OP), endogenous catecholamines (CA), and preloaded (^{3}H)norepinephrine from these cells was examined. Nicotine, veratridine, barium, or Ionomycin stimulated the secretion of OP, endogenous CA, and ^{3}H from the pheochromocytoma cells. In general, the different secretagogues were more potent in releasing OP than endogenous CA; ^{3}H secretion was intermediate. Secretion of OP was more sensitive to stimulation by the calcium ionophore Ionomycin and by veratridine than was CA secretion. Nicotine-evoked OP secretion was more sensitive to extracellular calcium concentration than was secretion of CA or ^{3}H. In contrast, bovine adrenal chromaffin cells show no such differential secretion of OP and CA in response to Ionomycin stimulation or to nicotine stimulation under conditions of varying extracellular calcium concentration. The results show that human pheochromocytomas secrete OP as well as CA and that there may be heterogeneous storage pools of CA and OP in cultured pheochromocytoma cells. (Auth. Abs.)

82-1250. Zahran, F.; Yousef, A. A.; Baig, M.H.A. **A Study of Carboxyhaemoglobin Levels in Cigarette and Sheesha Smokers in Saudi Arabia.** *American Journal of Public Health* 72(7): 722-724, July 1982.

A single carboxyhemoglobin (COHb) estimation of a late evening blood sample among nonsmokers, cigarette smokers, and sheesha smokers was evaluated among Saudis. Sheesha smokers consumed 15 to 20 grams of tobacco as Jurak in combination with other materials. Sheesha smoking for 15 minutes would provide the tobacco equivalent of one cigarette. The COHb level in smokers of 15 to 40 cigarettes a day ranged between 0.7 and 10.3 with a mean value of 6.1 ± 2.58 COHb. Values among sheesha smokers ranged between 6.5 and 13.9 with a mean value of 8.8 ± 1.83, significantly higher than those of cigarette smokers ($p < 0.001$) for a given degree of exposure to tobacco smoke. (Auth. Abs. Mod.)

MORTALITY AND MORBIDITY

82–1251. Armstrong, B.; de Klerk, N. **A Comparison of Premature Mortality Due to Cigarette Smoking and Road Crashes in Australia.** *Community Health Studies* 5(3): 243-249, 1981.

Indications are that smoking may have caused more premature mortality in Australia in 1978 than did road crashes. By use of data on smoking-induced death in Americans it was estimated that current levels of cigarette smoking in Australia caused some 10,434 premature deaths in 1978. These deaths represented a total loss of about 156,730 person-years of life, of which about 70,005 were lost before the age of 70 years. By way of comparison, in the same year, road crashes caused over 3,000 premature deaths with loss of about 152,211 person-years of life in total, or about 123,062 before the age of 70 years. The validity of this comparison of the importance of smoking and road crashes as causes of premature death is limited mainly by the uncertainty of the estimates of mortality due to smoking. Nevertheless, these results illustrate one method of setting priorities for action in preventive medicine. (Auth. Abs. Mod.)

82–1252. Ashley, M. J. **1. Morbidity and Mortality. Alcohol, Tobacco, and Drugs: An Audit of Mortality and Morbidity.** In: *Man, Drugs and Society—Current Perspectives.* Proceedings of the First Pan-Pacific Conference on Drugs and Alcohol, Canberra, Australia, February 26-March 5, 1980. Canberra, Australian Foundation on Alcoholism and Drug Dependence, 1981, pp. 350-365.

An audit of mortality and morbidity associated with alcohol, tobacco, and drugs is presented to provide a basis for public health policy formulation and program planning and development in the 1980s and to indicate deficiencies in present knowledge to which future research efforts may be directed. The overall mortality risk associated with alcoholism and heavy drinking is two to four times that expected, while that among current smokers is generally about 1.7 times. Although the excess mortality among opiate addicts is well-documented, the risks in chronic users of other psychoactive drugs are less well-defined. Characteristic disease profiles are associated with alcohol, tobacco, and drug use, and the impact on society and the individual is illustrated by sickness, absenteeism, and disability. Interactions resulting from multidrug use are increasingly important considerations, and the adverse effects of these substances on human reproduction must not be overlooked. The public health significance of drug associated mortality and morbidity is evident from its contribution to premature mortality, its burden on the health care system, and its costs. In Canada, for example, current smoking and/or hazardous drinking presently account for 18 percent of both premature deaths and potential years of lost life. (Auth. Abs. Mod.)

82–1253. Cullen, K.; Stenhouse, N. S.; Wearne, K. L. **Alcohol and Mortality in the Busselton Study.** *International Journal of Epidemiology* 11(1): 67-70, March 1982.

Nondrinking smokers of Busselton, Australia, had significantly higher mortality from deaths due to all causes than nondrinking nonsmokers. Also, a significantly higher cancer, cardiovascular, and coronary heart disease mortality was observed in smokers who drank, suggesting a synergistic action between smoking and drinking in relation to these diseases. Among nonsmokers, mortality from all causes was higher in the nondrinkers than in those who reported themselves as drinkers. However, the Busselton intake of alcohol was moderate (21.2 mls/day), and the possibility remains of increasing mortality when daily intake of alcohol exceeds 30 mls, especially in smokers. Mortality data were recorded over a 13-year period, in a group of 2,209 adults over 40 years of age.

82–1254. Ganguin, H.-G.; Wilde, J. **Rauchen oder Gesundheit. [Smoking or Health.]** *Zeitschrift fur die gesamte Hygiene und ihre Grenzgebiete* 26(4): 233-242, April 1980, German.

Statistical data is used to emphasize the significance of preventing cardiovascular, neoplastic, respiratory, and gastrointestinal diseases. A practicable way of achieving effective primary prevention of these diseases is seen in eliminating the dangers posed by tobacco. Most recent information published in international and national literature on the action of tobacco components on morbidity, life expectancy, invalidity, performance, and on the human organism is quoted in detail. The growing consumption of cigarettes in the German Democratic Republic, which is about 40 percent higher at present among people above 15 years of age compared with 1960 data, underlines the urgent need for a decisive and responsible stance vis-a-vis this problem on the part of the medical profession as a whole and of the individual physician. (Auth. Abs. Mod.)

82–1255. Kuller, L.; Meilahn, E.; Townsend, M.; Weinberg, G. **Control of Cigarette Smoking From a Medical Perspective.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 3. Palo Alto, California, Annual Reviews Inc., 1982, pp. 153-178.

A review of the impact of smoking cessation on lung cancer, chronic obstructive pulmonary disease, and arteriosclerosis risk is presented. The problems involved in assessing this impact are examined according to the variables traditionally measured: frequency of smoking, duration of exposure, amount of the cigarette consumed, type of cigarette smoked, form of tobacco used, biochemical measures of risk, and the response rates of the study populations involved. Also considered are the possible confounding variables of individual physiology, and psychosocial, environmental, and demographic factors. The current estimates of risk reduction from smoking cessation are discussed for cardiovascular disease, cancer, and the spirometric indices of lung function according to recent studies such as the Kaiser-Permanente studies, the Multiple Risk Factor Intervention Trial (MRFIT), the World Health Organization (WHO) studies, the Oslo study, studies of the British Medical Society, and others.

82–1256. Mellstrom, D.; Rundgren, A.; Jagenburg, R.; Steen, B.; Svanborg, A. **Tobacco Smoking, Ageing and Health Among the Elderly: A Longitudinal Population Study of 70-Year-Old Men and an Age Cohort Comparison.** *Age and Ageing* 11(1): 45-58, February 1982.

The prevalence and the adverse effects of smoking were studied among a sample of elderly men who were divided into smokers, ex-smokers, and nonsmokers. In the population study of 70-year-old people in Goteborg, 449 70-year-old men were examined in 1971-1972. Out of these, 331 were reexamined in 1976-1977 at age 75, together with a cohort-comparison group of 474 70-year-old men. Cohort differences and a lower smoking prevalence at higher ages were observed. The loss of body weight after the age of 70 was higher in smokers and was not due to differences in energy intake. The levels of certain blood components showed differences among smokers compared to nonsmokers, indicating an influence on liver metabolism and on the hormone function. Tobacco smoking was not only related to ill health but also to an impaired functional capability in bone mineral density, pulmonary function, and muscle strength. Results indicate that the hazards of tobacco smoking are increased by aging. (Auth. Abs. Mod.)

82–1257. Stille, W. T.; Tabershaw, I. R. **The Mortality Experience of Upstate New York Talc Workers.** *Journal of Occupational Medicine* 24(6): 480-484, June 1982.

Deaths for a 31-year period (1948 to 1978) were analyzed in a historical prospective cohort study of 655 white male talc (TMX) workers. Death rates from all causes, from cancer of the respiratory system, and from nonmalignant respiratory disease were not significantly different from those of the U.S. white male population. However, significant differences for these causes of death were found among workers who had previous occupational histories. An analysis of the latency periods of the observed lung cancer suggests that exposure to an etiologic agent during previous work experience or possible confounding with nonoccupational exposures may play a role in the development of lung cancer. One such nonoccupational exposure is smoking, and it is noted that the standard mortality ratio of 163 for respiratory cancer in this population is consistent with a smoking effect. While the actual smoking experience of this population is not known, insight into it is gained from knowing the smoking patterns of current TMX workers, many of whom are included in the study cohort. Among 151 workers given a medical examination at TMX in 1979, 69 (46 percent) were current smokers, 38 (25 percent) were past smokers, and 44 (29 percent) were nonsmokers. The percentage of current smokers is higher than in the general population, but is consistent with patterns seen in the mining industry. (Auth. Abs. Mod.)

82–1258. Tomori, Z. **Fajcenie a jeho Nepriaznive Ucinky a Moznosti ich Prevencie. [Harmful Effects of Smoking and Possibilities of Their Prevention.]** *Bratislavske Lekarske Listy* 76(3): 374-382, September 1981, Slovak.

The pathogenesis of the most important health consequences of smoking, the principal localized effects of active components of cigarette smoke, the early diagnosis of smoking consequences, and the prevention and treatment of smoking addiction are reviewed. It is concluded that the smoking habit has many harmful effects on the respiratory and cardiovascular systems and on other organs. Early detection of respiratory and other functional disorders in smokers is possible in a reversible stage of a disease. Individual researchers, medical diagnostic laboratories, specialists in related fields, and medical and health education communities must cooperate in smoking control. The importance of personal example and sustained effort is stressed for systematic prevention, early detection, and effective treatment of the harmful consequences of the smoking habit to human health.

See also, **82–1442**

NEOPLASTIC DISEASES

82–1259. Blot, W. J.; Fraumeni, J. F., Jr. **Changing Patterns of Lung Cancer in the United States.** *American Journal of Epidemiology* 115(5): 664-673, May 1982.

Reasons for the changing patterns of lung cancer in the United States are not totally clear, but there is little doubt that cigarette smoking has been the dominant cause of the male excess in lung cancer and its dramatic upward trend in incidence and mortality. It is likely that variations in smoking practices also contribute to a shift from the white to nonwhite predominance of lung cancer among males, an emerging excess among southern whites, and recently increasing rates among females of both races. In the mid-1950s, differences in smoking habits between the races were reported to be small, except for a lower percentage of heavy smokers among blacks; this was still the case by the late 1970s. Although the recent smoking prevalence data continue to show only minor geographic differences, the highest percentage of current white male smokers in the 45 to 64 years age range was in the south. This would help explain the higher rates for lung cancer in the south during the 1970s, but the magnitude of the excess mortality is larger than would be predicted simply by the higher percentage of current smokers. These assessments are based on limited smoking data, and it is not possible to precisely chronicle the age-, sex-, race-, and region-specific patterns in smoking habits over time. However, the substantial geographic and racial shifts in lung cancer suggest that factors other than smoking may play some role.

82–1260. Colburn, N. H.; Koehler, B. A.; Nelson, K. J. **A Cell Culture Assay for Tumor-Promoter-Dependent Progression Toward Neoplastic Phenotype: Detection of Tumor Promoters and Promotion Inhibitors.** *Teratogenesis, Carcinogenesis, and Mutagenesis* 1(1): 87-96, 1980.

Mouse epidermal cell lines have been identified which respond to tumor-promoting (but not nonpromoting) phorbol esters with an irreversible shift in anchorage independence, an in vitro marker of neoplastic phenotype. This response may be analogous to a later stage of tumor promotion in vivo. The shift occurs at 12-O-tetradecanoyl-phorbol-13-acetate (TPA) concentrations as low as 0.1 ng/ml (1.6×10^{-10} M). The specificity of the soft agar growth response is not limited to phorbol esters but extends to nonphorbol plant diterpenes such as mezerein, to detergents, to polycyclic hydrocarbons present in cigarette smoke, and to some growth factors. Maximal activity of the cigarette smoke was 8 percent of the TPA value. Active concentrations ranged between 10 and 40 μg/ml, but the concentration of active ingredients may be much lower. Clones which were highly responsive to phorbol esters were also highly responsive to other classes of promoters, indicating their usefulness both for promoter detection and mechanism studies. The anchorage-independence response to TPA was inhibited by a series of retinoids whose activity paralleled that for inhibiting tumor promotion in vivo. (Auth. Abs. Mod.)

82–1261. Cook-Mozaffari, P. **The Epidemiology of Cancer of the Oesophagus.** *Nutrition and Cancer* 1(2): 51-60, 1979.

In contrast to a dramatic variation in the incidence of esophageal carcinoma worldwide, the pattern of causation remains muted without the clear domination of any individual risk factors. Investigations in high incidence areas are inconclusive. In northeastern Iran, where alcohol consumption is nearly absent and there is very little smoking ("nass," a mixture of tobacco, lime, and woodash, is chewed by men only), dietary deficiencies appear to play an important role. In moderate incidence populations like Singapore, Japan, and Puerto Rico, alcohol consumption, cigarette smoking, and a preference for burning hot drinks were positive risk factors; however, where there were dietary deficiencies, incidence was higher. In Africa a 12-fold male preponderance of the disease indicated a social habit such as drinking or smoking, and in France excess risk was ascribed to the multiplicative effect of drinking and smoking. It is concluded that, at present, the items in the multifactorial equation appear to be: (1) a conditioning of individual susceptibility either from a very poor diet from infancy or the deleterious effect of alcohol on nutritional status, (2) a further heightening of susceptibility from long-term irritation of the esophageal mucosa, (3) the action of some carcinogenic agent, and (4) the possibility that alcohol could act as a solvent facilitating the absorption of harmful substances to the basal layers of the esophagus.

82–1262. Craighead, J. E.; Mossman, B. T. **The Pathogenesis of Asbestos-Associated Diseases.** *New England Journal of Medicine* 306(24): 1446-1454, June 17, 1982.

Surveys of asbestos-exposed workers consistently show that bronchogenic carcinoma is uncommon in those who do not smoke: cancer risk in these workers is substantially lower than the risk of a nonexposed smoker of 2 to 3 packs/day. The combined effects of asbestos and smoking appear to be multiplicative rather than additive. Asbestos has many of the properties of classic tumor promotors, as does cigarette smoke, which may additively or synergistically enhance the susceptibility of the respiratory mucosa to carcinogens. Another theory of bronchogenic carcinoma origin states that asbestos fibers phagocytized by the bronchial epithelium act as physical carriers of the carcinogens in cigarette smoke to the basal cell, where neoplasms may originate. Oropharyngeal and esophageal tumors also occur more frequently in smoking asbestos workers, though a relationship between smoking and asbestos-associated carcinoma of the large intestine and kidney has not been established. In this review of asbestos-associated diseases, asbestosis and pleural lesions are discussed, as well as the mineralology and uses of asbestos, the pathogenic potential of asbestos types, and regulatory considerations.

82–1263. Davis, D. L.; Bridbord, K.; Schneiderman, M. **Estimating Cancer Causes: Problems in Methodology, Production, and Trends.** In: Peto, R.; Schneiderman, M. (Editors). *Quantification of Occupational Cancer,* Banbury Report 9. Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, pp. 285-316.

The problems, methods, and current estimates for the causes of cancer are reviewed. It is difficult to estimate what proportion of cancers are due to smoking, lifestyle factors, or industrial production. The cancers attributed to smoking are lung, bladder, pancreas, esophagus, larynx, kidney, and buccal cavity and pharynx. For white men, it is estimated that 18 percent of the increased cancer incidence is due to smoking; in women the estimate is 45 percent. For blacks, the rates are 72 percent in men and 76 percent in women. Overall, as high as one in five cancers are associated with cigarettes. However, many of these cancers are occupation-confounded. Besides the hazards of multiple exposures, smokers, men, and blacks are more likely to work at a hazardous job than nonsmokers, women, and whites. This may explain why black men, of whom fewer smoke, who smoke less, and start at a later age, have a higher cancer rate than whites. Common industrial carcinogens, chemical production patterns, and problems in trend analysis are also reviewed, and two appendices are included.

82–1264. Davison, G.; Duffy, M. **Smoking Habits of Long-Term Survivors of Surgery for Lung Cancer.** *Thorax* 37(5): 331-333, May 1982.

The smoking habits of 52 patients who had survived more than 5 years after treatment for lung cancer were studied. Fifty (96 percent) of the patients were smokers prior to operation; 45 smoked only cigarettes, 4 only a pipe, and 1 both cigarettes and a pipe. Fifty-six percent (28) had stopped smoking preoperatively but 19 (68 percent of those who stopped smoking) only stopped in the 4 weeks leading up to the operation. No patients smoked in the immediate postoperative period but about half became regular smokers again, usually within a year of the operation. Those who had stopped before, rather than after, their operation were more likely to remain nonsmokers. Doctors were usually unsuccessful in persuading those patients who had restarted after surgery to stop smoking. Patients undergoing surgery for lung cancer should be advised to stop smoking before the operation. Those in whom resection is successful should receive regular support and encouragement from doctors, especially in the first postoperative year, to continue as nonsmokers. (Auth. Abs. Mod.)

82–1265. Decker, J.; Goldstein, J. C. **Current Concepts in Otolaryngology. Risk Factors in Head and Neck Cancer.** *New England Journal of Medicine* 306(19): 1151-1155, May 13, 1982.

In a review of chronic irritants associated with head and neck cancer, tobacco use is listed as a major risk factor in lip, oral cavity, pharyngeal, and laryngeal neoplasms. Lip cancer is largely associated with prolonged exposure to sunlight and all forms of tobacco use, particularly pipe smoking. Patients with neoplasms of the oral cavity, larynx, and pharynx commonly have a history of tobacco use combined with alcohol consumption and poor oral hygiene. The promoting effect of alcohol is notable in oral cancer, as tobacco-derived carcinogens are relatively dilute in the mouth. Cultural differences in tobacco use are apparent in the geographic and anatomic variations of oral neoplasms. Only 3 percent of cancers occur in the oral cavity in the United States, where cigarette use is most prominent; in Bombay, oral neoplasms represent 50 percent of cancer incidence. Laryngeal cancer displays a distinct subsite susceptibility in oral and pharyngeal cancer, consistent with dose—response effects of tobacco exposure promoted by alcohol consumption.

82–1266. Dunkley, G. C. **Aluminum Reduction and Bladder Cancer. (Letter).** *Canadian Medical Association Journal* 126(9): 1026, May 1, 1982.

A critique of a study (*Canadian Medical Association Journal* 124(4): 419-422, 427, February 15, 1981) on bladder cancer among aluminum workers states that the formula used for calculating risk was only applicable to a single dichotomous exposure. This did not allow the risk from smoking to be separated from that due to the aluminum reduction process. Thus the risk of bladder cancer from aluminum exposure, while still a significant excess, is overestimated. A more accurate calculation using Walter's formula reveals an excess of bladder cancer deaths which are due to factors such as smoking and other unidentified exposures. See also 81-1792.

82–1267. Feldman, J. G.; Kissin, B. **Form and Duration of Drinking and Risk to Head and Neck Cancer.** In: van Eys, J.; Seelig, M. S.; Nichols, B. L., Jr. (Editors). *Nutrition and Cancer.* Proceedings of the 18th Annual Meeting of the American College of Nutrition, Houston, June 9-10, 1977, New York, SP Medical and Scientific Books, 1979, pp. 263-275.

In a study of head and neck cancer patients ($n=216$), it was found that male patients were significantly more likely to be heavy (more than one pack/day) smokers ($p<0.001$) than were males with other cancers ($n=529$). Similar differences were found for females, but the results were not statistically significant. The drinking, smoking, and dietary histories of all patients were obtained by retrospective interviews for the 5-year period prior to diagnosis. Other cancer patients were subdivided into smoking related sites (lung and bladder) and nonsmoking sites. When compared with patients with nonsmoking sites, the relative risk of head and neck cancer was from 2.5 to 3 times greater for the heavy drinker or heavy smoker. The risk was almost 10 times greater for the individual who was both a heavy drinker and heavy smoker. When compared with smoking related sites, the relative risk was 2 to 2.5 times greater in the heavy drinker regardless of the level of smoking. Yet, the relative risk of developing lung or bladder cancer was 4 times higher among heavy smokers irrespective of the amount of alcohol consumed when compared with nonsmoking sites. The survey data reaffirm the association of alcohol and tobacco use with head and neck cancer.

82–1268. Flanders, W. D.; Rothman, K. J. **Occupational Risk for Laryngeal Cancer.** *American Journal of Public Health* 72(4): 369-372, April 1982.

Various occupations were analyzed for laryngeal cancer risk in a case—control study using the interview data from the Third National Cancer Survey. Effects were measured relative to a group of industries and occupations arbitrarily defined as low risk. Females were excluded and potential confounding controlled by stratification on age, tobacco use, alcohol use, and race. The strong association usually found between laryngeal cancer and smoking is evident in these data; adjusted for alcohol use, race, and age, the rate ratio estimate comparing heavy cigarette smokers with light or nonsmokers was 6.8, with an approximate 90 percent confidence interval of 3.1 to 14.1. Ratio estimates above 3.0 for workers were found in the railroad industry and the lumber industry, and for sheet metal workers, grinding wheel operators, and automobile mechanics. (Auth. Abs. Mod.)

82–1269. Fraumeni, J. F., Jr. **Epidemiologic Approaches to Cancer Etiology.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 3. Palo Alto, California, Annual Reviews Inc., 1982, pp. 85-100.

Tobacco is identified as the principal risk factor for many cancers in this review of the major epidemiologic investigations in cancer etiology. The wide variation in cancer incidence between countries and populations suggests that the bulk of cancer in humans is environmental, and thus preventable. Demographic patterns have provided important clues for epidemiologic investigations. In examining the causes of cancer, the principal hazard is tobacco, which produces cancers of the lung, larynx, mouth, pharynx, esophagus, bladder, pancreas, and probably kidney. In men, tobacco accounts for 25 to 35 percent of all cancer deaths. In women this figure is 5 to 10 percent. However, lung cancer rates are rising sharply in women due to increasing smoking rates. The other causes of cancer discussed include alcohol, sunlight, radiation, occupational and genetic factors, pollutions, medicines, infections, and nutrition.

82–1270. Gibel, W. **Lung Cancer and Smoking in the German Democratic Republic.** *World Smoking and Health* 7(1): 27-29, Spring 1982.

Since 1953, lung cancer incidence has been increasing in East Germany: more than 6,500 cases in men and 900 in women are recorded per year. Cigarette consumption has increased by 53 percent from 1960 to 1978, due to earlier onset of smoking, a growing proportion of female smokers, and higher per capita consumption. Presently, 53 percent of men and 17 percent of women smoke, with the number of women smokers increasing rapidly. Antismoking measures consist of: (1) educating the young for a healthy lifestyle and against smoking, (2) preventing smoking at the workplace, (3) advising patients at health centers to quit, and (4) reducing the toxic content of cigarettes.

82–1271. Grant, I.W.B. **Screening for Lung Cancer.** *British Medical Journal* 284(6324): 1209-1210, April 24, 1982.

Routine screening of large numbers of people for asymptomatic malignant disease produces heavy demands on clinical and laboratory facilities and personnel. Since most patients who develop lung cancer are smokers, the population to be screened is limited, but still very large. Moreover, there is doubt that early diagnosis contributes significantly to survival. In an ongoing Mayo Clinic project, sputum cytology and chest radiography are being used in early diagnosis of lung cancer. To date, survey results suggest that close surveillance screening can save some lives, but how many is not known. Medically and economically, the evidence is against implementing such screening on a large scale. The best current means of reducing the lung cancer death rate is to dissuade people from smoking.

82–1272. Green, J. P.; Brophy, P. **Carcinoma of the Lung in Nonsmoking Chinese Women.** *Western Journal of Medicine* 136(4): 291-294, April 1982.

A San Francisco-based study of the records of 452 lung cancer patients seen between 1972 and 1979 points out the relatively high percentage of nonsmoking Chinese women with adenocarcinoma and large cell undifferentiated carcinoma of the lung. Combining these two subsets, there are 31 women, of whom 20 (64.5 percent) have never smoked. Of these, 28 (90.3 percent) were born in China and at least 12 (43 percent) of these patients were of Cantonese origin. There were no differences found with regards to age, stage of disease, place of birth in China, or other characteristics that could account for the variation between the Chinese female nonsmokers and smokers in the adenocarcinoma and large cell undifferentiated carcinoma groups. It is concluded that Chinese women, specifically those of Cantonese origin in San Francisco and elsewhere, are exposed to a potent lung carcinogen other than tobacco that may interreact with some traditional exposure to increase their susceptibility to adenocarcinoma and large cell undifferentiated carcinoma of the lung. (Auth. Abs. Mod.)

82–1273. Greenwald, P.; Weisburger, E. K.; Aaronson, S. A.; Evans, C. H.; Fink, M.; Hartley, J. W.; Henson, D.; Herberman, R. B.; Johnson, G. S.; Kohn, K. W.; Levine, A. S.; Mulvihill, J. J.; Rabson, A. S.; Simon, R. M. (Editors). **Cancer Mortality in the United States: 1950—1977.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 59, NIH Pub. No. 82-2435, April 1982, 484 pp.

This source book on national trends in site-specific cancer mortality from 1950 to 1977 by age, sex, and race provides a balanced mix of tabular and graphic analyses, summary statistics, and absolute numbers of deaths and population estimates for calculation of rates. Each major site is illuminated with two- and three-dimensional charts of trends as well as tables of annual age-, race-, and sex-specific death rates, numbers of deaths, and percentage of all deaths attributable to that site. Unique three-dimensional charts enable the reader to visualize the contribution of individual age-at-risk and birth cohort groups to the mortality trends over time. Data relating to 40 general cancer sites are featured in the main tables and charts. However, counts of deaths by year, age, sex, and race are provided at the fourth-digit level for all rubrics in three recent revisions of the International Classification of Diseases (ICD); the sixth, seventh, and eighth. The joining of these three ICD rubrics has been done so that continuity of classification by site over time is provided. No interpretative analyses are provided, but, for most sites, references are given which provide perspective on the temporal trends in mortality (or incidence) for each site. Three appendices are included: (1) annual tumor mortality by the ICD; (2) the sixth, seventh, and eighth revisions of the ICD for cancer; and (3) a discussion on the accuracy of cancer death certificates and its effect on cancer mortality statistics. (Auth. Abs. Mod.)

82-1274. Hallgren, R.; Nou, E.; Arrendal, H.; Hiesche, K. **Smoking and Circulating IgE in Bronchial Carcinoma.** *Acta Medica Scandinavica* 211(4): 269-273, 1982.

The serum concentrations of total immunoglobulin E (IgE) were significantly raised in smokers compared to those who had never smoked ($p<0.005$) among male patients with bronchial carcinoma, while no differences were found between smoking and nonsmoking female bronchial carcinoma patients. The total IgE levels in male and female control patients with nonmalignant pulmonary diseases were not correlated to smoking habits. No significant differences in the IgE levels were observed between smoking males subgrouped according to the World Health Organization histological types of bronchial carcinoma. Males with carcinoma who had stopped smoking more than 10 years ago had significantly reduced IgE levels compared to male cancer patients continuously smoking ($p<0.01$). These data, indicating that smoking is associated with elevated IgE levels in males with bronchial carcinoma, might suggest that smoking in certain, preferably male, individuals induces an impaired cellular immunity which is reflected by an enhanced IgE synthesis and a depressed resistance to carcinogens of tobacco smoke. (Auth. Abs.)

82-1275. Harwick, R. D. **Early Cancer Detection of the Oral Cavity and Pharynx.** *Medical Times* 110(4): 26S-27S, 31S, April 1982.

The typical oral or pharyngeal cancer patient is male, over age 50, is a long-time heavy smoker, and uses alcohol to excess. Recently, however, increasing numbers of female smokers and drinkers are being treated for this disease. Most of the cancers are squamous cell carcinomas, preceded by a premalignant lesion such as leukoplakia. Physicians are encouraged to visually examine the area of complaint in smoking and drinking patients reporting chronic sore throat, difficulty in swallowing, or a lump in the neck. The use of antibiotics in such cases may only delay proper diagnosis. Patients treated for oral or pharyngeal cancer have a better chance for cure, and are less likely to develop a second primary cancer if they give up smoking and drinking.

82-1276. Hinds, M. W.; Cohen, H. I.; Kolonel, L. N. **Tuberculosis and Lung Cancer Risk in Nonsmoking Women.** *American Review of Respiratory Disease* 125(6): 776-778, June 1982.

Among non-Caucasian women who have never smoked, those with a history of pulmonary tuberculosis infection may be at approximately eight times greater risk of lung cancer than nontubercular nonsmoking women. However, in the smoking women, no excess increased risk from tuberculosis infection was found. The increased incidence in nonsmokers may be due to physiologic changes in the tuberculous scar formation associated with cancer development, or to factors which predispose persons to both diseases. However, only 5.5 percent of the never-smokers with lung cancer had a history of tubercular infections; thus this factor may be relatively unimportant in lung cancer etiology. It is suggested that the already-shortened lifespan of smokers may mask any effect of tubercular infection on cancer incidence, especially if a long latent period were necessary.

82-1277. Hirayama, T. **Proportion of Cancer Attributable to Occupation Obtained From a Census, Population-Based, Large Cohort Study in Japan.** In: Peto, R.; Schneiderman, M. (Editors). *Quantification of Occupational Cancer*, Banbury Report 9, Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, pp. 631-649.

The proportion of cancer deaths attributable to various occupations was studied using data from a large-scale, census population-based ongoing cohort study in Japan since 1965. Findings showed 1.1 percent of deaths from cancer of all sites were due to industrial occupation. However, when interaction with daily cigarette smoking was considered, the percentage went up to 3.1 percent. A standardized mortality ratio (SMR) and 95 percent confidence intervals were calculated for cancer of all sites and cancer of the lung. A special analysis was done on the SMR for cancer of all sites, lung, and stomach in metal material workers, taking into consideration interaction with cigarette smoking. Though metal workers appear to carry a higher risk of cancer of all sites and selected sites, smoking interaction studies showed no elevation of risk for nonsmokers, but a rather striking risk elevation in daily smokers. Therefore, compared to the attributable risk (AR) due to smoking, AR due to this particular occupation was observed to be of limited value. It is concluded that because primary prevention is promising in the case of occupational cancer, the detection and control of occupational hazard and the detection and monitoring of lifestyle risk factors must have a high priority in any program of cancer prevention. Type of occupation showing significantly elevated risk for cancer of each site is listed in tabular form. (Auth. Abs. Mod.)

82-1278. Hirayama, T. **Changing Patterns in the Incidence of Gastric Cancer.** In: Fielding, J.W.L.; Newman, C. E.; Ford, C.H.J.; Jones, B. G. (Editors). *Gastric Cancer*, Advances in the Biosciences, Volume 32, Oxford, Pergamon Press, 1980, pp. 1-15.

Vital statistics in Japan from 1955 to 1978 and results of the National Nutritional Survey from 1949 to 1978 provided data which show a steady decline in gastric cancer in recent years, although in Japan the rate is still higher than in any other country. A review of factors possibly related to the changing trend reveals that the relative risk of stomach cancer in daily smokers compared to nonsmokers was 1.47 in males and 1.25 in females. Although this was much lower than the risk of lung cancer (4.13 in males and 2.10 in females), the absolute magnitude of excess deaths per 100,000 among daily cigarette smokers for gastric cancer was similar to that for lung cancer, 64.2 and 64.8 in males and 19.6 and 17.4 in females, respectively. The attributable risk percent related to daily cigarette smoking was 26.3 percent for gastric cancer, whereas it was 69.4 percent for lung cancer in males. The influence of age at initiation of smoking was even more striking than daily amount of smoking. However, frequent intake of green-yellow vegetables was noted to lower the gastric cancer risk in smokers and nonsmokers. Cessation of smoking and dietary improvement, particularly increased intake of vitamin A, are recommended.

82–1279. Jindal, S. K.; Malik, S. K.; Dhand, R.; Gujral, J. S.; Malik, A. K.; Datta, B. N. **Bronchogenic Carcinoma in Northern India.** *Thorax* 37(5): 343-347, May 1982.

Results of a study of 336 patients with bronchogenic carcinoma in Chandigarh, Northern India, suggest that its epidemiology differs in several respects from that in western countries, particularly in the younger age of the patients and the higher proportion of nonsmokers. Almost a third of all patients and 94.4 percent of the 54 women had never smoked. The peak frequency of bronchogenic carcinoma occurred between the ages of 51 and 60 years; 14.6 percent of the patients were less than 41 years. Of the 232 smokers and ex-smokers, 48.3 percent had smoked only cigarettes, 28.4 percent only bidis (made of naturally cured tobacco), 19.8 percent both cigarettes and bidis, and 3.4 percent hookahs. There was a clear association between duration of smoking and frequency of carcinoma. Tumors were classified in 287 (85 percent) of the patients. Squamous-cell carcinoma was relatively more frequent (32.4 percent) than any other tumor type and occurred almost exclusively in smokers. Adenocarcinoma was found in 13.2 percent of patients and was the most frequent tumor in nonsmokers. No differences of histological type were found between cigarette smokers and bidi smokers. (Auth. Abs.)

82–1280. Kaur, S.; Ali, B. **Stimulation of Drug and Carcinogen Metabolism by Prolonged Oral Tobacco Consumption.** *Biochemical Pharmacology* 31(8): 1595-1597, April 15, 1982.

Oral administration of tobacco to rats for 21 days caused remarkable stimulation of the metabolism of phenacetin, aniline, and benzo(*a*)pyrene, a carcinogen, by hepatic microsomal mixed function oxidases (MFO). Such treatment for 6 days resulted in a small increase in the activities of phenacetin *O*-dealkylase and aromatic hydrocarbon hydroxylase (AHH) without affecting aniline hydroxylase activity. Nicotine given orally was found to be a relatively weak inducer of phenacetin *O*-dealkylase and aniline hydroxylase, and elicited a maximum increase in their activities within 6 days which remained unchanged even after 21 days of continuous administration. However, these two enzyme systems were not affected following only one or two doses of tobacco and nicotine. Both tobacco and nicotine inhibited these biotransformations in vitro. (Auth. Abs.)

82–1281. Lamb, D.; Lumsden, A. **Intra-Epithelial Mast Cells in Human Airway Epithelium: Evidence for Smoking-Induced Changes in Their Frequency.** *Thorax* 37(5): 334-342, May 1982.

Differences in mast cells of the respiratory epithelium in smokers and nonsmokers were studied in 20 lungs removed for treatment of carcinoma. Of the lung cancer patients, 16 were smokers and 4 were nonsmokers. Besides typical mast cells, which normally contain metachromatic granules, surface microvillous processes, and intracytoplasmic granules with a whorled substructure, cells devoid of various features were also recognized. A spectrum of change occurred between those cells devoid of granular material and those with full granule content. A few cells contained both typical dense, whorled granules and large lucent vacuoles devoid of such material. Quantitation of all respiratory epithelial cells by light microscopy shows that mast cells comprise 0-2 percent of the total population. A higher proportion of mast cells was found in the epithelium of smokers than in nonsmokers. However, there was no relationship between loss of granule content and smoking, or granule content and number of mast cells. Because of the presence of many epithelial mast cells devoid of granule content, the use of traditional 4 μm paraffin sections stained with thiazine dyes to study mast cells may lead to a marked underestimation of the epithelial population. (Auth. Abs. Mod.)

82–1282. Laskarzewski, P.; Khoury, P.; Morrison, J. A.; Kelly, K.; Mellies, M.; Glueck, C. J. **Cancer, Cholesterol, and Lipoprotein Cholesterols.** *Preventive Medicine* 11(3): 253-268, May 1982.

The relationships of parental mortality and longevity to cancer and coronary heart disease risk factors (cholesterol and lipoprotein cholesterols) in their adult progeny are assessed utilizing the cross-sectional Cincinnati Lipid Research Clinic's Prevalence Study. Multivariate analysis of covariance was used to determine the effect of parental cause of death, if dead, and parental longevity, if alive, for plasma total cholesterol, triglyceride, high- and low-density lipoprotein cholesterol (C-HDL, C-LDL), of their adult progeny. The age, sex, race, cigarette use, alcoholic beverage consumption, dietary saturated fat and cholesterol intakes, recall group, and Quetelet index of the offspring served as covariables; the mothers' and fathers' "death" outcome served as classification variables. Family history was available on 1,702 parents (851 fathers, 851 mothers) of 851 adult offspring. Of the 42 percent of the progeny who were smokers, 23 percent smoked 1-10 cigarettes/day, and 48 percent smoked 11-20 cigarettes/day. Results indicated several significant parental mortality history covariable interactions for progeny's plasma cholesterol, C-HDL, and C-LDL. With respect to smoking, there was a significant interaction between cigarette smoking of progeny and their mother's cause of death. An increase in cigarette consumption from 0-50 cigarettes/day was associated with a fall in plasma cholesterol in progeny whose mothers died of heart disease. This decrease was significantly greater than the fall in plasma cholesterol with increasing cigarette use in progeny whose mothers were alive and older than 75 years, and also significantly different from the increase in cholesterol with cigarette use in progeny whose mothers died of cancer. It is concluded that despite the absence of cancer type and location, known and postulated cancer-specific risk factors, dietary cholesterol, alcohol intake, and cigarette use appeared in significant interaction terms with parental mortality for progeny's cholesterol and lipoprotein cholesterols. (Auth. Abs. Mod.)

82-1283. McCredie, M.; Ford, J. M.; Taylor, J. S.; Stewart, J. H. **Analgesics and Cancer of the Renal Pelvis in New South Wales.** *Cancer* 49(12): 2617-2625, June 15, 1982.

A lifetime history of analgesic and tobacco consumption was obtained by questionnaire from 67 patients with carcinoma of the renal pelvis (40 women, 27 men) and 180 control subjects. Regular consumption of analgesics conferred a tenfold relative risk of renal pelvic cancer in women and in men a risk ratio of 4 to 8. In women patients, some 74 percent of carcinomas of the renal pelvis and in men 43 percent could be attributed to analgesics. The corresponding attributable risks were 43 and 40 percent for tobacco: no dose—response relationship was demonstrated. Relative excess risks suggested a synergistic effect when both analgesics and tobacco had been consumed. Standardized risk ratios indicated a dose—response relationship for both phenacetin-containing and nonphenacetin analgesics: moderate consumption doubled the risk of renal pelvic cancer, and heavy consumption increased the risk to 6 to 16 times that for nonconsumers. (Auth. Abs. Mod.)

82-1284. McGregor, J. F. **Enhancement of Skin Tumorigenesis by Cigarette Smoke Condensate Following β-Irradiation in Rats.** *Journal of the National Cancer Institute* 68(4): 605-611, April 1982.

Results of a study of the tumor-promoting ability of cigarette smoke condensate (CSC), in rat skin after β-irradiation, showed: (1) the reduction in yield of noncarcinoma tumors for CSC treatment beginning immediately after irradiation, (2) the later increase in carcinoma yield for the same treatment, (3) the failure of CSC by itself to produce tumors, and (4) the relevance of the findings to human carcinogenesis. Skin tumors from male rats were classified into two groups: carcinomas and other noncarcinoma tumors. A statistically significant increase in tumor yield occurred after CSC treatment that began 2 months after irradiation, confirming a previous observation (*Journal of the National Cancer Institute* 56: 429-430, 1976). When CSC treatment began immediately after irradiation, the yield of noncarcinoma tumors was significantly reduced ($p<0.01$), whereas the increase in carcinoma yield was not significant. The increased yield of noncarcinoma tumors is attributed to a significant increase of acute ulceration caused by CSC on recently irradiated skin. The increase in carcinoma yield resulted from an increase in the rate of conversion of noncarcinoma tumors to cancer. Carcinoma yield was also increased by CSC treatment beginning 2 months after irradiation, but the increase was not significant. This lack of statistical significance may be ascribed to the insufficient number of cancers produced by the treatments. The relative ratios of cancer yields (1.7 and 2.5) did not differ greatly from the 2.2 ratio for the increase of noncarcinoma tumors. The possible relevance of the findings to human carcinogenesis is also discussed. (Auth. Abs. Mod.) See also 76-0338.

82-1285. McMichael, A. J. **Differentiating Work From Life-Style in Cancer Causation.** In: Peto, R.; Schneiderman, M. (Editors). *Quantification of Occupational Cancer,* Banbury Report 9. Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, pp. 577-587.

Whether or not lifestyle is able to be differentiated from occupation, it is concluded that the confounding and interactive effects of off-the-job factors in the quantification of occupational cancer must not be neglected. Omission of confounding effects will lead to an incorrect estimate of the risk attributable to occupational factors, while the omission of any interactive effects will lead to a misunderstanding of both the biological role and the relative importance of the occupational factor within the complex process of carcinogenesis. Since social class, as defined by occupation, has a class-related lifestyle, any comparison of cancer risks between jobs of different social classes will entail some degree of lifestyle-related confounding. However, in considering interactive possibilities, a further caution must be taken as in the case of the protective effect of "vitamin A" in lung cancer. If two populations differ in both smoking profile and vitamin A status, standardization via the conventional arithmetic for the confounding effects of smoking would be inappropriate, because the lung cancer-producing effect of smoking would depend in turn upon the vitamin A status of the smokers. The distinction between lifestyle and occupation must be considered when an epidemiologist attempts to understand and quantify occupation-related cancers.

82-1286. Napalkov, N. P.; Barchuk, A. S.; Dzhioyev, F. K. **Profilaktika Raka Legkogo. [Prevention of Lung Cancer.]** *Voprosy Onkologii* 28(2): 10-15, 1982, Russian.

The role of smoking in the development of lung cancer, other malignant tumors, and other chronic diseases (bronchitis, pneumonia, tuberculosis) is reviewed. Smoking is considered to be the principal risk factor for lung cancer, the incidence in heavy smokers being 15 to 30 times higher than in nonsmokers. Inhalation of cigarette smoke is an additional harmful factor. Animal experiments are cited which provide evidence of metaplastic changes in the bronchi of smokers: these pathologic changes regress after smoking cessation so that the risk of cancer after 5 to 10 years decreases to that of nonsmokers. The risk of lung cancer from atmospheric pollutants is higher for smokers than for nonsmokers. Prevention of lung cancer depends to a high degree on successful control of smoking. Activation of antismoking campaigns is considered essential, particularly among adolescents, women of childbearing age, pregnant women, and adult heavy smokers. Restrictions on smoking in public places and transportation, elimination of smoking from TV, movies, and theaters, and labeling of tobacco products as very dangerous to health are urged as smoking prevention measures. Mass medical examination of all long-term smokers over 50 is considered necessary.

82–1287. Napalkov, N. P.; Barchuk, A. S.; Merabishvili, V. M. **Vekotoryye Voprosy Profilaktiki Raka Legkogo i Organizatsiya Spetsializirovannoy Pomoshchi Zabolevshim. [The Problems of Lung Carcinoma Prevention and Organization of Specialized Aid to Patients.]** *Vestnik Khirurgii imeni I. I. Grekova* 128(1): 20-25, 1982, Russian.

Air pollution and smoking are considered the principal factors in the development of lung cancer in the Soviet Union and in most European countries. The cited statistics show a steady increase in morbidity and mortality rates of lung cancer in the industrialized countries. Soviet studies confirmed that the risk of lung cancer is directly proportional to the number of cigarettes smoked, that heavy smokers are 15 to 20 times more likely to get lung cancer than nonsmokers, and that the risk increases if one starts smoking at an early age or inhales smoke. In the 1970 to 1977 period, over 400,000 people died from lung cancer in the Soviet Union. It is therefore necessary to: (1) intensify the campaigns against smoking, particularly among students, soldiers, women of child-bearing age, pregnant women, and adult heavy smokers; (2) restrict smoking in public places, conferences, and in transportation; (3) cease showing smokers on TV and in movies; and (4) convince managers of the tobacco industry that labeling tobacco products as extremely dangerous to health is necessary. In discussing clinical measures of lung cancer prevention, chronic bronchitis in smokers is considered as a precancerous condition, and such patients, as well as all long-term smokers over 50, are classified as presenting "increased risk" of lung cancer.

82–1288. Pershagen, G.; Wall, S.; Taube, A.; Linnman, L. **On the Interaction Between Occupational Arsenic Exposure and Smoking and Its Relationship to Lung Cancer.** *Scandinavian Journal of Work Environment and Health* 7(4): 302-309, 1981.

A multiplicative effect is indicated in the interaction between occupational arsenic exposure and tobacco smoking and its relationship to lung cancer mortality among 228 deceased Swedish copper smelter workers. The case-referent technique was used to perform the study. Arsenic exposure was assessed via detailed company records, and information on smoking habits was gathered from the next of kin. The age standardized rate ratio for death from lung cancer was 3.0 for arsenic-exposed nonsmokers and 4.9 for smokers without occupational arsenic exposure in relation to nonarsenic-exposed nonsmokers. For arsenic-exposed smokers the rate ratio was 14.6, indicating a multiplicative effect of the two exposures. Eighty-five percent of all deaths from lung cancer among the smelter workers could be "explained" by arsenic exposure and/or smoking. The interaction between arsenic and smoking suggests that a strong preventive effect on lung cancer incidence could be obtained by decreasing either one of the exposures or by disaggregating them. (Auth. Abs. Mod.)

82–1289. Peto, R. **Trends in U.S. Cancer Onset Rates.** In: Peto, R.; Schneiderman, M. (Editors). *Quantification of Occupational Cancer.* Banbury Report 9. Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, pp. 269-284.

The estimation of trends in cancer onset rates is fraught with difficulties that must be allowed for in analysis to avoid errors that are of the same order of magnitude, or even greater, than the true trends. At present, the U.S. data demonstrate large and continuing increases in respiratory cancer that are due chiefly to the delayed rapid effects of people starting to smoke cigarettes still outweighing the lesser (though important) effects of recent decreases in tar yields and cigarette usage. The U.S. data also indicate large decreases in cancer of the stomach and uterus, large relative increases in melanoma, and a mixture of minor increases and decreases in the various other nonrespiratory cancers, with liver cancer mortality constant and bladder cancer mortality decreasing much faster than can be accounted for plausibly by improvements in cancer therapy. Future trends may differ substantially from recent trends, but at present the U.S. data contain no clear evidence for any generalized increase in cancer except for the delayed effects of tobacco. Opposite conclusions by other commentators appear to derive chiefly from methodological oversights. Such an examination of national trends does provide suggestive evidence against the view that a large percentage of today's cancers are due to occupational and environmental factors that were introduced during this century, but does not provide evidence against the more reasonable view that a small percentage are. But, even a small percentage of all cancers may represent several thousand U.S. cancer deaths a year. This is a large enough absolute number to justify efforts to seek out and reduce or eliminate any likely causes of substantial future numbers of cases of occupational cancer. (Auth. Abs.)

82–1290. Peto, R.; Schneiderman, M. (Editors). **Quantification of Occupational Cancer.** Banbury Report 9, Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, 776 pp.

Examples of efforts to trace the influence of substances encountered in the workplace on subsequent illness and death from cancer are explored, and what is known about the overall impact of occupation on cancer deaths is reviewed in over 40 papers. Subjects covered include asbestos and other mineral fibers, radiation risks, animal experiments, industry-wide cancer studies, trends in U.S. cancer rates and the proportion due to occupation, methodology and related problems, broad approaches to occupational cancer quantification, and future needs. Smoking's relationship to cancer rates in general and cancer of selected sites, particularly lung cancer, and to general occupational factors as well as specific occupations is discussed. A name index and a subject index are provided.

82–1291. Preston-Martin, S.; Henderson, B. E.; Pike, M. C. **Descriptive Epidemiology of Cancers of the Upper Respiratory Tract in Los Angeles.** *Cancer* 49(10): 2201-2207, May 15, 1982.

A descriptive epidemiology of cancer of three upper respiratory tract sites (lip; mouth and pharynx, excluding nasopharynx; and nose, sinus, and nasopharynx) was investigated using data from the Los Angeles County, California, cancer registry covering the period 1972-1976. Attributable risks calculated from earlier case—control studies were used to estimate the proportion of Los Angeles cases at each site related to tobacco use. Cases not attributable to tobacco use were also studied. Lip cancer was found to be a disease predominantly of white males with a male/female ratio of 9.8 for the lower lip and of 1.0 for the upper lip. Tobacco use is responsible for 60 percent of male lip cancers, and incidence rates of lip cancer after subtraction of the proportion of the incidence due to tobacco use were more than three times greater in men than in women. A hypothesis is proposed that an important etiologic factor is exposure to sun, wind, and cold which results in chapping; this exposure is most intense for the lower lip, and the use of lip coverings in women protects their lips both from the sun and from chapping. Cigarette smoking was responsible for 51 percent of mouth and pharynx cancer in men and women in Los Angeles County: after subtraction of the proportion of the incidence due to smoking, rates in men and women were similar. Poor dentition and nutritional deficiencies may help explain demographic characteristics such as the higher rates of mouth and pharynx cancer among men and women in the lower social classes and the secular decline in incidence rates. Cancers of the nose, sinus, and nasopharynx were found to occur among workers in a wide range of occupations and industries which involved inhalation of dust, fumes, and vapors. High incidence rates of nasopharynx cancer were observed among Chinese in Los Angeles and high rates of sinus cancer among Spanish-surnamed residents. Cancers at these sites appear to be related to the action of carcinogens on tissue chronically irritated by repeated upper respiratory infections or allergic responses. In addition, racial differences in the physical structure of the nasal passage may influence where inhaled particles are deposited. (Auth. Abs. Mod.)

82–1292. Radford, E. P. **Radon Daughters in the Induction of Lung Cancer in Underground Miners.** In: Peto, R.; Schneiderman, M. (Editors). *Banbury Report 9—Quantification of Occupational Cancer.* Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, pp. 151-159.

Five long-term studies of lung cancer incidence in miners are reviewed with respect to the role of smoking and radiation in etiology. Among U.S. uranium miners (1950-1974), cancers at first occurred only in smokers: cases among nonsmokers did not appear until later in the followup period. The Newfoundland fluorspar miners (1949-1971) were heavy smokers, and cancer deaths were three times as high as in the U.S. miners. Excess risk in these miners was also dose-related to radon daughter exposure. The Czechoslovakian uranium miners (1950-1975) were 70 percent smokers, yet showed a dose-rate effect of uranium exposure regardless of smoking status. An equivalent risk between smokers and nonsmokers also existed in the study of Swedish zinc miners (1956-1970). Among Swedish iron miners (1951-1976), however, excess risk of lung cancer in smokers was high compared to nonsmokers. The principal types of cancers have been epidermoid and small cell neoplasms with some excess of adenocarcinomas also occurring. Cancer cell type appears to be unrelated to smoking status.

82–1293. Reif, A. E. **The Causes of Cancer.** *American Scientist* 69(4): 437-447, July-August 1981.

An analysis of interrelationships among genetic factors, chemicals, diet, industry, radiation, estrogens, and viruses in the etiology of cancer shows that while some cancers are genetically fated to appear, most have now been traced to environmental factors with cigarette smoking dramatically advancing the time of death. Cigarette smoke is one of the most potent and highly relevant carcinogens in the environment, releasing 6,800 chemicals. The unaged gas phase of cigarette smoke contains several carcinogens of which dimethylnitrosamine (13 ng), hydrazine (32 ng), and vinyl chloride (12 ng) are representative. Several other classes of carcinogens are attached to the unaged smoke particles, the most prominent being benzopyrene (20 ng), polonium-210 (0.03-1.3 pCi), arsenic (1,000-25,000 ng), and the bladder carcinogen beta-naphthylamine (20 ng). Because of synergistic or cocarcinogenic factors, there is an excess number of lung cancer cases in city dwellers who smoke; among asbestos and uranium workers who smoke there is a tenfold risk. Bladder cancer presents a more complicated situation as many carcinogens target the bladder. However, heavy smokers present a threefold higher risk than nonsmokers in contracting this disease. It is concluded that since many cancer-causing agents are now identifiable, avoidance of these agents should lead to a reduction in incidence as evidenced by the dramatic reduction in lung cancer risk when one quits smoking.

82–1294. Rimington, J. **Cigarette Filters and Lung Cancer.** *World Smoking and Health* 7(1): 38-42, Spring 1982.

Results of a prospective study reveal a reduced risk of lung cancer in smokers of filtered vs. nonfiltered cigarettes. During 1970 and 1971, 10,414 men at least 40 years of age volunteered for mass radiography. Lung cancers which developed among the volunteers were recorded and identified by cancer registration records. After approximately 6 years, followup data showed that though filtered cigarette smokers were still at considerable risk for lung cancer, the risk is significantly lower than that of nonfiltered cigarette smokers. Also, it is possible that the difference in risk is underestimated, since most of the filter users smoked nonfiltered brands for many years prior to changing to filtered cigarettes.

82–1295. Rothschild, H.; Mulvey, J. J. **An Increased Risk for Lung Cancer Mortality Associated With Sugarcane Farming.** *Journal of the National Cancer Institute* 68(5): 755-760, May 1982.

Smoking was not found to be a confounding factor in this study of the association between increased lung cancer mortality and sugarcane farming. Employment histories (type of specific duties and materials handled) and smoking information were obtained in next-of-kin interviews of 284 of the total 815 lung cancer deaths that occurred in the 10 parishes (Louisiana) surveyed. Of the deaths reviewed, 38 percent of the victims had been employed as sugarcane workers for 6 months or longer. No heterogeneity was found between the odds ratios of smokers and nonsmokers with the use of a log linear model to detect a three-way interaction term. Decedents for whom cigarette pack/year data were available were classified as "moderate" ($\leq$ 40 pack/year), or "heavy" ($>$ 40 pack/year) smokers. No significant heterogeneity in the odds ratios for lung cancer was found for moderate smokers (odds ratio = 1.7) and heavy smokers (odds ratio = 2.7). The odds ratio for sugarcane farmers adjusted for smoking was 2.4 (95 percent confidence interval 1.7-3.6).

82–1296. Schottenfeld, D. **Snuff Dipper's Cancer.** *New England Journal of Medicine* 304(13): 778-779, March 26, 1981.

An excess of oral cancer occurred among women of the rural southeastern United States, where snuff dipping is a common habit. Approximately 30 percent of North Carolina women use snuff: the proportion of oral and pharyngeal cancer which would be prevented by elimination of snuff use is estimated to be 35 percent for white women and 21 percent for black women. In the northeastern United States, oral cancer mortality is higher among men, and is attributed to tobacco combined with alcohol. Where the use of snuff, chewing tobacco, or quid (tobacco combined with the betel nut) is common, such as India, China, Thailand, Ceylon, and the Central Republics of the Soviet Union, mortality rates from oral cancer are among the highest in the world. Mixing snuff and the smoking habit apparently does not potentiate oral cancer occurrence.

82–1297. Shillitoe, E. J.; Greenspan, D.; Greenspan, J. S.; Hansen, L. S.; Silverman, S., Jr. **Neutralizing Antibody to Herpes Simplex Virus Type 1 in Patients with Oral Cancer.** *Cancer* 49(11): 2315-2320, June 1, 1982.

Neutralizing antibody to Herpes simplex virus type 1 (HSV-1), type 2, and measles virus was measured in the serum of patients with oral cancer (n=40), patients with oral leukoplakia (n=21), and in control subjects who were smokers (n=20) and nonsmokers (n=21). Significantly higher titers to HSV-1 were found in controls who smoked than in controls who did not smoke. Patients with untreated oral cancer (n=21) had HSV-1 neutralizing titers similar to those of the controls who smoked, but those with later stage tumors had higher titers than those with earlier stage tumors. In patients who were tumor-free after treatment for oral cancer (n=19), higher antibody titers to HSV-1 were associated with longer survival times. No association was found between clinical status and antibody to measles virus. The data are consistent with a role for both HSV-1 and smoking in the pathogenesis of oral cancer. (Auth. Abs.)

82–1298. Swartz, J. B.; Epstein, S. S. **Problems in Assessing Risk From Occupational and Environmental Exposure to Carcinogens.** In: Peto, R.; Schneiderman, M. (Editors). *Quantification of Occupational Cancer.* Banbury Report 9. Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1981, pp. 559-575.

Studies on occupational cancer have often minimized the carcinogenic effect of occupational exposure and thus discouraged critically needed research and regulation. Previous methodological limitations include: (1) improper consideration of the tumor induction period, (2) the effect of cessation of exposure, (3) the age effect, (4) competing risks, and (5) the healthy worker effect. In refuting the mythology that smoking is responsible for the overwhelming majority of lung cancer in workers, and that recent increases in lung cancer rates are due largely to smoking, it is noted that smoking rates in the United States are declining for men, so it would seem reasonable to infer that the number of lung cancers attributable to smoking should be going down, not up. In addition, various estimates have placed the percent of male lung cancer among nonsmokers at approximately 20 percent, although some data indicate that this rate doubled between 1958 and 1969, and in many occupational groups, the overall lifetime incidence for smokers is not substantially different than for nonsmokers. The role of occupation was not considered in most classical studies on lung cancer, and the high relative risk figures for the carcinogenic effects of smoking are not borne out or are contradicted by other carefully documented studies. It is also noted that smoking cannot account for the sharp increase in lung cancer among blacks.

82–1299. U.S. Department of Health and Human Services. **The Health Consequences of Smoking: Cancer. A Report of the Surgeon General.** U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health, DHHS Pub. No. (PHS) 82-50179, 1982, 341 pp.

This volume represents the 14th report of the Department of Health and Human Services to the Congress on the health consequences of smoking. Unlike other reports in the series, the current volume examines the relationship of smoking, tobacco and tobacco use to a single disease entity—cancer. The report is divided into four major sections: a review of the biomedical evidence linking smoking and tobacco use by cancer site; the mechanisms of carcinogenesis including a detailed discussion of tobacco carcinogenesis; the relationship between involuntary smoking and lung cancer in nonsmokers; and cessation of smoking among adolescents and adults. The major findings included the following: Tobacco use is the single major cause of cancer mortality in the United States, estimated to be responsible for 30 percent of all cancer deaths. Smoking, particularly cigarette smoking, is a major cause of cancer of the lung, larynx, oral cavity and esophagus and is a contributory factor in cancer of the bladder, kidney, and pancreas. An association was noted between smoking and stomach cancer and cancer of the uterine cervix; however, there was insufficiant evidence for a judgment of causality. A link between involuntary smoking and lung cancer among nonsmoking wives of husbands who smoked was noted, and concern for this relationship was expressed due to the large number of people in the population who are potentially exposed. Nonsmokers were advised to avoid passive, or involuntary smoking exposures to the extent possible. A dose-response relationship was noted between smoking and most cancer sites, with the risk of cancer mortality increasing with increased exposure as measured by number of cigarettes smoked per day, early age of initiation, number of years smoked, inhalation patterns, and the type of cigarette smoked. Cessation of smoking was accompanied by a reduction in cancer risk. Former smokers, 10 to 15 years off cigarettes, had overall site-specific cancer death rates that were similar to those observed in nonsmokers. Numerous animal studies have demonstrated the carcinogenicity of tobacco and tobacco smoke. Individual compounds identified in smoke have been found carcinogenic to laboratory animals. A number of tobacco-specific nitrosamines have been identified and certain tobacco products, such as snuff, have the highest levels of such carcinogens in any consumer product taken into the body. Long-term users of snuff as well as chewing tobacco have been found to have elevated risks for oral cancers as well as noncancerous oral diseases. An estimated 9 out of 10 smokers who quit do so on their own without the aid of formal smoking cessation programs. Research evaluations of self-help aids have reported success rates of up to 50 percent cessation at extended periods; however, most estimates of success rates report only around 5 to 20 percent. Social support for smokers who are attempting to quit was emphasized.

82-1300. Vena, J. E. **Air Pollution as a Risk Factor in Lung Cancer.** *American Journal of Epidemiology* 116(1): 42-56, July 1982.

Retrospective data on residential and employment history and on smoking were obtained from 417 white male lung cancer patients and 752 controls with nonrespiratory, nonneoplastic diseases, from Erie County, New York, admitted to Roswell Park Memorial Institute from 1957 to 1965. Total suspended particulate data and a historical review of problem point sources of pollution were used to delineate air pollution zones. The findings did not support the hypothesis that air pollution alone significantly increased risk for lung cancer. However, there was increased risk from smoking and occupational exposures if there was also long-term exposure to air pollution. The risk for heavy smokers with heavy exposure to air pollution was over four times that of men with none of the high exposure traits. The findings suggest an apparent synergistic mechanism involving smoking and air pollution and smoking and occupational exposures. (Auth. Abs.)

82-1301. Weisburger, J. H.; Williams, G. M. **Chemical Carcinogens.** In: Casarett, L. J.; Doull, J.; Klaassen, C. D.; Amdur, M. D. (Editors). *Casarett and Doull's Toxicology: The Basic Science of Poisons.* New York, MacMillan, 1980, pp. 84-138.

A detailed discussion of chemical carcinogenesis is presented including classification, mechanism of action and reaction, modifying factors, and bioassays. The two categories of carcinogens are: genotoxic, which affect genetic material by single exposure, cumulative, and/or synergistic effects; and epigenic, or noninteractive with genetic material, such as solid-state, hormones, immunosuppressants, cocarcinogens, and promoters. The best known plant carcinogen is tobacco, which contains nitrosonornicotine and related agents even before burning and pyrolysis, and tobacco smoke, containing diverse classes of carcinogens, cocarcinogens, accelerators, and promoters which can act on the same target organ with additive and sometimes syngeristic effects. Asbestos, a solid-state carcinogen which remains permanently in lung tissue, contributes synergistically to lung carcinoma development when it potentiates the action of other carcinogens such as cigarette smoke. Alcohol is also an excellent cocarcinogen. Whereas smokers rarely develop carcinoma in the upper gastrointestinal tract or oral cavity, those who drink are at increased risk, as alcohol promotes the action of the procarcinogens present in tobacco. Promotion is a reversible activity requiring a continuous presence of the promoting agent. If the effect of tobacco smoke were attributable only to primary carcinogens, the risk would not decrease so appreciably upon cessation of the habit.

82-1302. White, M. **A Retrospective Study of 100 Cases of Carcinoma of the Lung.** *Irish Medical Journal* 75(4): 106-107, April 1982.

Despite advances in diagnosis and treatment, survival among lung cancer patients remains essentially unaltered. To update a 1971 analysis of lung cancer status in Ireland, a retrospective study was done of 100 patients with histologically proven carcinoma of the lung. Only 9.8 percent had survived for 3 years. Of the 100 patients, over 80 percent were smokers, with an average consumption of 27 cigarettes per day. The age range was 26 to 85 years, with a mean age at presentation of 61.54 years. The mean duration of symptoms before presentation was 9.8 weeks; it is possible that urban patients seek medical advice earlier than the rural patients of previous studies. It is also noted that the increase in lung cancer among females has lessened the male:female ratio from 11.5:1 in 1958 to 4:1 at the present. The younger patients and those with resectable lesions (reflecting early diagnosis) survived the longest. Of key importance is the accurate diagnosis of the tumor, its cell type, and its extent.

NON—NEOPLASTIC RESPIRATORY DISEASES

82–1303. Akbarkhanzadeh, F. **Combined Effects of Cigarette Smoking and Occupational Air Pollution in Welding Environment.** *Medicina del Lavoro* 72(1): 61-66, January-February 1981.

Although smoking impairs lung function, this effect was found to be clear and separate from the effect of welding fumes on pulmonary function in this study of shipyard workers in Sunderland, England. There were no differences among respiratory symptoms categories of the 318 subjects (65.7 percent welders, 34.3 percent nonwelders) with regard to their smoking habits. Chronic effects of smoking on lung function were approximately the same as those of welding fumes. The reduction in pulmonary function indices was significantly correlated to the daily consumption of tobacco and period of smoking in nonwelders, especially in ex-smokers. Smoking prior to lung function testing or long-term exposure to welding fumes seem to obscure the probable correlations.

82–1304. Aronson, M. D.; Weiss, S. T.; Ben, R. L.; Komaroff, A. L. **Association Between Cigarette Smoking and Acute Respiratory Tract Illness in Young Adults.** *Journal of the American Medical Association* 248(2): 181-183, July 9, 1982.

A study of cigarette smoking and the occurrence and severity of an acute respiratory tract illness (ARTI) revealed approximately a two-fold risk among smokers. Clinical data were obtained prospectively on 867 men and women with an ARTI and on a control group of 289 women. Of the 534 women in the ARTI group, 307 (57 percent) were smokers, compared with 97 (34 percent) of the 289 women in the control group, a highly significant difference. Of all those with ARTIs, 506 were smokers. Smokers had a statistically significant greater likelihood of having a lower respiratory tract illness (57 percent vs. 45 percent), a longer duration of cough (8.9 vs. 6.8 days), and a greater frequency of abnormal auscultatory findings (31 percent vs. 20 percent) than did the 361 nonsmokers. Cigarette smoking may thus contribute to the substantial morbidity and lost productivity resulting from ARTI. (Auth. Abs.)

82–1305. Barnes, R. **The Relative Importance of Smoking Compared to Occupation in Decreased Pulmonary Function.** *Medical Journal of Australia* 1(1): 25-28, January 9, 1982.

Following a long-term study of smoking and nonsmoking workers in New South Wales who had become disabled by pneumoconiosis, it is concluded that smoking affects lung function to a far greater degree than does occupational exposure to dust. The study compared the forced vital capacity and forced expiration volume in 1 second of 684 workers grouped according to smoking habits and to disability from pneomoconiosis. The study was longitudinal and, although the pulmonary function tests decreased with age, as expected, the fall was greater in smokers without occupational lung disease than in nonsmokers with occupational lung disease. (Auth. Abs. Mod.)

82–1306. Bauer, L. A.; Blouin, R. A. **Influence of Age on Theophylline Clearance in Patients With Chronic Obstructive Pulmonary Disease.** *Clinical Pharmacokinetics* 6(6): 469-474, November-December 1981.

Theophylline total body clearance was calculated in 59 adult patients requiring intravenous aminophylline therapy. Group 1 consisted of 36 chronic obstructive pulmonary disease patients who were cigarette smokers without other conditions known to alter theophylline kinetics (age range: 22 to 79 years). Group 2 consisted of 23 chronic obstructive pulmonary disease patients who were cigarette smokers with a similar degree of congestive heart failure, but free of other characteristics or diseases that affect theophylline disposition (age range: 41 to 81 years). When individual theophylline total body clearance values were plotted against age in each group, poor correlation coefficients were found, indicating that age is not a good predictor of total body clearance of theophylline (group 1: $r=0.101$; group 2: $r=0.276$). Each group was also broken into "younger" and "older" patients. No significant differences in theophylline total body clearance values were found between the younger and older patients for either group 1 or 2, suggesting that a theophylline dosage reduction is not necessary in cigarette smoking patients over an arbitrary age limit. (Auth. Abs.)

82–1307. Beaty, T. H.; Cohen, B. H.; Newill, C. A.; Menkes, H. A.; Diamond, E. L.; Chen, C. J. **Impaired Pulmonary Function as a Risk Factor for Mortality.** *American Journal of Epidemiology* 116(1): 102-113, July 1982.

Prospective followup information obtained between 1976 and 1981 on mortality among 2,539 individuals showed that pulmonary function impairment is a risk factor for short-term mortality, even when risk factors such as age, sex, and smoking are considered. Smoking has a significant impact on mortality, with never-smokers having far fewer deaths than heavy smokers. However, predicted risk curves for impaired individuals (those with forced expiratory volume in 1 second less than 68 percent of forced vital capacity) are consistently higher among all race—sex categories over all ages. Survival analysis using the proportional hazards model shows a steeper decline in estimated survival among individuals with poor pulmonary function compared to those with good pulmonary function, adjusted for age, race, and smoking effects. These observations are consistent with the concept that impairment of pulmonary function is a risk factor for morbidity and mortality from several nonrespiratory as well as respiratory diseases, and that it acts by contributing to various pathogenic mechanisms in different organ systems. (Auth. Abs. Mod.)

82–1308. Calverley, P.M.A.; Leggett, R. J.; McElderry, L.; Flenley, D. C. **Cigarette Smoking and Secondary Polycythemia in Hypoxic Cor Pulmonale.** *American Review of Respiratory Disease* 125(5): 507-510, May 1982.

The red cell mass (RCM) in 47 hypoxic patients with chronic obstructive pulmonary disease (COPD) was studied relative to their smoking habits and carboxyhemoglobin concentrations. The mean RCM was 42.5±8.0 ml/kg in the 31 patients who still smoked, significantly higher than in the 16 who were currently nonsmokers (RCM, 29.7±4.4 ml/kg). Measurements of arterial oxygen tension (Po_2), pH, oxygen tension at 50 percent hemoglobin saturation (P_{50}), and carboxyhemoglobin (COHb) showed that the arterial oxygen saturation (SO_2A) was less well correlated with RCM in the smokers than was the saturation of total oxygen binding sites (SO_2T), including a corrective term for COHb. The RCM correlated well with the mean outpatient COHb measured repeatedly over 6 to 36 months in 40 of the patients but poorly with their average arterial oxygen saturation. In 15 patients given long-term oxygen therapy for 12 months, RCM decreased significantly only in those who stopped smoking, as shown by a decrease in COHb. It is concluded that cigarette smoking may determine the severity of secondary polycythemia in patients with hypoxic COPD, and prevent its correction by long-term oxygen therapy. (Auth. Abs. Mod.)

82–1309. Clement, J.; Van De Woestijne, K. P. **Rapidly Decreasing Forced Expiratory Volume in One Second or Vital Capacity and Development of Chronic Airflow Obstruction.** *American Review of Respiratory Disease* 125(5): 553-558, May 1982.

In a prospective study of 2,406 members of the Belgian Air Force, followed for 3 to 15 years, 325 demonstrated a yearly decline in forced expiratory volume in 1 second (FEV_1) and/or in vital capacity (VC) that was significantly faster than that expected in healthy nonsmokers. Such rapid declines were found more frequently in heavy smokers and in subjects with a reduced FEV_1 or VC. There was no clear-cut association between rapid declines and a history of respiratory diseases. It may be predicted, if the rates of declines do not vary with aging, that about 0.5 percent of nonsmokers and 4 percent of heavy smokers in the investigated population will reach disabling airway obstruction (FEV_1 of 1.2 L or less) at 65 years of age. At least 6 to 8 years of followup are required to appreciate with precision the rates of declines in FEV_1 or in VC. (Auth. Abs.)

82–1310. Coggins, C.R.E.; Lam, R.; Morgan, K. T. **Chronic Inhalation Study in Rats, Using Cigarettes Containing Different Amounts of Cytrel Tobacco Supplement.** *Toxicology* 22(4): 287-296, 1982.

Cigarettes containing differing amounts of the tobacco substitute Cytrel produced proportionately fewer respiratory tract lesions in rats. Histological examinations after chronic inhalation of cigarette smoke containing 0, 25, 50, and 100 percent Cytrel revealed considerably less marked changes according to tobacco vs. Cytrel content, despite similar total smoke deliveries. The decrease in metaplastic changes in the trachea and alveoli was also approximately proportional to the Cytrel level of the cigarette. The mean weights in all exposed groups were significantly lower than controls: there were no differences in weight between the exposed groups. Cytrel cigarettes generally have lower smoke yields, and the smoke chemistry is less complex. No novel effects from Cytrel were observed, and there should be no objections to blending Cytrel with tobacco products.

82–1311. Coggins, C.R.E.; Musy, C.; Ventrone, R. **Changes in the Minute Ventilation of Rats Exposed to Different Concentrations of Cigarette Smoke.** *Toxicology Letters* 11: 181-185, 1982.

Measurements of tidal volume and breathing frequency were made in rats during a 30-minute period of exposure to cigarette smoke, and were compared with those obtained during a 10-minute pre-exposure period. All measurements were made in animals previously habituated to smoke exposure. Both tidal volume and breathing frequency were decreased by low-dose exposure to smoke, resulting in a 34 percent decrease in minute volume. At high-dose exposures (double the low-dose smoke concentration) tidal volumes showed a 23 percent increase over pre-exposure values with little change in breathing frequency, giving a net 20 percent increase in minute ventilation. Results indicate an apparent inverse dose-relationship in that the response at low-dose exposure was a reduction in minute ventilation, whereas a greater "insult" resulted in increased ventilation. It is possible that vapor phase constituents such as formaldehyde and acrolein are involved in the minute volume response, as a depression in breathing frequency resulting from exposures to such compounds has been reported. (Auth. Abs. Mod.)

82–1312. Cohn, M. A.; Rao, A.S.V.; Broudy, M.; Birch, S.; Watson, H.; Atkins, N.; Davis, B.; Stott, F. D.; Sackner, M. A. **The Respiratory Inductive Plethysmograph: A New Non-Inva-**

sive Monitor of Respiration. *Clinical Respiratory Physiology* 18(4): 643-658, 1982.

The respiratory inductive plethysmograph (RIP) is a good semiquantitative, noninvasive monitor of breathing in normal subjects and smokers with and without minimal respiratory disease. The RIP consists of insulated coils of wire encircling the rib case and abdomen in which the self-inductances of the coils and frequencies of the connected oscillators change with respiration as a function of the cross-sectional areas of the enclosed compartments. Calibration to spirometry is obtained by (a) solving two simultaneous equations which relate varied contributions of rib cage and abdominal displacements to the sum during breathing in two body postures, or (b) employing an isovolume maneuver in a single position. The device was calibrated using technique (a) and changes of lung volumes measured by RIP were compared with those measured by simultaneous spirometry (SP) in 33 young, healthy subjects. Eighty-eight percent of the means of tidal volumes measured by RIP in seven different body positions fell within ±10 percent of SP. For slow vital capacity, 80 percent of mean values by RIP fell within ±10 percent of SP. For maximum voluntary ventilation maneuvers, 81 percent of values by RIP fell within ±20 percent of SP. In eight subjects in which RIP was calibrated with the two simultaneous equation method, mean tidal volumes were measured simultaneously with RIP and SP, and the isovolume line angles in each of seven body positions. Eighty-six percent of mean tidal volumes measured by RIP fell within ±20 percent of SP, but 50 percent of the isovolume line angles were less than 40° or greater than 50°. Thirty degrees of anteroposterior and lateral hip flexion did not alter validation of RIP, but 90° anterior hip flexion gave unsatisfactory validation (+ 14 percent) to SP. The two simultaneous equation method promotes volume equivalency of RIP to SP over a wide range of body postures and spinal attitudes at the expense of variable errors in determination of the rib cage to abdominal contributions to tidal volume. No differences occurred between the smokers and nonsmokers in the spirometry or RIP values. (Auth. Abs. Mod.)

82-1313. Elwood, P. C.; McAulay, I. R.; Elwood, J. H. **The Flax Industry in Northern Ireland Twenty Years On.** *Lancet* 1(8281): 1112-1114, May 15, 1982.

Because of the high cost to industry of the great increase in byssinosis compensation claims, a followup of a 1961-1962 survey of respiratory symptoms in 2,528 flax mill workers in Northern Ireland was undertaken in 1978. Flax dust appears to have an acute, reversible, specific effect on the respiratory systems of exposed persons and it is suggested that this effect is more noticeable in those whose respiratory tract is irritated or whose lung function is impaired. Such a view is consistent with the much higher prevalence of byssinosis in smokers than in nonsmokers. In a detailed examination of the interrelations between dust levels and byssinosis and chronic bronchitis incidence, it was found that, whereas byssinosis and dust levels were slightly correlated, there was no association between chronic bronchitis and dust after taking into account the effects of smoking. Overall, the followup study found no evidence of an effect on survival of either exposure to dust or byssinosis and, therefore, concluded that financial settlements reached appeared unreasonable. It is recommended that (1) rigorous preemployment medical examinations along with regular check-ups, including lung function tests, be instituted for those at risk for byssinosis; (2) workers with early signs of byssinosis must be transferred to jobs where they will not be exposed to flax dust; and (3) advice on the additional dangers of tobacco smoking must be given repeatedly.

82-1314. Fawer, R. F.; Gardner, A. W.; Oakes, D. **Absences Attributed to Respiratory Diseases in Welders.** *British Journal of Industrial Medicine* 39(2): 149-152, May 1982.

Certified sickness rates of 36 male welders were examined for the period 1970 to 1979. They were compared with 36 male controls from the same petrochemical plant, matched for age, smoking habits, duration of employment, and social class. Indices of severity (average annual duration), duration (average length of spell), and frequency (inception rate of spells and inception rate per worker) were calculated. Absences for all diseases were similar, but absences attributed to respiratory diseases were slightly higher in welders in severity, duration, and frequency. The proportion of days lost attributed to respiratory diseases was two to three times higher in welders compared with controls. This was due to a large increase in absences attributed to lower respiratory tract diseases, the ratio of welders to controls being more than four to one for both severity and inception rate of spells. The comparison between welders and controls in smokers and nonsmokers confirms other studies which show that smokers tend to be more affected by welding fumes than nonsmokers. (Auth. Abs.)

82-1315. Frey, J.; Chamson, A. **Cytopharmacologie sur Culture de Fibroblastes. [Cytopharmacology in Fibroblast Culture.]** *Annales de l'Anesthesiologie Francaise* 21(6): 603-608, 1980, French.

The toxic effect of tobacco smoke extracts on cell growth and collagen synthesis was studied in human fibroblast culture, as a model for observing the pathology of pulmonary fibrosis. The tobacco smoke extracts were prepared by bubbling smoke into a buffered aqueous solution at pH 7.0. The experiments showed that tobacco smoke toxicity cannot be attributed exclusively to the nicotine content of the extract; a 50 percent inhibition of cell growth was obtained with the dark or light tobacco extracts at a dose corresponding to 15 moles of nicotine/liter, as well as with 970 μmol/l of pure nicotine. Biosynthesis of total proteins and collagen increased in fibroblast cultures treated with the tobacco smoke extracts. Also, secretion of the synthesized noncollagen proteins was disturbed by the tobacco smoke extract; the type III collagen was decreased, and the type I trimer collagen was increased. The number of mitochondria and vacuoles increased in the presence of tobacco smoke extract at a given nicotine concentration.

82-1316. Fridriksson, H. V.; Malmberg, P.; Hedenstroem, H.; Hillerdal, G. **Reference Values for Respiratory Function Tests in Males: Prediction Formulas With Tobacco Smoking Parameters.** *Clinical Physiology* 1(4): 349-364, 1981.

Lung function reference values are needed before the effects of smoking and occupational exposures can be assessed. For this reason, prediction equations for respiratory function tests were developed from a multiple regression analysis of data from 263 healthy males. The subjects ranged from 20 to 70 years in age, about one-third each of nonsmokers, smokers, and ex-smokers. Measurements were taken of lung volumes (with body plethysmograph), airways resistance, ventilatory capacity including flow-volume registration, gas distribution and closing volume, transfer factor, and static elastic recoil pressures of the lung with calculation of static compliance. The parameters age, height, weight, years of tobacco smoking, and grams of tobacco smoked each day showed significant correlation with test results of most of the respiratory functions. Therefore, a set of basic regression equations including these parameters was calculated. In addition, an extended set of equations was calculated for prediction of some tests with inclusion of nonlinear terms and the parameter "years of abstinence from smoking." The reduction in variance which followed inclusion of tested parameters was moderate (20-69 percent). (Auth. Abs. Mod.)

82-1317. Gortmaker, S. L.; Walker, D. K.; Jacobs, F. H.; Ruch-Ross, H. **Parental Smoking and the Risk of Childhood Asthma.** *American Journal of Public Health* 72(6): 574-579, June 1982.

The relationship between parental smoking and the prevalence of asthma in children aged 0-17 was assessed in two random population surveys. Data from a 1977 Midwestern urbanized county indicate that, if mothers smoked, the prevalence of parent-reported asthma increased from 5.0 percent to 7.7 percent (estimated relative risk of 1.5), and the prevalence of functionally impairing asthma increased from 1.1 percent to 2.2 percent (relative risk of 2.0). In a more rural eastern county in 1980, a lower overall prevalence of asthma was noted. However, similar estimated relative risks of asthma (1.8) and functionally impairing asthma (2.4) were found to be associated with maternal smoking. Inconsistent relationships were found between the estimated prevalence of asthma and paternal smoking. When multivariate controls were introduced, the relationships between maternal smoking and asthma persisted. Estimated attributable risks indicate that between 18 percent and 34 percent of the asthma reported in these samples can be attributed to maternal smoking. Implications of these findings for primary care physicians are discussed. (Auth. Abs.)

82-1318. Hedenstierna, G.; Jorfeldt, L.; Bygdeman, S. **Flow-Volume Curves in Healthy Non-Smokers and in Smokers.** *Clinical Physiology* 1(4): 339-348, 1981.

Maximum expiratory flow-volume curves were recorded in lung healthy nonsmokers (n=231) and in smokers (n=95). Multiple linear regression analysis was performed with regard to sex, age, height, and weight. The regressions for maximum expiratory flow at 50 percent and 25 percent of vital capacity (MEF$_{50}$ and MEF$_{25}$, respectively) on age were significantly different for nonsmokers and smokers, in males as well as females, but the large scatter around the regression line resulted in more than 50 percent of presently studied smokers to fall within 1 SD of the reference values for nonsmokers. It is concluded that the individual variability in MEF$_{50}$ and MEF$_{25}$ is relatively large even when sex and age are accounted for and that there is no further benefit in including height or weight in the regression equations. The discriminating ability of the MEF$_{50}$ and MEF$_{25}$ variables is small, indicated by considerable overlapping between nonsmokers and smokers. (Auth. Abs. Mod.)

82-1319. Hopkin, J. M.; Tomlinson, V. S.; Jenkins, R. M. **Variation in Response to Cytotoxicity of Cigarette Smoke.** *British Medical Journal* 283(6301): 1209-1211, November 7, 1981.

The cytotoxic effect of cigarette smoke condensate on human polymorphs was investigated in vitro by the method of vital dye exclusion. Exposure to 1/800 of the smoke from one high-tar cigarette killed a detectable proportion of a population of 10^6 cells. The response among the cells from 40 healthy people varied widely, the percentage of dead cells ranging from 3 to 66 percent and from 17 to 87 percent at exposure levels of 125µg and 250µg of cigarette smoke condensate, respectively. Differences in individuals' responses were reproducible and unrelated to age or sex or smoking habit. The cells from 10 patients with irreversible obstructive airways disease and probable emphysema were significantly more sensitive than those from 10 patients with no respiratory disability matched for age and smoking habits. Genetically influenced variation in cellular response to cytotoxicity may be an important determinant of the risk of developing emphysema among smokers. (Auth. Abs.)

82-1320. Jarvholm, B.; Bake, B.; Lavenius, B.; Thiringer, G.; Vokmann, R. **Respiratory Symptoms and Lung Function in Oil Mist-Exposed Workers.** *Journal of Occupational Medicine* 24(6): 473-479, June 1982.

The prevalence of respiratory symptoms was registered and ventilatory function was determined in 164 men exposed to aerosols from straight oils (turners), emulsions (grinders), and condensation products of evaporated oil (hardeners), and 159 controls (office workers) from the same company. Tabulated data show the importance of stratification into smoking categories, although exposed workers, both smokers and nonsmokers, reported more respiratory symptoms than controls, indicating a dose—response relationship to oil mist exposure. Multivariate analysis of age, height, and exposure did not produce any significant differences in ventilatory function, but smoking habits did influence the results, indicating some lung function impairment due to smoking. The lung function of 25 nonsmoking exposed men was further examined with other lung function tests. The mean values for closing volume, slope of the alveolar plateau, total lung capacity, residual volume, elastic recoil at various lung volumes, and diffusion capacity did not differ significantly from controls.

82-1321. Jeffery, P. K.; Ayers, M.; Rogers, D. F. **The Mechanisms and Control of Bronchial Mucous Cell Hyperplasia.** *Chest* 81(5, Supplement): 27S-29S, May 1982.

Pulse labeling of cell nuclei of the left main extrapulmonary bronchus of rats was employed to study the effects of tobacco smoke exposure with and without the addition of anti-inflammatory agents. Clinically pathogen-free male rats, exposed to tobacco smoke daily for up to 6 weeks, were examined to determine (1) the relative importance of cell division, differentiation, and transformation on observed mucous cell increase; and (2) the extent to which different anti-inflammatory agents might inhibit or modify these smoke-induced changes. In preliminary tests of cell differentiation, which compared normal bronchial epithelium with tobacco smoke-exposed bronchus, the smoke is shown to have altered the normal (time scale) pattern of differentiation. Recent studies have indicated that the anti-inflammatory drug phenylmethyloxadiazole, as well as other nonsteroidal anti-inflammatory agents, will inhibit the thickened epithelium, cell hypertrophy, increase in epithelial mucous cell number and dividing cells in animals exposed to tobacco smoke. In this study, tobacco smoke exposure produced a significant increase in number of mucous cells at all airway levels examined. All the agents tested (dexamethasone, prednisolone, hydrocortisone, indomethacin) except hydrocortisone showed degrees of inhibition, varying from 2 to 170 percent. Indomethacin had a significantly greater effect in the trachea and the proximal halves of the upper and lower lateral bronchioli, while dexamethasone had a greater effect in the distal halves of the lateral bronchioli. All drugs had a similar inhibitory effect in the intrapulmonary axial airway.

82-1322. Kawakami, Y.; Irie, T.; Shida, A.; Yoshikawa, T. **Familial Factors Affecting Arterial Blood Gas Values and Respiratory Chemosensitivity in Chronic Obstructive Pulmonary Disease.** *American Review of Respiratory Disease* 125(4): 420-425, April 1982.

Evidence that there are familial factors involved in determining arterial blood gases and ventilatory response to hypoxia in chronic obstructive pulmonary disease (COPD) is provided from a study of 25 patients with COPD and their 34 sons; 17 patients with silicosis and their 22 sons serving as controls. Smoking habits were compared between patients and sons in terms of age at which smoking was started, cigarettes per day, years of smoking, and the Brinkman index (cigarettes per day multiplied by years of smoking). Smoking habits in patients with COPD did not correlate significantly with those of their sons. However, of the many intrafamilial environmental factors, smoking habits would be the most important, but they are extremely difficult to quantitate. It appears that smoking habits were not the same in patients with COPD and their sons, but such habits could not be ruled out as a possible familial factor because of difficulties in quantitation and the high concordance ratio of smoking habits in patients with COPD (85.3 percent vs. 54.5 percent in patients with silicosis, $p < 0.02$).

82-1323. Klima, H. **Chronische Bronchitis in Wien, Epidemiologische Ergebnisse der "Wiener Gesundheitsstudie 79." [Chronic Bronchitis in Vienna. Epidemiologic Data of the "79 Vienna Health Study."]** *Wiener Medizinische Wochenschrift* 131(21): 529-532, 1981, German.

The incidence of chronic bronchitis (excluding asthmatic bronchitis) and its relationship to other respiratory diseases, to social and occupational risk factors, and to smoking habits are analyzed, using data involving residents of Vienna, Austria. Age and sex distributions of chronic bronchitis are shown. The study reveals that 51 percent of the 2,787 subjects surveyed were smokers or ex-smokers and 33 percent were nonsmokers (16 percent did not answer). The percentage of bronchitic subjects among the smokers of all ages and both sexes was 10 percent vs. 3 percent among the nonsmokers. The incidence of chronic bronchitis among the 25-year-old male smokers was 4.2 percent vs. zero in the same category of nonsmokers; the incidence among 40-year-old male and female smokers was 9.7 and 8.8 percent, respectively, vs. 2.1 and 0.7 percent for the corresponding categories of nonsmokers. Twice as many bronchitic subjects were found among the 60-year-old male and female smokers as among the same categories of nonsmokers. Thus, the study confirmed that smokers are particularly vulnerable to chronic bronchitis.

82-1324. Linn, W. S.; Fischer, D. A.; Medway, D. A.; Anzar, U. T.; Spier, C. E.; Valencia, L. M.; Venet, T. G.; Hackney, J. D. **Short-Term Respiratory Effects of 0.12 ppm Ozone Exposure in Volunteers With Chronic Obstructive Pulmonary Disease.** *American Review of Respiratory Disease* 125(6): 658-663, June 1982.

Smoking did not have a significant effect on the responses to ozone (O_3) exposure among 25 volunteers with mild to moderately severe chronic obstructive pulmonary disease (COPD). All except three of the subjects (8 current smokers, 14 ex-smokers) gave a history of moderate to heavy smoking. The volunteers underwent 1-hr exposures to 0.12 ppm O_3 in purified air with intermittent mild exercise. Their responses were assessed in terms of forced expiratory performance, ear oximetry, and reported symptoms. Control studies consisted of similar exposures to purified air alone. Control studies were separated from O_3 exposures by 1 month, and the order was randomized. All studies took place in a controlled-environment chamber, and were preceded by approximately 1 hour of rest in a purified-air environment. Smoking status apparently had no effect on the response to ozone. Ex-smokers, however, had a generally lower arterial hemoglobin oxygen saturation than present smokers or nonsmokers. (Auth. Abs. Mod.)

82–1325. Love, G. J.; Lan, S.-P.; Shy, C. M.; Riggan, W. B. **Acute Respiratory Illness in Families Exposed to Nitrogen Dioxide Ambient Air Pollution in Chattanooga, Tennessee.** *Archives of Environmental Health* 37(2): 75-80, March-April 1982.

The incidence of acute respiratory illness in families in Chattanooga, Tennessee, was studied in 1972 and 1973 to determine its relationship with ambient concentrations of nitrogen dioxide. Illness data were collected by telephone at 2-week intervals and illness rates per 100 person-weeks of observation were contrasted with air pollution concentrations measured no more than 3.2 km from the home. Other factors studied include smoking in the home, crowding, use of gas stoves, and educational level of the parents. In 1972, higher rates of respiratory illness continued to occur in the high pollution area. These were associated with current higher short-term concentrations of nitrogen dioxide even though the long-term mean concentrations of the pollutant were little higher than those in the low pollution area. Smoking among adults or parental smoking in the home showed an inconsistent relationship to illness rates. It was not possible to attribute the excesses in illness to specific pollutants or to specific exposure periods. (Auth. Abs. Mod.)

82–1326. Love, R. G.; Miller, B. G. **Longitudinal Study of Lung Function in Coal-Miners.** *Thorax* 37(3): 193-197, March 1982.

Longitudinal loss of lung function in 1,677 coal miners from five British collieries has been calculated. The results of serial cross-sectional epidemiological surveys were compared with current individual dust exposures and partially estimated cumulative exposures. Loss of forced expired volume in 1 second (FEV_1) over approximately 11 years was found to increase with previous cumulative dust exposure after allowing for the effects of age, height, smoking, and overall colliery differences. The magnitude of the effect on FEV_1 due to dust exposure appeared to be about one-third of the loss attributable to smoking. This relationship was found to hold with concurrent dust exposure only when colliery differences were ignored. (Auth. Abs. Mod.)

82–1327. Malo, J. L.; Filiatrault, S.; Martin, R. R. **Bronchial Responsiveness to Inhaled Methacholine in Young Asymptomatic Smokers.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 52(6): 1464-1470, June 1982.

Methacholine inhalation challenges were performed in 10 young smokers who denied having respiratory symptoms and in 10 nonsmokers of the same age. There were five males and five females in each group. The resulting bronchoconstriction was evaluated with specific lung conductance, maximum partial expiratory flows assessed at 40 percent vital capacity breathing both air and a helium-oxygen mixture, and with maximum complete flows breathing helium/oxygen. Dose—response curves were studied for threshold concentration, where values depart by more than two standard deviations from the baseline, and the provocative concentration causing a fixed fall in a test parameter. Smokers differed significantly from nonsmokers in threshold and provocative concentrations in all parameters except lung conductance and forced expiratory volume. Induced bronchoconstriction and small airways obstruction was similar in males and females. Density dependence of maximum partial flows after helium/oxygen breathing tended to increase in nonsmokers and decrease in smokers: this may indicate a more peripheral site of the reaction in smokers. (Auth. Abs. Mod.)

82–1328. McCullagh, S. F. **Respiratory Morbidity in Waterside Workers. (Letter).** *Medical Journal of Australia* 2(9): 453-454, October 31, 1981.

A previous study (*Medical Journal of Australia* 2(3): 139-141, August 8, 1981) citing asbestos exposure as the cause of increased respiratory morbidity among Brisbane, Australia, waterside workers is criticized. In the study, the waterside workers, 80 percent of whom are smokers, were compared with a group of nonsmoking controls. It is noted that the smoking habit is much more important in determining the prevalence of pleural plaques than asbestos exposure, and duration of employment on the wharves did not correlate with respiratory symptoms. Thus, the study did not convincingly show that any occupationally attributable respiratory morbidity from asbestos is occurring in the waterside workers: smoking, rather than asbestos, should be cited as the crucial risk factor. See also 82-0709.

82–1329. Miralles Lozano, F.; Fernandez Sanchez, J.; Valencia Rodriguez, A.; Carpintero Avellaneda, J. L.; Merino Berdugo, J. **Pathophysiological Disturbances in Bagassosis and Bagasse Workers.** *Clinical Respiratory Physiology* 18(4): 623-632, 1982.

Two groups of bagasse workers were studied and compared: 5 subjects (4 smokers) who presented with bagassosis and 26 workers (18 smokers) conventionally considered as normal. Significant differences were found between both groups in the blood leucocyte count, serum electrophoretogram gamma-fraction, serum IgG, and, even more important, in some pulmonary function tests when carried out after exposure to daily work conditions: forced vital capacity (FVC), forced expiratory flow during middle half of VC ($FEF_{25\text{-}75}$), peak expiratory flow (PEF), and maximum expiratory flow at 50 percent of FVC ($Vmax_{50}$). When bagassosis cases were removed from the workplace, the ciliary X-ray pattern disappeared along with the symptoms within 1 to 2 months. Indications of small airway disease found 1 year later might be ascribed to smoking except in the case of the single nonsmoker. The individual prognosis is favorable. (Auth. Abs. Mod.)

82–1330. Oxhoj, H.; Andreasen, H.; Henius, U. M. **Respiratory Symptoms and Ventilatory Lung Function in Machine Shop Workers Exposed to Coolant-Lubricants.** *European Journal of Respiratory Diseases* 63(Supplement 118): 85-89, 1982.

Smoking habits and respiratory symptoms were recorded in 375 machine shop workers, and ventilatory lung function measured in 295 workers exposed to four different types of coolant-lubricants (CL): mineral oil, emulsions, semisynthetic, and synthetic CL. The smoking habits of the workers did not differ significantly between the four exposure groups. The occupational environment was analyzed with respect to CL and various contaminant concentrations. Subjects were divided into nonsmokers, ex-smokers, and present smokers. Data revealed a significant association between smoking habits (regardless of occupational exposure), oil aerosol concentrations (regardless of smoking habit), and prevalences of chronic cough and phlegm, suggesting a dose—effect relationship. After adjustment for smoking habits, prevalences of respiratory symptoms and lung function values were not different in the four exposure groups. (Auth. Abs. Mod.)

82–1331. Posner, V.; Posner, B. **Organizing a General-Practitioner Therapeutic Trial.** *Practitioner* 226(1367): 975-977, May 1982.

The standard inhaler salbutamol (Ventolin) was compared to an inhaler of ipratropium bromide (Atrovent) in relief of airways obstruction. The 28 subjects were 16 to 60 years of age (mean age 48), and showed at least 20 percent improvement in peak flow after one inhalation of salbutamol: 9 were smokers, 19 were nonsmokers; 18 were asthmatics, and 10 were bronchitics. A mini peak flow meter was provided to each of the 28 patients for use at home, and data were also taken during weekly clinic visits. There was no significant difference between the two drugs, except that ipratropium bromide was superior for smokers and during illness of short duration. Salbutamol appeared to be more appropriate for asthmatics, nonsmokers, and those who had been incapacitated for many years. The human elements of therapeutic trials and recommendations for accommodating them are noted.

82–1332. Rai, U. C.; Nancy, N. R. **Effect of Snuff on Pulmonary Function Tests.** *Indian Journal of Chest Diseases and Allied Sciences* 22(3): 147-151, July-September 1980.

Because snuff is not inhaled deeply, it affects only the upper larger airway and not the flow rates which reflect the condition of small airways. These conclusions were reached following a series of pulmonary function measurements from 50 hospital employees (25 snuff users and 25 controls) of similar socioeconomic status. Forced expiratory spirograms and peak expiratory flows (PEF) were obtained from the 25- to 45-year-old subjects. Forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV_1), forced vital capacity (FEV_1/FVC percent), forced expiratory flows (FEF) between 80 and 70 percent, FEF between 55 and 45 percent, FEF between 30 and 20 percent FVC, maximal expiratory flow, forced mid-expiratory flow, and forced expiratory time were determined. PEF, FEF, FVC, and FEV_1 were lower in snuff users than in controls, but the difference is statistically significant ($p < 0.05$) only for PEF. The body weight of snuff users is significantly lower than that of controls even though the age and height are comparable in both groups. This observation may be due to the specific effect of snuff.

82–1333. Rees, P. J.; Higenbottam, T.; Clark, T.J.H. **Responses to Cigarettes of Various Tar Contents.** *British Journal of Diseases of the Chest* 76(2): 143-146, April 1982.

When airway resistance measurements are made 3 to 10 min after smokers have smoked a cigarette in their usual fashion, significant bronchoconstriction has been found to be uncommon. Specific conductance was measured before and within 10 min of smoking a cigarette in 36 smokers on 210 occasions. Five types of cigarettes were used including the equivalent of a 1956 unfiltered cigarette. Cigarettes were smoked in the smokers' usual fashion. Significant decreases in specific conductance occurred on only 19 occasions in 13 subjects and were most frequent with the cigarette with the highest tar content currently available. Examination of the overall results shows the only significant change to be an increase in specific conductance in the control, nonsmoking period. It is concluded that irritant qualities of tobacco smoke increase with tar content but airway narrowing lasting up to 10 minutes is an uncommon event when cigarettes are smoked normally. Most smokers adapt to or avoid any sustained acute airway response to tobacco smoke whatever its tar content. (Auth. Abs. Mod.)

82–1334. Schenker, M. B.; Samet, J. M.; Speizer, F. E. **Effect of Cigarette Tar Content and Smoking Habits on Respiratory Symptoms in Women.** *American Review of Respiratory Disease* 125(6): 684-690, June 1982.

Smoking habits of 5,686 adult women of rural Pennsylvania were surveyed to determine the effects of tar content and inhalation on respiratory symptoms. Only women were questioned to avoid confounding occupational factors from coal mining. Among current smokers ($n = 1,424$), the number of cigarettes smoked per day was consistently the strongest risk factor for chronic cough, chronic phlegm, wheeze, and dyspnea. The odds ratios for these symptoms in a smoker of 25 or more cigarettes per day vs. a smoker of 1-14 cigarettes per day were 4.74, 4.21, 2.05, and 3.71, respectively. Ex-smokers ($n = 648$) showed risks between those of current and never smokers. Cigarette tar content was a significant risk factor for chronic cough ($p = 0.005$) and of borderline significance for chronic phlegm ($p = 0.077$). The effect of cigarette tar was linear and independent of cigarettes smoked per day. Depth of inhalation was a significant risk factor for chronic cough

($p=0.029$) and for chronic phlegm ($p=0.038$). However, neither tar content nor degree of inhalation was significantly related to wheeze or dyspnea. (Auth. Abs. Mod.)

82–1335. Schneider, W. D.; Rebohle, E. **Zur Fruhdiagnostik mittels Fluss-Volumen-Kurve bei Inhalativ Exponierten Werktaetigen. [Early Diagnosis by Means of Flow-Volume Curves in the Inhalation-Exposed Workers.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 157(3): 291-296, June 1981, German.

In the search for a better diagnostic method for pulmonary function disorders, the parameters of the flow-volume curves and the spirometric parameters were determined in 432 electric welders exposed to welding fumes and in 420 unexposed controls from the same industry. The smoking habits of the two groups were not significantly different. Within each group, the following significant differences between smokers and nonsmokers were found: the forced expiratory volume in 1 sec (FEV_1), with lower values for smokers regardless of age; the respiratory quotient (V_3/V_1) only in welders under 45 years of age; and the forced expiratory flow when 50 percent and when 75 percent of forced vital capacity have been expired ($Vmax_{50\ percent}$, $Vmax_{75\ percent}$), in workers over 45. All cited parameters except vital capacity (VC) are shown to be significantly influenced by the smoking habit, with FEV_1 and $Vmax_{75\ percent}$ values having the highest degree of reliability and significance. All parameters except the VC of the smokers exhibit the same trend. Thus, the results do not support the use of flow-volume curves for screening non-preselected groups of inhalation-exposed workers.

82–1336. Searle, C. E. **Smoking and FOREST. (Letter).** *New Scientist* 88(1231): 734-735, December 11, 1980.

The conclusion of an earlier report (*New Scientist* 88(1221): 10-13, October 2, 1980) that passive smoking is harmful to the health of nonsmokers is supported and a letter (*New Scientist* 88(1223): 182, October 16, 1980) from FOREST (The Freedom Organization for the Right to Enjoy Smoking Tobacco), which found the report "totally unacceptable," is criticized. To support the original conclusion, reference is made to a study of the lung function in 2,100 middle-aged subjects (*New England Journal of Medicine* 302(13): 720-723, March 27, 1980), which found statistically significant differences between nonsmokers who had worked in clean environments and nonsmokers who had spent over 20 years in jobs where they were exposed to tobacco smoke. Lung function in the second group had fallen to levels similar to those in groups of noninhaling smokers and light smokers (1-10 cigarettes/day). See also 82-1409, 82-1394, and 80-1190.

82–1337. Sprince, H.; Smith, G. G.; Parker, C. M.; Rinehimer, D. A. **Protection Against Cyanide Lethality in Rats by L-Ascorbic Acid and Dehydroascorbic Acid.** *Nutrition Reports International* 25(3): 463-470, March 1982.

L-ascorbic acid (LAA) and dehydroascorbic acid (DHA) have been found to protect rats against lethal doses of cyanide. Cyanide is known to be a respiratory toxicant of cigarette smoke and has been associated with visual disturbances ("tobacco-alcohol amblyopia") and tropical ataxic dietary neuropathy due to cassava root ingestion. Pretreatment of male albino rats with high oral doses of LAA or DHA gave good protection (90 to 100 percent survivors) against lethal (LD 90) oral doses of sodium cyanide. At lower doses, DHA was the better protectant. Pretreatment with high intraperitoneal doses of DHA also gave good protection (80 to 90 percent survivors) against oral cyanide, but intraperitoneal LAA was only weakly protective if at all. Thus, protection by DHA seems to be systemic in nature. The greater effectivity of DHA than LAA as a protectant would appear to support the hypothesis of cyanide detoxification by carbonyl groups via nontoxic cyanohydrin formation. It is cautioned, however, that further animal experimentation is necessary before these findings can be considered for human use. (Auth. Abs. Mod.)

82–1338. Stovin, P.G.I.; Partridge, P. **Pulmonary Asbestos and Dust Content in East Anglia.** *Thorax* 37(3): 185-192, March 1982.

In the general population of East Anglia nonoccupational exposure to asbestos is not occurring as a continuing background experience but probably represents occasional short episode(s) of exposure, and these episodes result in fiber counts up to about 30,000 per gram of dried lung. This conclusion resulted from asbestos fiber and dust content measurements of samples from 96 surgically excised lungs, 42 necropsies on patients with lung cancer, 11 necropsies on patients with nonpulmonary malignancies, and 59 necropsies on patients without any malignant disease. The patients' ages ranged from 45 to 74 years at the time of study. None of the patients had asbestosis. The distribution of fibers and dust content of the lungs showed a log-normal distribution. There was no significant difference in fiber counts or dust content between men and women, and between lung cancer and noncancer patients. The only group with an association with a high asbestos fiber count was four necropsy cases of pleural mesothelioma. There was no significant relationship between asbestos fiber count and dust content of the lung. All but three of the surgical cases were smokers. The smokers averaged 57 pack-years (±SD 40) and there was no significant difference in the lung dust content between the light (25 or less pack-years) and the heavy smokers (more than 98 pack-years). Unfortunately, the smoking and occupational histories were incomplete for the necropsy patients, so no meaningful analysis was possible. The present data suggest that asbestos fiber counts below 100,000 per gram of dried lung are not related to specific asbestos disease, although in the surgical cases who were closely questioned on their residential and occupational histories most of those with fiber counts above 30,000 per gram dried lung had had occasions of definite or very likely asbestos exposure. (Auth. Abs. Mod.)

82–1339. Taylor, B.; Golding, J.; Wadsworth, J.; Butler, N. **Breast-Feeding, Bronchitis, and Admissions for Lower-Respiratory Illness and Gastroenteritis During the First Five Years.** *Lancet* 1(8283): 1227-1229, May 29, 1982.

Maternal smoking was found to be one of the risk factors for bronchitis and gastroenteritis during a child's first 5 years of life in a national British study of 13,135 children. Breastfeeding was found to have no significant association with rates of bronchitis or hospitalization for lower respiratory illness after allowance was made for influences such as maternal smoking and socioeconomic status which are associated with both lower respiratory illness and likelihood of breastfeeding. Admissions to a hospital for gastroenteritis in the first year were marginally less likely in breastfed infants. Socioeconomic status and birth weight were also found to be more significant than breastfeeding on the incidence of bronchitis and gastroenteritis. (Auth. Abs. Mod.)

82–1340. Villiger, B.; Heymach, G. J.; Broekelmann, T. J.; Kelley, D. G.; Kuhn, C.; McDonald, J. A. **Human Bronchoalveolar Fibronectin: Quantification of an Opsonic Protein and Its Synthesis by Alveolar Macrophages.** *Chest* 81(5, Supplement): 45S-47S, May 1982.

To compare the fibronectin (FN) concentration in the lungs of smokers and nonsmokers, bronchoalveolar lavage fluid (BAL) and pulmonary alveolar macrophages (PAM) were obtained from 29 subjects, 16 smokers and 13 nonsmokers aged 21 to 37 years. Fibronectin is an opsonic protein, critical for the phagocytosis of particulates and microorganisms in the terminal airways by PAMs. The albumin-to-protein ratio in BAL was equal in the smokers and nonsmokers, though 5 times more PAM was recovered in smokers. Thus the mean FN content of BAL fluid was significantly higher in the smokers. Also, a higher FN to albumin ratio in smokers' BAL fluid than in plasma was found. This, taken with other research results, indicated that PAM is a likely source of FN in BAL fluid, and that FN is not a membrane-bound receptor in PAM, but appears at the cell membrane binding sites for gelatin-coated particles. Thus PAM-FN may serve as a specific endogenous opsonin for uptake of denatured collagen, fibrin, or staphylococci in human lungs.

82–1341. Wallis, T. W.; Rogers, W. R.; Johanson, W. G., Jr. **Effects of Acute and Chronic Exposure to Nicotine Aerosol on Bronchial Reactivity to Inhaled Methacholine.** *Journal of Applied Physiology* 52(4): 1071-1076, April 1982.

Cigarette-smoking baboons demonstrate reduced bronchial reactivity to inhaled methacholine compared with sham-smoking controls after 6 pack-years of smoking. To evaluate the role of nicotine in blunting airway reactivity, the effects of both acute and chronic nicotine inhalation on reactivity to methacholine in baboons was studied. Inhalation of 2 mg of nicotine had no acute effect on lung function but blunted bronchial reactivity in highly reactive animals. This effect was not diminished after daily inhalation of nicotine for 90 days. Marked interindividual differences in bronchial reactivity to methacholine were not abolished by propanolol, suggesting that factors other than β-adrenergic tone account for this intersubject variability. (Auth. Abs.)

82–1342. Wettengel, R. **Praevention, Langzeittherapie und Rehabilitation. [Prevention, Long-Term Therapy and Rehabilitation.]** *Verhandlungen der Deutschen Gesellschaft fuer Innere Medizin* 87: 274-280, April 26-30, 1981, German.

Prevention of smoking initiation may be the best way to reduce the incidence of bronchitis, since smoking is the highest risk factor for bronchitis. Numerous studies have shown the cause and effect relationship between smoking and bronchitis, in which the primary symptom is the loss of respiratory function. Results of two epidemiologic studies are cited to show the close correlation between the loss of respiratory function expressed by the forced expiratory volume in 1 second and smoking history (length of habit and average cigarette consumption). In spite of such clinical evidence, the problems of initiation of smoking among children and adolescents, and of cessation of smoking in adults, have not been solved. Progress can be expected only if teachers, psychologists, politicians, and physicians cooperate in the effort. Studies of long-term therapy for bronchitics, particularly continuous oxygen therapy and rehabilitation, are reviewed.

CARDIOVASCULAR DISEASES

82–1343. Angelkort, B.; Kiesewetter, H. **Influence of Risk Factors and Coagulation Phenomena on the Fluidity of Blood in Chronic Arterial Occlusive Disease.** *Scandinavian Journal of Clinical and Laboratory Investigation* 41(Supplement 156): 185-188, 1981.

Results from a study of erythrocyte fluidity in patients with chronic arterial occlusive disease, healthy normals, heavy smokers, and groups with isolated risk factors and coagulation phenomena suggest that heavy smoking can adversely affect flow rates. In chronic arterial occlusive disease (CAOD) the fluidity of erythrocytes is markedly reduced. Erythrocyte fluidity was assessed by flow rates through a 5-μm pore filter system in simulation of microcirculatory conditions. Risk factors such as heavy smoking, hypercholesterolemia, and hypertriglyceridemia, as well as hyperfibrinogenemia, resulted in flow rates similar to those in patients with CAOD. (Auth. Abs. Mod.)

82–1344. Arkwright, P. D.; Beilin, L. J.; Rouse, I.; Armstrong, B. K.; Vandongen, R. **Effects of Alcohol Use and Other Aspects of Lifestyle on Blood Pressure Levels and Prevalence of Hypertension in a Working Population.** *Circulation* 66(1): 60-66, July 1982.

In an examination of the association between alcohol consumption and blood pressure, cigarette smokers, overall, had slightly lower diastolic pressure than nonsmokers despite a higher heart rate. An employed population of 491 males, aged 20 to 45 years, was studied by questionnaire and measurements of blood pressure, heart rate, and body size. The effect of smoking on diastolic pressure was present at all levels of alcohol consumption, and was more marked in teetotalers and heavy drinkers. The effect of smoking on systolic pressures was not significant, although for three of the four levels of alcohol consumption analyzed, the mean systolic pressure was lower in smokers than nonsmokers. Other variables examined included age, exercise, education, and coffee and tea consumption. It was found that average weekly alcohol consumption correlated with systolic pressure but not diastolic pressure, and that the effect of alcohol on systolic blood pressure was independent of the effects of age, obesity, cigarette smoking, and physical activity.

82–1345. Barboriak, J. J.; Anderson, A. J.; Hoffmann, R. G. **Smoking, Alcohol and Coronary Artery Occlusion.** *Atherosclerosis* 43(2, 3): 277-282, June 1982.

Smoking showed an exposure-related enhancing effect on the extent of coronary artery occlusion in a study of 2,989 men undergoing a diagnostic coronary arteriography. Patients were divided into nonsmokers (not presently smoking), moderate smokers (smokers of cigars, pipes, and ≤ 20 cigarettes daily for <20 years), and heavy smokers (>20 cigarettes daily for 10 years or more, or 20 cigarettes daily for $>$ 20 years), and into subgroups based on drinking habits. Alcohol consumption demonstrated an attenuating dose-related effect, offsetting the increased coronary occlusion associated with smoking. It is suggested that the effect of smoking and other risk factors on the extent of coronary artery occlusion and prevalence of coronary heart disease may be underestimated if one does not consider the attenuating action of simultaneous alcohol consumption. (Auth. Abs. Mod.)

82–1346. Barrett-Connor, E.; Suarez, L. **A Community Study of Alcohol and Other Factors Associated With the Distribution of High Density Lipoprotein Cholesterol in Older vs. Younger Men.** *American Journal of Epidemiology* 115(6): 888-893, June 1982.

To determine whether factors that correlate with high density lipoprotein cholesterol (HDL-C) are similar in younger and older men, 311 men from a community sample were dichotomized into younger (30-59) and older (60-89) age groups. HDL-C is independently and inversely correlated with the risk of coronary artery disease. Older men had significantly higher HDL-C levels and were leaner and less likely to smoke cigarettes, but did not differ significantly from younger men with regard to exercise or reported alcohol consumption. In the younger men, only age, alcohol consumption, and serum aspartate aminotransferase (AST) were positively and significantly correlated with HDL-C. Obesity and cigarette smoking were inversely related to HDL-C levels, but the coefficients were not statistically significant. In older men, only alcohol consumption and AST were significant correlates of HDL-C. Age, obesity, and cigarette smoking were not correlated with HDL-C levels after age 60. Pairwise correlations for the selected attributes show that in younger men, alcohol consumption was positively and significantly associated with age, cigarette smoking, and AST, and negatively and significantly associated with exercise. In older men, alcohol and AST showed significant positive correlation; both alcohol and cigarette consumption were inversely related to age. (Auth. Abs. Mod.)

82–1347. Benowitz, N. L.; Jacob, P., III; Jones, R. T.; Rosenberg, J. **Interindividual Variability in the Metabolism and Cardiovascular Effects of Nicotine in Man.** *Journal of Pharmacology and Experimental Therapeutics* 221(2): 368-372, May 1982.

The relationship between plasma concentration of nicotine and its cardiovascular effects was studied in 14 healthy young men. The mean metabolic clearance was 1089±279 ml/min; mean renal clearance (in acid urine) was 203±58 ml/min, averaging 15.7 percent of total clearance. The steady-state volume of distribution was large (mean 183±49 l). Nicotine infusion increased the heart rate and blood pressure and decreased fingertip skin temperature in a manner similar to cigarette smoking. Heart rate increased promptly, reaching a maximum within 5 or 10 min, despite progressively increasing plasma nicotine concentrations. The sensitivity of heart rate acceleration to low concentrations of nicotine and rapid development of tolerance to higher concentrations suggests that cardiac responses may not differ when smoking low- vs. high-nicotine cigarettes. The decline in skin temperature, however, was related to blood nicotine concentration and showed no evidence of tolerance. (Auth. Abs. Mod.)

82–1348. Bidet, J. M.; Gilbert, M.; Cheynel, J.; Duchene-Marullaz, J.; Lemarie, J. C. **Red Blood Cell Filterability in Non-Diabetic Patients With Chronic Occlusive Arterial Disease.** *Scandinavian Journal of Clinical and Laboratory Investigation* 41(Supplement 156): 189-191, 1981.

In testing 150 nondiabetic patients with stage II chronic occlusive arterial disease (COAD) for red blood cell filterability (RBF), smoking was associated with a greater decrease of filtration time, without relation to a variation of mean corpuscular volume usually observed among smokers. Glycemia, uricemia, plasma cholesterol and triglycerides, fibrinogen, hematocrit, white blood cells (granulocytes and lymphocytes) count, and mean corpuscular volume were also measured. Thirty healthy people, tested for the same biological parameters, were used as controls. RBF was performed, using the Reid and Dormandy technique, with the same batch of filters for patients and controls. Filtration time was expressed in $s.ml^{-1}$. Values obtained in patients (41.967 ± 0.889) were significantly different ($p<0.005$) from controls (32.1 ± 1.329). RBF alteration was correlated ($p<0.05$) with white blood cells count, hematocrit, and plasma fibrinogen values. Although no correlation was observed with other biological parameters, a good correlation ($p<0.01$) appeared with smoking among the patients analyzed; 46 percent of the smokers compared with 25 percent of the nonsmokers had a flow filtration rate less than $22.5\ \mu l.s^{-1}$. (Auth. Abs. Mod.)

82–1349. Brooks, W. W.; Bing, O.H.L.; Huber, G. L.; Abelmann, W. H. **Contractile Performance of Rat Myocardium After Chronic Tobacco Smoke Inhalation.** *Archives of Environmental Health* 37(2): 93-97, March-April 1982.

The mechanical performance of isolated left ventricular muscle preparations from rats exposed to smoke from Kentucky Reference cigarettes was examined for possible chronic effects. Eight rats were subjected to smoke for periods of 10 min/hr for 5 hr/day for 180 days. Nineteen additional rats served as either sham-smoked controls or weight-matched, food-deprived controls. Rats exposed to tobacco smoke had a significant diminution in body and left ventricular weight compared to sham-smoked controls. When compared to food-deprived rats, no differences in weights were observed. Contraction mechanics were measured for each muscle at the peak of its length tension curve. No significant difference was found between smoke-exposed rats and control animals with respect to contractile performance under oxygenated conditions, muscle performance during 60 min of hypoxia or subsequent reoxygenation, or sensitivity of mechanical performance to isoproterenol. Thus, chronic cigarette smoke exposure did not alter the intrinsic mechanical performance of isolated rat ventricular muscle. (Auth. Abs.)

82–1350. Cleophas, T.J.M.; Fennis, J.F.M.; van't Laar, A. **Finger Temperature After a Finger-Cooling Test: Influence of Air Temperature and Smoking.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 52(5): 1167-1171, May 1982.

To examine smoking's effect on finger temperature recovery, the skin temperature of one finger was measured before and after immersion of both hands in 16°C water for 5 minutes. The subjects were in a room temperature ($T\alpha$) of either 20 or 24°C. At 24°C, 23 of 25 nonsmokers (92 percent) had finger rewarming to above 24°C within 12 minutes after immersion in cold water; at $T\alpha$ 20°C, only 1 of 12 (8 percent) had similar rewarming. Among 12 smokers, only 4 (33 percent) rewarmed above 24°C ($T\alpha$ 24°C) following an hour's abstinence from smoking, though 8 (67 percent) did so after a 24-hour abstinence. Only 2 of these 8, however, did so in a retest 10 minutes after smoking a cigarette. The smokers were not tested at $T\alpha$ 20°C. In testing finger temperature recovery for Raynaud's syndrome, it is recommended that smoking and ambient temperature be accounted for. (Auth. Abs. Mod.)

82–1351. Cook, D. G.; Bartley, M. J.; Cummins, R. O.; Shaper, A. G. **Health of Unemployed Middle-Aged Men in Great Britain.** *Lancet* 1(8284): 1290-1294, June 5, 1982.

Current cigarette smoking and heavy drinking were more common among the unemployed than the employed in an examination of the frequencies of several factors, including major physical disease, in men enrolled in the British Regional Heart Study (BRHS). The BRHS is a prospective study of cardiovascular disease in middle-aged men selected at random from general practices in 24 towns. The unemployed group was subdivided into those who said they were unemployed because of ill health and those who regarded their unemployment as not due to illness. The ill unemployed reported a much higher rate of doctor-diagnosed illnesses than the not-ill unemployed or the employed. The frequencies of bronchitis, obstructive lung disease, and ischemic heart disease were higher in the unemployed than the employed, with the highest rates in the ill unemployed. The frequency of hypertension was the same in employed and unemployed men. After standardizing for age, social class, and town of residence there were no significant differences in numbers of heavy drinkers between the employment categories. The differences in current cigarette smoking remained, though they were smaller. Use of tranquillizers was three to four times more common in the ill unemployed than in the not-ill unemployed or the employed. The unemployed had far more chronic physical illnesses than the employed, whether or not the employed men regarded themselves as ill. (Auth. Abs. Mod.)

82–1352. Cooper, R.; Schatzkin, A. **Recent Trends in Coronary Risk Factors in the USSR.** *American Journal of Public Health* 72(5): 431-440, May 1982.

The distribution of coronary risk factors, and the secular trends in diet and cigarette consumption, provide a potential explanation for the increased incidence of coronary heart disease in the Soviet Union over the last 15 years. The animal fat content of the Soviet diet has been steadily enriched since the 1950s and cigarette production increased 72 percent from 1959 to 1980. Per capita consumption in 1974 was approximately 2,600 cigarettes, a smoking rate similar to Europe. A recent survey in Moscow revealed 44.2 percent of men and 10.1 percent of women over age 16 smoked. In the 20 to 29 age group, the numbers were 60.4 and 16.8, respectively. Consumption rate in the forties' age group was 18.1 cigarettes per day for men, and 14.7 for women. Smoking rates also followed a clear social class gradient; 64.8 percent in those with only a primary education, 59.7 percent in the high-school-educated, and 37.2

percent in those with higher education. Cigarette consumption, a high-fat diet, uncontrolled hypertension, and high alcohol consumption rates have resulted in Soviet mortality patterns increasingly similar to patterns in western capitalist countries. The post-Stalin orientation of the Soviet economy toward a policy of motivating the workforce primarily through the provision of consumer goods, in a pattern comparable to western capitalist countries, appears to have laid the basis for these developments. (Auth. Abs. Mod.)

82–1353. Freestone, S.; Ramsay, L. E.; Houben, H.; Thien, Th.; Van't Laar, A. **Haemodynamic Effects of Cigarette Smoking During β-Adrenoceptor Blockade. (Letters).** *British Journal of Clinical Pharmacology* 13(2): 258-259, 1982.

The conclusion that blood pressure changes induced by smoking are not altered with treatment by β-adrenoceptor blockade (*British Journal of Clinical Pharmacology* 12(1): 67-72, 1981) is criticized. It is argued that high doses (100-600 mg) of metoprolol were used, and that cardioselectivity is a relative property which tends to be lost at such high plasma levels. Thus no realistic conclusion could have been made about the hemodynamic effects of smoking. The authors reply, and agree that cardioselectivity is lost at high dose levels. However, regardless of dose, even the patients with the largest increase in mean arterial pressure during metoprolol treatment showed a greater pressor response when adrenaline was infused. When the patients were divided according to dose no correlation was found between dose, plasma level of the drug, and smoking-induced changes in blood pressure and heart rate. See also 82-0122.

82–1354. Glover, M. U.; Kuber, M. T.; Warren, S. E.; Vieweg, W.V.R. **Myocardial Infarction Before Age 36: Risk Factor and Arteriographic Analysis.** *American Journal of Cardiology* 49(7): 1600-1603, May 1982.

Myocardial infarction before age 36 is a disease of men who smoke and who often have a family history of premature coronary artery disease. In this study, 120 consecutive patients 35 years of age or younger underwent coronary arteriography after clinical myocardial infarction. Ninety-two percent were men. Four distinct subgroups were identified: 94 patients (78 percent) had significant coronary artery disease (greater than 50 percent diameter narrowing of at least one major coronary artery), 20 (17 percent) had normal coronary arteries, 5 (4 percent) had major coronary arterial anomalies, and 1 patient had coronary arteritis. Of the patients with obstructive coronary disease, risk factors were smoking in 89 percent, positive family history of coronary artery disease in 48 percent, hypertension in 21 percent, and a history of lipid abnormality in 20 percent. Risk factors were distinctly less frequent in the groups without coronary atherosclerosis. (Auth. Abs. Mod.)

82–1355. Hermann, T.; Vasselon, C.; Geyssant, A.; Healy, J. C. **RBC Filterability, Oxygen Saturation, ATP Intracellular Stock, and Cerebral Microcirculation.** *Scandinavian Journal of Clinical and Laboratory Investigation* 41(Supplement 156): 213-216, 1981.

The filterability of erythrocytes through Nucleopore filters of 5μm was studied with a new device (Hemorheometer SPO1) which produces a reliable and reproducible index of filterability (IF), depending on only the red blood cell deformability. The results showed an alteration of red blood cell deformability in some pathological circumstances such as smoking, and that deformability depends on the nature of hemoglobin, the ATP intracellular stock, and the saturation of hemoglobin by oxygen. Measurements carried out on 30 smokers showed an increase of filterability index ($x=15.1$, $SD=8.8$). The value of filterability by normal subjects ($n=53$) was 9.7 ($SD=3.37$). The cerebral microcirculatory consequences of alterations of red blood cell deformability have been studied in vitro and in vivo. The results showed an increase of cerebral ascensional time and mean transit time of the cineangioscintigraphy and a decrease of regional cerebral blood flow in patients presenting with a decrease of whole blood filterability flow. (Auth. Abs. Mod.)

82–1356. Hjermann, I.; Holme, I.; Leren, P. **Cholesterol, Smoking, and the Oslo Study. (Letter).** *Lancet* 1(8279): 1015, May 1, 1982.

In reply to a criticism (*Lancet* 1(8270): 503-504, February 27, 1982) of an analysis of the effect of antismoking and diet on acute myocardial infarction (AMI) incidence in the Oslo study (*Lancet* 2(8259): 1303-1310, December 12, 1981), it is conceded that the numbers were small and cause-and-effect results should be interpreted with care. Available computer programs precipitated the change factors used as baseline variables. However, the changes occurred rapidly, persisted, and were monitored individually throughout the study. Thus, it is believed that the results were fairly correct for the lipid factor although more caution should be exercised when interpreting the rather negative conclusion about cigarette smoking. A group-specific favorable psychological effect on the intervention group was not statistically associated in the difference in AMI incidence. The probability of a type II error is great because of the small differences (17 cases) in AMI incidence; however, changes in serum cholesterol explained approximately 10 of the 17 cases prevented and cigarette smoking, at best, 4 cases. Thus, little is left over for a psychological effect. See also 82-0751, 82-0454.

82–1357. Lancet. **Coronary Disease and Multiple Risk Factor Intervention.** *Lancet* 1(8286): 1395, June 19, 1982.

Multifactorial intervention trials aimed at coronary heart disease (CHD) prevention may only add confusion to the field. In the Oslo Heart Study, reduction of CHD events was associated with decreases in both cigarette consumption and serum cholesterol. The MRFIT results, however, revealed an association only with lipid-lowering efforts. CHD incidence rates were greater in those subjects in the bottom quintile of change in cigarette consumption than those in the top quintile. However, since the association with change in smoking rates was not statistically significant, no conclusions could be drawn concerning the contribution of smoking intervention to the trial results. It is noted that high-density lipoprotein (HDL) cholesterol levels were unaffected by either the lipid or smoking interventions, yet were a powerful predictor of CHD incidence in both the control and intervention groups. Future studies should account for HDL cholesterol as a risk factor.

82–1358. Landgraf, H.; Ehrly, A. M. **"Chronic" Cigarette Smoking and Flow Properties of Blood.** *Clinical Hemorheology* 1(3): 241-249, 1981.

The flow properties from blood of 60 healthy young smokers (30 female and 30 male, average age 24.4 years and 25.6 years, respectively) were compared with the flow properties of blood obtained from 60 healthy nonsmokers (30 female, average age 23.7 years and 30 male, average age 27.2 years). Statistically significant differences were found for the hematocrit values in both sexes and for the relative whole blood viscosity in the men's group; relative whole blood viscosity in the women's group and the other rheological parameters such as relative plasma viscosity, RBC filtration rate, and RBC aggregation, showed some discrepancy but not statistically significant differences. The cause for the differences between the results presented and those of other studies might be seen in different measuring devices, including extent and duration of exposure to cigarette smoke, and in the different criteria used for selecting the investigated volunteers. (Auth. Abs. Mod.)

82–1359. Lee, H. P. **Smoking and Blood Pressure Among Adults in Singapore.** *Annals of the Academy of Medicine, Singapore* 9(4): 416-420, October 1980.

In a nationwide survey of blood pressure in Singapore, the prevalence of current smoking was estimated at about 27 percent for all persons aged 20 years and above. There is a male predominance of 5:1, with the peak age among them at 40-49 years. The majority are cigarette smokers, with mean daily consumption at 16.1 for males and 10.7 for females. The study has shown no significant differences in mean systolic and diastolic blood pressures between cigarette smokers and nonsmokers at every age group from 20 to 69 years. There is also no higher prevalence of hypertension among smokers. These findings confirm various other epidemiological studies, especially those in the West where smoking is at a much higher level. Smoking as a risk factor in ischemic heart disease cannot, therefore, be attributed to long-term effects on blood pressure. It is also not likely to be a cause of essential hypertension. (Auth. Abs.) .

82–1360. Lewis, B. **Part II. Ischemic Heart Disease: The Scientific Bases of Prevention.** In: Yu, P. N.; Goodwin, J. F. (Editors). *Progress in Cardiology 10.* Philadelphia, Lea and Febiger, 1981, pp. 21-44.

A review of the epidemiological means of preventing heart disease names the three major risk factors as smoking, hypertension, and lipoprotein abnormalities. In men under 45 years of age, those smoking 25 or more cigarettes per day have a 15-fold excess risk compared with nonsmokers, and heavy smokers have a 4-fold increase in severity of coronary atherosclerosis. Data from varying sources affirm a progressive fall in mortality rate among ex-smokers, and the risk of recurrent myocardial infarction is decreased within 1 year of cessation. However, it appears that smoking, hypertension, and diabetes operate as risk factors only in the presence of sufficiently high levels of cholesterol or low-density lipoprotein. Abnormalities in lipid transport, hypertension, diabetes, and other risk factors are extensively reviewed.

82–1361. Meijler, F. L. **Part III. Prevention of Coronary Heart Disease: A Cardiologist's View.** In: Yu, P. N.; Goodwin, J. F. (Editors). *Progress in Cardiology 10.* Philadelphia, Lea and Febiger, 1981, pp. 44-63.

In a discussion of the unresolved problems in coronary heart disease (CHD), it is noted that despite immense research effort, the occurrence of CHD cannot be predicted, and CHD cannot be predictably prevented. The causes are still unknown. Epidemiological studies are of questionable value to the cardiologist, who deals with the individual. The concept of the risk factor demands that incidence of the disease diminishes upon elimination of the factor; only cigarette smoking seems to satisfy this criterion. Of other traditional risk factors, hypertension does not fit the trend for ischemic heart disease mortality in the United States at all; physical activity fits only the rising curve, serum cholesterol fits only the falling curve, and only smoking fits both. However, these factors do not apply necessarily on the individual level; they are based on epidemiological studies. The cardiologist cannot pinpoint a cause nor identify a future victim. Research should focus on why some smokers do not suffer from CHD, and why some nonsmokers do. Also discussed are nutritional considerations, pathophysiology, subgroups of CHD, atherosclerosis, and the "Galileo phenomenon" operating between epidemiology and cardiology.

82–1362. Neverov, I. V.; Yevtushenko, G. N. **O Roli Kureniya v Vozniknovenii Ishemicheskoy Bolezni Serdtsa. [The Role of Smoking in Development of Ischemic Heart Disease.]** *Sovetskaya Meditsina* (10): 60-64, 1981, Russian.

Soviet epidemiologic studies have revealed a positive correlation between smoking and ischemic heart disease (IHD). However, other Soviet studies have concluded that such a correlation does not exist in 55- to 59-year-old men. In a sample of males aged 40 to 59 in Moscow, 46.5 percent smoked a minimum of 10 cigarettes/day vs. 51.4 percent in a similar sample in Berlin. These studies cite nicotine and other components of tobacco smoke, particularly carbon monoxide and carboxyhemoglobin levels, as the risk factors for IHD. Smoking prevention measures are reviewed. Further research of therapeutic and preventive measures is considered necessary, particularly means of controlling nicotine addiction.

82–1363. Reed, D.; McGee, D.; Cohen, J.; Yano, K.; Syme, S. L.; Feinleib, M. **Acculturation and Coronary Heart Disease Among Japanese Men in Hawaii.** *American Journal of Epidemiology* 115(6): 894-905, June 1982.

A cohort of 4,653 men of Japanese ancestry living in Hawaii, with traditional Japanese social and cultural lifestyles, was studied for the association of the psychosocial factors with coronary heart disease (CHD) (Honolulu Heart Program, 1971-1979). Two of four scales of acculturation were significantly associated with coronary heart disease prevalence independently of 11 other risk factors, but none of the acculturation scales were associated with incidence in multivariate analyses. However, acculturation variables were significantly associated with other CHD risk factors in that the more traditional Japanese men had lower serum cholesterol and uric acid, were less obese, were more physically active, and smoked fewer cigarettes than the more westernized men. Number of cigarettes consumed per day was correlated with incidence of both myocardial infarction and total CHD. However, smoking was not related to overall prevalence of coronary disease. Some of the acculturation scales were indirectly associated with CHD because of confounding associations with other risk factors. (Auth. Abs. Mod.)

82–1364. Renaud, S.; Morazain, R.; Godsey, F.; Dumont, E.; Symington, I. S.; Gillanders, E. M.; O'Brien, J. R. **Platelet Functions in Relation to Diet and Serum Lipids in British Farmers.** *British Heart Journal* 46(5): 562-570, November 1981.

Coagulation and platelet aggregation induced by thrombin, adenosine diphosphate, adrenaline, and collagen were studied in three groups of 20 to 22 middle-aged male farmers. Serum lipids were similar in the three groups. In the west of Scotland group, however, platelet reactivity was significantly greater than in the east of Scotland group. This was associated with a diet higher in saturated fat but also lower in polyunsaturated fat and alcohol. Platelet function in the southern England group correlated with dietary fats and inversely with calcium intake. The east Scotland group had the highest smoking incidence, highest alcohol intake, and lowered platelet function. No relationship was found between platelet function and smoking when 10 hours had lapsed from the last cigarette. However, 10 minutes after smoking, the platelet function tests were much increased. In all of the 63 farmers, the platelet function tests were significantly correlated with the intake of saturated fat regulated by that of calcium, alcohol, and smoking. The dietary effects on platelets appear to be mediated by the fatty acid composition of plasma lipids and of platelet phospholipids, and more dependent upon the dietary factors associated with coronary heart disease such as saturated fats, calcium, alcohol, and tobacco than upon serum lipids. (Auth. Abs. Mod.)

82–1365. Reunanen, A.; Takkunen, H.; Aromaa, A. **Prevalence of Intermittent Claudication and Its Effect on Mortality.** *Acta Medica Scandinavica* 211(4): 249-256, 1982.

The prevalence of symptoms of intermittent claudication and their association with 5-year mortality were examined in a population of 5,738 men and 5,224 women (aged 30 to 59 years) from four geographic areas of Finland. According to a structured interview, 2.1 percent of the men and 1.8 percent of the women reported typical symptoms of intermittent claudication. Claudication was most prevalent in East Finland and among persons with agricultural occupations. The symptoms were more frequent in diabetics and persons with symptoms and signs of coronary heart disease (CHD) than in persons without these diseases. High serum cholesterol and smoking were associated with these symptoms but high blood pressure was not. The risk of death from cardiovascular causes was nearly threefold in men with claudication compared to men without claudication. Symptoms of chest pain and smoking increased significantly the mortality risk of male claudicants. The validity of symptoms was poorer in women than in men and they were also less reliable predictors of death in women. A small part of the effect of claudication on mortality was due to its association with conventional CHD risk factors. However, after adjusting for symptoms and signs at CHD, claudication had no independent effect on mortality in men. (Auth. Abs.)

82–1366. Richard, J. L. **Cigarettes, Inhalation, Leucocytes et Cardiopathies Ischemiques. L'Etude Prospective Parisienne. [Cigarettes, Inhalation, Leukocytes, and Ischemic Heart Disease. A Prospective Study in Paris.]** *Gazette Medicale de France* 89(4): 331-332, 337-339, January 29, 1982, French.

The effects of cigarette smoking, smoke inhalation, and leukocyte count on the incidence of ischemic heart disease have been determined in a 6.5-year followup study. The subjects were 7,434 initially healthy male residents of Paris, aged 43 to 54 years. Smoking habits of the subjects were determined by questionnaire. During the period of study 239 subjects developed an ischemic heart disease including 89 cases of angina pectoris, 100 cases of myocardial infarct, and 50 deaths from coronary heart disease. The study revealed that the risk of severe coronary complications (infarct and sudden death) is increased for all smokers, by a factor of 4.4 for heavy smokers (those who smoke 20 or more cigarettes/day) compared with nonsmokers. For heavy cigarette smokers who inhale, the risk factor is 5.2 and for heavy smokers who inhale and whose leukocyte count exceeds 10,000 per cu mm, the excess risk is 10 or higher.

82–1367. Siess, W.; Lorenz, R.; Roth, P.; Weber, P. C. **Plasma Catecholamines, Platelet Aggregation and Associated Throm-**

boxane Formation After Physical Exercise, Smoking or Norepinephrine Infusion. *Circulation* 66(1): 44-48, July 1982.

Platelet aggregation and thromboxane$_2$ (TXB$_2$) formation were studied during smoking- or exercise-induced sympathoadrenergic activation both before and after propanolol pretreatment in six healthy men. Exercise markedly increased plasma norepinephrine from 128±28 to 998±418 pg/ml, and plasma renin activity from 1.0±0.5 to 4.2±1.8 ng AI/ml-hour. Smoking predominantly increased plasma epinephrine, from 47±25 to 154±76 pg/ml. Propranolol did not consistently influence these variables, but blunted the circulatory response to exercise and smoking. Despite the marked increases of plasma catecholamines after both stimuli with and without β blockade, platelet aggregation stimulated by ADP, 1-epinephrine, collagen, and arachidonic acid and associated TXB$_2$ formation were not enhanced. Moreover, as already suggested by a trend toward reduced aggregability in these settings, plasma norepinephrine levels in the same range (745±368 pg/ml) due to infusion significantly reduced platelet aggregation with low-dose collagen, 1-epinephrine, and ADP. These data do not support a role of endogenous catecholamines in initiating platelet activation and TXB$_2$ formation. (Auth. Abs. Mod.)

82–1368. Singhi, S.; Singhi, P.; Lall, K. B. **Blood Pressure Changes Among Boys Smokers.** *Indian Journal of Pediatrics* 48(392): 337-338, May-June 1981.

Blood pressure was taken in 422 boys, aged 8 to 16 years, of Ajmer, India, to determine the effects of smoking. Of the study group, there were 30 regular smokers, 22 occasional smokers, and 370 nonsmokers. It was found that young smokers have higher systolic blood pressure than nonsmokers. A transient increase was observed immediately following smoking or exercise. An increase in diastolic pressure was also observed, but was not statistically significant. These blood pressure changes may be due to nicotine-caused release of catecholamines, resulting in accelerated heart rate and increased blood pressure, or to persistent high carboxyhemoglobin levels, and increased hematocrit and red cell volume.

82–1369. Sirotin, B. Z.; Shevtsov, B. P.; Davidovich, I. M.; Zharsky, S. L. **Vliyaniye Kuryeniya na Mikrotsirkulyatsiyu b Socudakh Bulybarnoy Konbywnktivy u Molodykh Bolynykh Yshyemichyeskoy Boleznyu Syerdtsa. [Effect of Tobacco Smoking on Microcirculation in the Bulbar Conjunctiva Vessels in Young Patients With Ischemic Heart Disease.]** *Terapevticheskii Arkhiv* 54(3): 70-73, 1982, Russian.

Microcirculation in the bulbar conjunctiva vessels of 52 young patients with ischemic heart disease was studied immediately and 15 minutes after smoking a cigarette. Development of arteriolar and venular constriction, and hemorrheological disorders, was manifested by the emergence or intensification of hemostasis. It is concluded that microcirculatory disorders occurring during smoking may be a factor in the pathogenesis of angina pectoris and arrhythmia in ischemic heart disease patients. (Auth. Abs. Mod.)

82–1370. Sorlie, P. D.; Garcia-Palmieri, M. R.; Costas, R.; Cruz-Vidal, M.; Havlik, R. **Cigarette Smoking and Coronary Heart Disease in Puerto Rico.** *Preventive Medicine* 11(3): 304-316, May 1982.

Cigarette smoking habits were assessed in over 9,000 men, aged 45 to 64 years, who participated in a prospective study of coronary heart disease (CHD) in Puerto Rico beginning in 1965. Fewer Puerto Rican men smoked cigarettes, and they smoked fewer cigarettes per day than in comparable studies in the mainland United States. However, even in this lower-smoking, low-CHD population, cigarette smoking showed a significant independent association with the incidence of myocardial infarction (MI) over an 8-year period, though this association was not statistically significant for other manifestations of CHD. Pipe and cigar smokers had a risk of MI similar to nonsmokers. Those men who used filter cigarettes or who stated that they did not inhale showed the same risk of MI as other cigarette smokers. Ex-smokers had a risk of MI intermediate to that of nonsmokers and current smokers. The risk of MI did not increase with the number of cigarettes smoked, but the risk of MI in smokers as a group relative to those who had never smoked was 3.4 in the rural area and 2.0 in the urban area. These results emphasize that there is an excess risk of MI even in those cigarette smokers who reported that they used filter cigarettes, did not inhale, or smoked a small number of cigarettes a day. (Auth. Abs.)

82–1371. Stamler, J. **Part IV. Primary Prevention of Epidemic Premature Atherosclerotic Coronary Heart Disease.** In: Yu, P. N.; Goodwin, J. F. (Editors). *Progress in Cardiology 10.* Philadelphia, Lea & Febiger, 1981, pp. 63-100.

The scope and nature of the problem of prevention of atherosclerotic coronary heart disease (CHD) is discussed, growing public awareness and scientific advances over the last several years are noted, and possible advances in primary prevention in the next few decades are projected. Lifestyle changes, such as cessation of smoking, better dietary habits, controlling blood pressure, and increased physical activity, practiced by more Americans today, may account for the reduced mortality from CHD since 1968. The number of regular smokers has shown a steady decline—from 41.7 percent of the population over 17 in 1965 to 33.2 percent in 1978—although smoking among women and adolescents is still a major problem. Progress in areas of diet, physical activity, and other necessary lifestyle changes are reviewed, and comparisons with other countries are made, noting a relationship which infers that lower CHD mortality rates follow positive trends in lifestyles. The prerequisites for prevention of mass disease, the predicted advances in CHD prevention in the 1980s and 1990s, and predicted advances in research relating to prevention are also discussed. It is concluded that with the scientific and social details already known, any epidemic of premature CHD can be prevented in the years and decades just ahead.

82–1372. Stubbe, I.; Eskilsson, J.; Nilsson-Ehle, P. **High-Density Lipoprotein Concentrations Increase After Stopping Smoking.** *British Medical Journal* 284(6328): 1511-1513, May 22, 1982.

Concentrations of plasma lipoproteins in 10 men who were habitual smokers were monitored for 6 weeks after they stopped smoking and were related to changes in diet and body weight. The energy intake increased by 10 percent due to a higher consumption of carbohydrate and fat, and body weight increased by 2 percent. Plasma triglyceride, cholesterol, and low-density lipoprotein cholesterol concentrations did not change significantly. However, high-density lipoprotein (HDL) concentrations rose from comparatively low values by 29 percent within 2 weeks and remained at this value throughout the observation period. In three subjects who resumed smoking after the end of the study, HDL levels fell to initial values within 6 weeks. The initial increase in concentration could be accounted for mainly by an increase in the esterified fraction and only to a lesser extent in the free cholesterol fraction. The changes in concentration were accompanied by similar but less pronounced rises in HDL phospholipid and in apolipoprotein AI concentrations, whereas HDL triglyceride concentrations did not change significantly. The changes in HDL concentrations may be partly due to nutritional factors. (Auth. Abs. Mod.)

82–1373. Theriault, G.; De Guire, L.; Gingras, S.; Laroche, G. **Raynaud's Phenomenon in Forestry Workers in Quebec.** *Canadian Medical Association Journal* 126(12): 1404-1408, June 15, 1982.

The prevalence of Raynaud's phenomenon was higher among smokers than among ex-smokers and nonsmokers in a survey of 1,540 forestry workers in Quebec. A prevalence of the phenomenon of 30.5 percent among chain-saw users and 8.7 percent among nonusers was related directly to the duration of use of the chain-saw. Relative risks among those using a chain-saw for more than 10 years were 3.60 for nonsmokers and 6.55 for smokers. This association corresponds to an additive rather than a synergistic effect. Other variables discussed include family history, previous injuries to the arms, climate, and type of residence during the wood-cutting season.

82–1374. Thompson, D. **Smoking and Heart Disease.** *Nursing Mirror* 154(1): 33, January 6, 1982.

The belief that smoking is a cause of heart disease and specifically that stopping smoking decreases ischemic heart disease (IHD) mortality is questioned. The belief results from research which assumes that ex-smokers are a representative sample of smokers, except that they have stopped smoking. The validity of this assumption has not been proven, as is indicated by cited research that contradicts such conclusions. Further, although a positive association between smoking and IHD has generally (but not invariably) been observed, such an association does not necessarily imply causation. The constitution hypothesis (whereby the genetic factors that predispose to smoking associate positively with those that predispose to IHD) is noted as the main alternative to the causal relationship.

82–1375. Tonascia, J.; Szklo, M.; Goldberg, R.; Kennedy, H. **Predictors of Ventricular Fibrillation or Cardiac Arrest in Patients Hospitalized for Acute Myocardial Infarction.** *Clinical Cardiology* 4(4): 168-171, July-August 1981.

A community-wide study of 1,308 patients hospitalized with acute myocardial infarction in metropolitan Baltimore was conducted to examine sociodemographic and clinical characteristics in association with ventricular fibrillation and cardiac arrest (VF/CA). Multivariate analyses revealed that the variables associated with increased occurrence of VF/CA included older age (60 years or older), male sex, and a history of cigarette smoking. The smoking habit was also a significant predictor of VF/CA complicating a myocardial infarction. These factors allow the identification of subgroups of patients hospitalized with acute myocardial infarction at high risk for the subsequent development of VF/CA, in whom prophylactic therapy and close surveillance are especially recommended. (Auth. Abs. Mod.)

82–1376. Weir, F. W.; Fabiano, V. L. **Re-Evaluation of the Role of Carbon Monoxide in Production or Aggravation of Cardiovascular Disease Processes.** *Journal of Occupational Medicine* 24(7): 519-525, July 1982.

As an environmental pollutant, carbon monoxide (CO) is without equal in ubiquity. By far, the greatest source of individual exposure to CO is tobacco smoke, which averages about 40,000 parts per million (ppm) CO by volume; 400 ppm has been estimated as the proportion inhaled. Additionally, CO concentration in the smoke increases as the cigarette becomes shorter, and tends to be greater in filter-tipped cigarettes. Extensive epidemiological studies have produced statistically convincing evidence of an association between smoking and cardiovascular disease (CVD). In addition to CO and nicotine, smoke contains other compounds and elements which may be of pathogenic consequence; however, to date, CVD research has focused on nicotine and CO. Results of these studies indicate that nicotine is probably not an etiological factor in atherosclerosis development, but does play a role in precipitating sequelae of coronary artery atherosclerosis, such as arrhythmias and myocardial infarction. It is concluded that no convincing evidence is available to indicate that chronic CO exposure increases the risk of developing clinically significant atherosclerotic disease.

82–1377. Wilhelmsen, L.; Bjure, J.; Ekstrom-Jodal, B.; Aurell, M.; Grimby, G.; Svardsudd, K.; Tibblin, G.; Wedel, H. **Nine**

Years' Follow-Up of a Maximal Exercise Test in a Random Population Sample of Middle-Aged Men. *Cardiology* 68(Supplement 2): 1-8, 1981.

Smoking was identified as one of the three main risk factors (the others being high cholesterol and high blood pressure (BP)) for myocardial infarction (MI) or sudden coronary death (SD) in a bicycle exercise test of 793 Swedish men, all 54 years of age. The stepwise exercise test measured the maximum work load—the subject's recognition of exhaustion or a plateau of heart rate when the work load was increased in small steps. High respiratory rate during exercise characterized those who later suffered MI or SD. In addition to the three main risk factors, low maximal performance, but not ST changes during exercise, increased the risk of MI and/or SD. Pulse rate and systolic BP at both submaximal and maximal work load were positively correlated with the initial blood pressure level. BP at these work loads was also positively correlated with subsequent BP increase. (Auth. Abs. Mod.)

82-1378. Wilhelmsson, C.; Vedin, A.; Wilhelmsen, L. **Cost-Benefit Aspects of Post-Myocardial Infarction Intervention.** *Acta Medica Scandinavica* Supplementum 651: 317-321, 1981.

In a cost-benefit analysis of post-myocardial infarction intervention methods, smoking cessation was found to demonstrate a positive benefit, as did chronic beta-blockage and bypass surgery for certain categories. After myocardial infarction (MI) the mortality during the first post-hospital year declines from approximately 10 percent to 5 percent during the second year. Simplistically, the prognosis during the first year is related to the extent of the myocardial damage and thereafter, primary risk factors become more important. Thus, it seems logical in the short-term perspective to treat myocardial damage and related arrhythmias, and in the long-term perspective to reduce primary risk factors. Studies have shown a 50 percent reduction in mortality after MI with cessation of smoking. In addition, the cost of quitting was negligible since the advice was given by the attending physician in the hospital. (Auth. Abs. Mod.)

82-1379. Wolfgang, J.; Dennison, D. **An Investigation of Cardiovascular Disease Risk Factors in an Adolescent Population.** *Journal of School Health* 52(4): 218-221, April 1982.

Self-reported behavior and biomedical cardiovascular disease risk factors in an adolescent population were statistically analyzed. Self-reported data were collected with a Heart Health Profile (HHP) from a random sample of students (mean age 17.03 years, 47.5 percent female) enrolled in a required health education program of a western New York high school. The HHP was a personal diary using a "bogus pipeline" concept to encourage accurate and honest responses. Behavior variables included mean daily smoking frequency, percentage of dietary intake as fat, percentage of dietary fat intake as saturated fat, and amount of dietary cholesterol intake. An Ecolyzer was used to measure the biomedical variable of alveolar carbon monoxide levels (CO), and blood analysis was performed. It was evident from the data that the adolescent sample was at an elevated behavioral risk. The mean (X) of the sample was greater than the recommended level (R) for smoking (R=0; X=2.4 smoking incidents). Although 32 percent of the population smoked, no significant differences were found between males and females for average smoking and for mean CO values. The smoking/high density lipoprotein cholesterol relationship found in the correlation supports the concept found in adult research. The smoking/CO correlation was expected, since CO is a byproduct of smoking. Because behaviors considered to be precursors of heart disease may not become problematic until later in life and because one of every three students smoked, the need for smoking education components was evident.

See also, **82-1282, 82-1412**

PREGNANCY AND INFANT HEALTH

82-1380. Ayromlooi, J.; Desiderio, D.; Tobias, M. **Effect of Nicotine Sulfate on the Hemodynamics and Acid Base Balance of Chronically Instrumented Pregnant Sheep.** *Developmental Pharmacology and Therapeutics* 3: 205-213, 1981.

The maternal and fetal effects of nicotine sulfate (15 mg/10 min) upon the hemodynamics and acid-base balance were evaluated in nine intravenous infusions to five chronically instrumented pregnant sheep preparations of 97-117 days of gestation. The ewes received a nicotine dose about equal to what they would absorb by inhaling the smoke from four cigarettes within 10 minutes. Experiments were performed from 2 to 11 days postoperatively. Maternal (n=9) and fetal (n=8) arterial blood samples were simultaneously drawn at 0, 10, 30, and 60 min from the start of infusion. Maternal arterial pH transiently increased at 10 min, while arterial base deficit simultaneously fell. Maternal blood pressure increased during the infusion at 10 min, remaining elevated at 30 min. Maternal heart rate transiently rose at 1 min, returning to control levels by 10 min. Ten min into infusions a decrease in uterine blood flow was recorded below preinfusion values. A gradual recovery was observed at 60 min. Fetal blood pressure also decreased transiently during the infusion. Fetal arterial carbon dioxide partial pressure fell at 10 min. A transient increase was seen in fetal heart rate at 30 min. No significant changes were seen in fetal arterial oxygen partial pressure and O_2 percent saturation of hemoglobin. All maternal and fetal values except uterine blood flow returned to preinfusion levels by 60 min. Chronically decreased uterine blood flow may partly account for the higher incidence of small-for-gestational-age infants to smoking mothers. (Auth. Abs. Mod.)

82–1381. Deshpande, T. V.; Hinton, G. G. **Intrauterine Growth Retardation and Its Impact on the Neurological Status of the Newborn.** *Indian Journal of Pediatrics* 48(392): 305-311, May-June 1981.

A neurological study of 40 small-for-date (SFD) newborn infants by quantitative and qualitative methods revealed a significant incidence of poor muscle tone and diminished reflex activity in SFD infants compared with a control group of 42 appropriate-for-date newborn infants. Significantly reduced head circumference was noticed in term SFD infants. The neurological score which correlated with the head circumference showed a greater reduction in the complicated group of SFD infants compared to the uncomplicated group. Maternal smoking was the chief factor related to dysmaturity in this study. In addition to the growth retarding influence leading to dysmaturity, the toxic effect of maternal smoking on the developing brain has been of some concern. Since the strong correlation of maternal smoking to dysmaturity and increased perinatal mortality is well documented, maternal smoking, which is a preventable factor, deserves serious attention by clinicians involved in perinatal care. (Auth. Abs. Mod.)

82–1382. Drabick, D.; Duch, P. **Synergistic Effects of Aspirin, Barbiturates, Caffeine and Nicotine (The "Head Embryo").** In: Proceedings of the 26th Annual Technical Meeting, Philadelphia, May 11-14, 1980, Institute of Environmental Sciences, 1980, pp. 388-390.

The synergistic effects of coffee (caffeine-C), aspirin (salicylic acid-A), cigarettes (nicotine-N), and barbiturates (barbituric acid-B) were tested on the developing chick embryo. In determining the dosage of each drug that was teratogenic and at what time during embryogenesis the drug appeared to be teratogenic without being lethal, it was found that the embryos were too susceptible to all four drugs at 0 hours and that 0.1M was a lethal dose of all the drugs tested. Therefore, the synergistic effects of these drugs were tested on 24-hour-old embryos with .0001M dosages and a total application volume of 0.5 ml. The application of the combined drugs involved cutting windows in the egg shells, adding the test agents on top of the embryo, sealing off the windows with paraffin and cover slips, and incubating the eggs at 37.5°C for an additional 48 hours. Comparisons of experimental embryos and control embryos after incubation for 72 hours showed that: no treated embryos were normal and although various anomalies and various degrees of abnormalities were present in all experimental embryos, the most significant finding was the inhibition of node regression in the embryos treated with aspirin and barbiturates. (Auth. Abs. Mod.)

82–1383. Fribourg, S.; Krous, H. F. **Cigarette Smoke and Sudden Infant Death Syndrome. (Letters).** *American Journal of Obstetrics and Gynecology* 142(7): 934, April 1, 1982.

A number of questions stimulated by the reading of an article (*American Journal of Obstetrics and Gynecology* 140(7): 743-746, August 1, 1981) relating cigarette smoking to sudden infant death syndrome (SIDS) are listed. These are followed by the suggestion that although the report might be considered thought-provoking, one might conclude that the rat model used is not necessarily consistent with the subject matter analyzed and, therefore, the conclusion drawn may be totally unrelated to the relationship, if any, of smoking and SIDS in the human. In response, Krous (one of the authors of the original report) concedes the possibility that the rat model does not explain the enhanced risk of SIDS in infants of smoking mothers, but does attempt to demonstrate a causal relationship between one noxious component (nicotine) of cigarette smoke and fetal brain stem damage. See also 81-1889.

82–1384. Garn, S. M.; Ridella, S. A.; Petzold, A. S.; Falkner, F. **Maternal Hematologic Levels and Pregnancy Outcomes.** *Seminars in Perinatology* 5(2): 155-162, April 1981.

The relationship between maternal hematologic levels during pregnancy and the outcomes of pregnancy were explored for blacks and whites, using the data of 50,000 consecutive pregnancies followed in the National Collaborative Perinatal Project of the National Institute of Neurological Disorders and Stroke. Since maternal smoking could affect these results, smokers were separated from nonsmokers in the analysis. The finding of a systematically curvilinear "U-shaped" relationship between maternal hematologic levels and the proportion of unfavorable pregnancy outcomes make it clear that both extremes greatly exert a negative effect. Smoking, toxemias, and even hypertension, while important in their own right, did not appreciably alter the effect of hematologic extremes in blacks or whites.

82–1385. Halac, J.; Arias, M. E.; Numa, R. Y.; Halac, E. **Fetal Growth Rates in Argentina—A National Collaborative Study.** *Clinical Pediatrics* 21(6): 342-347, June 1982.

Argentine infants do not grow at the same rate as those in other countries and, as expected, social and geographic variations can influence fetal growth. A sample of 7,520 pregnancies, representative of the national population, was used to construct intrauterine growth charts. These are applicable nationwide and since 1974, when first designed, have proved efficient and more reliable in evaluating Argentine neonates than the Colorado Growth Charts. Smoking mothers constituted 32 percent of the sample. These mothers were free of illness and socioeconomic burdens, and smoking was the only significant variable. Their babies had lower birth weight and length but normal head circumferences. The usefulness of the charts was evaluated, and despite certain changes, they remain as accurate and valuable as when originally developed. (Auth. Abs. Mod.)

82-1386. Kuhnert, P. M.; Kuhnert, B. R.; Bottoms, S. F.; Erhard, P. **Cadmium Levels in Maternal Blood, Fetal Cord Blood, and Placental Tissues of Pregnant Women Who Smoke.** *American Journal of Obstetrics and Gynecology* 142(8): 1021-1025, April 15, 1982.

Since cigarette smoking is a major source of exposure to cadmium (Cd), a study to determine the degree of exposure to Cd of smoking pregnant women and its concentration in the maternal-fetoplacental unit was conducted. The results indicate that pregnant women who smoke expose themselves and their placentas to higher levels of Cd than normal. The increase in Cd due to smoking was 32 percent in the placenta and 59 percent in maternal blood, increases which are statistically significant. The mean levels of Cd in maternal blood, cord blood, and placental tissues of pregnant women who smoked were all higher than the mean levels of Cd in pregnant women who did not smoke. In addition, the levels of Cd in the maternal blood of smokers were significantly higher than levels of Cd in the cord blood of their infants, a relationship not found in nonsmokers. On the basis of the data, the fetuses of pregnant women who smoke apparently receive very little additional exposure to Cd; however, this does not lessen concern for the fetus. The reported increase in exposure to Cd of pregnant women due to smoking must be viewed as undesirable because Cd has been shown to alter placental function in animals, and because Cd has no known biologic function. (Auth. Abs.)

82-1387. Naeye, R. L. **Factors That Predispose to Premature Rupture of the Fetal Membranes.** *Obstetrics and Gynecology* 60(1): 93-98, July 1982.

Causes of premature rupture of the fetal membranes were explored in a study of 10,450 pregnancies. Five risk factors were analyzed: cigarette smoking, coitus, parity, prior surgery of the cervix, and chorioamnionitis. Premature rupture of the fetal membranes is more frequent in smokers than in nonsmokers, in pregnancies that reached full term. However, since heavy smokers did not have more premature ruptures than light smokers, the clinical significance of smoking is considered small. Preterm delivery due to premature rupture of the membranes was 11 times more frequent when recent coitus was present in conjunction with chorioamnionitis than when both were absent. Such ruptures were increased only threefold when either recent coitus or chorioamnionitis was present without the other. (Auth. Abs. Mod.)

82-1388. Papoz, L.; Eschwege, E.; Pequignot, G.; Barrat, J.; Schwartz, D. **Maternal Smoking and Birth Weight in Relation to Dietary Habits.** *American Journal of Obstetrics and Gynecology* 142(7): 870-876, April 1, 1982.

A study on dietary and smoking behavior during pregnancy, performed between January 1976 and September 1979 at the St. Antoine Maternity Hospital in Paris, is described. In addition to the routine clinical examinations at the third, sixth, eighth, and ninth months of pregnancy, the women were systematically questioned about their dietary and tobacco habits. No dietary advice was given by the dietitians at any time, nor was there special counseling against smoking. Of the 534 women who were followed up, 200 (37 percent) were smokers before pregnancy. At the sixth month, half of them had stopped smoking and the others had reduced their consumption. The analysis showed that the mean caloric intake and the gain in weight were the highest in the women who continued to smoke and the lowest in nonsmokers. Intermediate results were found in smokers who stopped. Birth weight of the infant was, on the average, 70 gm lower among those who smoked throughout the pregnancy compared with the other two groups, but this difference was not significant. These findings are consistent with the hypothesis that the negative effects of smoking on the fetus could be compensated for, to a great extent, by extra food. (Auth. Abs.)

82-1389. Ross, S.; Naeye, R. L. **Racial and Environmental Influences on Fetal Lung Maturation.** *Pediatrics* 68(6): 790-795, December 1981.

Claims that fetal lung maturation is more rapid in blacks than in whites were investigated. Histologic measurements of lung maturation and the frequency of hyaline membrane disease were compared in four groups of neonates: 490 South African blacks, 841 Ethiopians, 767 U.S. blacks, and 560 U.S. whites. Lungs matured much more rapidly in the Ethiopian and more slowly in the South African than in the U.S. fetuses. The rate of maturation was about the same in U.S. blacks and whites. Hyaline membrane disease was more frequent in the South Africans and less frequent in the Ethiopians than in either U.S. group. Environmental factors in the mother may explain some of these differences. Lungs matured more rapidly in the fetuses of cigarette smokers than in fetuses of nonsmokers. Half of the U.S. mothers but almost none of the South African mothers smoked during pregnancy. Smoking reduces uteroplacental blood flow; thus underperfusion is a possible explanation for the more rapid lung maturation in the offspring of smokers. The rate of lung maturation had an inverse correlation with maternal body weights. The South African women were heavier and the Ethiopians lighter than women in the United States. (Auth. Abs. Mod.)

82-1390. Smith, N.; Austen, J.; Rolles, C. J. **Tertiary Smoking by the Fetus. (Letter).** *Lancet* 1(8283): 1252, May 29, 1982.

The conclusion of a study (*Lancet* 1(8275): 791, April 3, 1982) that only prolonged passive smoking by a nonsmoking pregnant woman would lead to "appreciable" amounts of cotinine in the amniotic fluid around her fetus is questioned. Problems cited include: (1) the lack of definition of "appreciable," (2) the lack of data on the level of gestation when the sample was taken, and (3) the lack of knowledge concerning the source of cotinine in amniotic fluid (produced by the fetus or maternally derived). Reference is made to a study conducted in Southampton in which samples of amniotic fluid were taken from 14 women (10 smokers, 4 nonsmokers) of 16-18 weeks' gestation. All four of the nonsmokers had cotinine concentrations of 9 ng/ml or more. Results of the study suggest that one-third to two-thirds of all fetuses in Southampton are either secondary or tertiary smokers; but to what degree this is harmful is still unknown.

OTHER DISEASES AND CONDITIONS

82-1391. Bennet, K. R.; Reade, P. C. **Salivary Immunoglobulin A Levels in Normal Subjects, Tobacco Smokers, and Patients With Minor Aphthous Ulceration.** *Oral Surgery, Oral Medicine Oral Pathology* 53(5): 461-465, May 1982.

There appears to be no relationship between time of day and salivary immunoglobulin A (SIgA) concentration in pooled unstimulated saliva. Such concentrations are more accurately expressed as a range of normal. A selected group of tobacco smokers showed a decrease in SIgA concentrations when compared with a matched control group (nonsmokers), and a group of patients with minor aphthous ulcerations showed no deviation from the control range. The significance of the decrease in SIgA concentrations in chronic tobacco smokers is discussed relative to animal experiments on the immunosuppressive effect of the combustion products of tobacco and the possibility of the incidence of intraoral neoplastic disease being increased in tobacco smokers by this effect. (Auth. Abs. Mod.)

82-1392. Bianchi Porro, G.; Prada, A.; Petrillo, M.; Lazzaroni, M.; Achord, J. L. **Gastric Acid Secretion, Smoke and Duodenal Ulcer Healing. (Letters).** *Gastroenterology* 82(2): 394-395, February 1982.

Contrary to the conclusion of Dr. J. L. Achord (*Gastroenterology* 81: 15-18, 1981), which found a correlation between gastric acid secretion and both duodenal ulcer activity and duodenal ulcer healing, smoking is identified as the only factor which seems to influence the ulcer healing rate. A study compared basal acid output and maximal acid output in 265 patients with active duodenal ulcer (ADU) and 38 patients with duodenal scar (untreated for a few weeks prior to gastric analysis). No significant difference was observed between the two groups. In 214 ADU patients treated with either H_2-blockers ($n=134$) or placebo ($n=80$), a significantly lesser healing rate was observed in smokers compared with nonsmokers (64 percent vs. 82 percent in the H_2 treated patients and 26 percent vs. 41 percent in the placebo group). Data were also accumulated which show that smoking not only reduces short-term healing rate but appears to promote recurrence. In reply, Dr. Achord states that comparing data is difficult as acid and pepsin decrease occurred only after several months of inactive disease in the original study while the 38 patients with duodenal scar were analyzed after only a few weeks off the medication. It is acknowledged that the relationship of smoking to peptic ulcer disease was not addressed.

82-1393. Dang, C. V. **Tobacco—Alcohol Amblyopia: A Proposed Biochemical Basis for Pathogenesis.** *Medical Hypotheses* 7(11): 1317-1328, 1981.

Tobacco—alcohol or nutritional amblyopia is a rare disorder of decreased central vision associated with nutritional deficiencies and tobacco smoking. Although folate deficiency is common in patients with tobacco—alcohol amblyopia, the role of folic acid in this disorder has been underplayed. The role of cyanide from tobacco smoke, folate, and other dietary deficiencies is reviewed. A new model of the pathogenesis of tobacco—alcohol amblyopia is proposed based on the presumptive alteration of methionine and S-adenosyl-L-methionine metabolism. (Auth. Abs.)

82-1394. Evans, G.; Reader, K. **Totally Unacceptable. (Letters).** *New Scientist* 88(1223): 182, October 16, 1980.

Two opposing views of an earlier article (*New Scientist* 88(1221): 10-13, October 2, 1980) on the possible hazards of passive smoking are presented. Evans criticizes the article's comparison of atmospheric levels of carbon monoxide which is made without noting that some were found under natural conditions and others under extreme experimental conditions. Also, the article does not mention accepted air quality standards and maximum safe working levels, and describes "safety" as a relative term, with nothing completely free of risk. Reader describes unpleasant symptoms experienced after exposure to tobacco smoke, including severe chest pains, loss of strength from chest and shoulder muscles, and release of tears. The symptoms later recurred in milder form after another exposure. It is stated that at other times tobacco smoke has caused sore eyes, headache, sore palate, and sore throat. See also 82-1409.

82-1395. Evans, S. C. **The Effect of Alcohol and Tobacco on Eye Health.** Oulton, England, Teecoll Publications, 1981, 57 pp.

The relationship between smoking, drinking, and drug taking and the onset and progress of cataract, glaucoma, retinopathy, and other serious eye disorders leading to blindness are discussed. Following an explanation of the main features of the human eye and those parts that are especially liable to damage through alcohol, tobacco, and drug use, seven pathological eye disorders are described. These disorders are: tobacco amblyopia, macular degeneration, cataract, glaucoma, keratitis, retinopathy and chorodítis, and early presbyopia.

82-1396. Ginter, E.; Kallner, A.; Hornig, D.; Hartmann, D. **Ascorbic Acid in Smokers. (Letters).** *American Journal of Clinical Nutrition* 35(5): 1043-1045, May 1982.

A previous study (*American Journal of Clinical Nutrition* 34(7): 1347-1355, July 1981) which demonstrated an increased turnover of ascorbic acid in smokers is criticized. The reported turnover reflects a higher intake than recorded, probably due to either increased absorption or an error in the pharmacokinetic analysis. Thus the ascorbic acid turnover in smokers relative to nonsmokers is overestimated. The authors of the original study deny the possibility of increased absorption or error in analysis. The divergence between proclaimed intake and calculated total turnover is probably due to extra vitamin C intake by smokers who did not maintain a constant diet. However, as the results were based on calculated total turnover and not on intake, an overestimation did not occur. See also 81-2043.

82-1397. Harries, A. D.; Jones, L.; Heatley, R. V.; Rhodes, J. **Smoking Habits and Inflammatory Bowel Disease: Effect on Nutrition.** *British Medical Journal* 284(6323): 1161, April 17, 1982.

The effect of smoking habits on weight and other anthropometric variables in Crohn's disease vs. ulcerative colitis patients vs. controls was investigated. Nonsmokers in the control and ulcerative colitis groups had more subcutaneous fat and were heavier than smokers. However, both smokers and nonsmokers with Crohn's disease were undernourished: the nonsmokers had significantly reduced weight compared to those of the other two groups. There was no difference in smokers' weights between the three groups.

82-1398. Kent, D. C.; Cenci, L. **Smoking and the Workplace: Tobacco Smoke Health Hazards to the Involuntary Smoker.** *Journal of Occupational Medicine* 24(6): 469-472, June 1982.

In a survey of top management and medical officials of 3,000 U.S. companies, almost 15 percent had a smoking program for employees. These included restrictions on workplace smoking, hiring policies, how-to-quit programs, and literature. More than 3,000 toxic substances have been identified in cigarette smoke; some of these are in higher concentrations in sidestream smoke than mainstream smoke. Over an 8-hour workday, the nonsmoker may be exposed beyond threshold limits to carbon monoxide, ammonia, nicotine, toluene, dimethylnitrosamine, aniline, naphthalene, and others. The listed effects of involuntary smoking include increased heart rate; reduced maximum exercise capacity; eye, nose, and throat irritation; and changes in psychomotor function. As people spend 70 to 80 percent of their time indoors, indoor air pollution may prove to be a greater problem than outdoor air pollution. Also, the estimated cost of the habit to business alone is 11 billion dollars, though few employers surveyed had calculated their costs. Proposals for curbing employee smoking, published by Action on Smoking and Health, are reiterated.

82-1399. Kriss, A., Carroll, W. M.; Blumhardt, L. D.; Halliday, A. M. **Pattern- and Flash-Evoked Potential Changes in Toxic (Nutritional) Optic Neuropathy.** *Advances in Neurology* 32: 11-19, 1981.

In a sample of 24 toxic optic neuropathy patients (mean age 52.6 years), 71 percent smoked in excess of 40 cigarettes per day and 92 percent drank more than 4 pints of beer per day. Sixty-three percent both smoked and drank. Occipital potentials to flash and pattern reversal, and corneal electroretinograms were recorded, and compared to controls. Acuity from 79 percent of the examined eyes was worse than 6/12 at the time of the evoked potential test, and all patients showed color vision defects and some form of scotomata. In 13 patients who were recorded more than once, acuity improved considerably with vitamin B_{12} therapy, abstinence, and time, such that 85 percent had acuities of 6/12 or better. Whether vitamin B deficiency or toxins such as cyanide in tobacco smoke cause these visual disturbances has not been established.

82-1400. Lee, P. N. **Passive Smoking.** *Food and Cosmetics Toxicology* 20: 223-229, 1982.

While more research is certainly needed, there seems at present to be no convincing evidence that passive smoking results in any material risk of serious disease for the healthy nonsmoker. Before 1980 the argument that passive smoking was a serious health hazard was rather tenuous. It was claimed that it produced allergic reactions, impaired driving ability, reduced exercise tolerance in patients with cardiorespiratory disease, and increased the risk of bronchitis and pneumonia in first-year children. However, none of these claims provided convincing evidence relevant to the normal healthy adult nonsmoker. Many studies indicate that nonsmokers are unlikely to inhale more than a very small amount of those components of tobacco smoke traditionally considered harmful. It was surprising, therefore, when a study carried out in the United States (*New England Journal of Medicine* 302(13): 720-723, March 27, 1980) showed reduced airways function, and studies from Japan (*British Medical Journal* 282: 183-185, January 15, 1981) and Greece (*International Journal of Cancer* 27(1): 1-4, January 15, 1981) showed an increased lung cancer incidence in nonsmokers passively exposed to tobacco smoke in comparison with nonsmokers not so exposed. A review of the detail of these studies suggests that none provides conclusive evidence that passive smoking is seriously harmful, a view supported by a recent large study that was carried out in the United States (*Journal of the National Cancer Institute* 66(6): 1061-1066, June 1981) and in which no significant relationship was found between passive smoking and lung cancer. More research is urgently needed, particularly to explore the influence of potentially confounding factors. (Auth. Abs. Mod.) See also 80-1190, 81-1088, 81-0785, and 81-1418.

82-1401. Martin, F.; Farley, A.; Gagnon, M.; Bensemana, D. **Comparison of the Healing Capacities of Sucralfate and Cimetidine in the Short-Term Treatment of Duodenal Ulcer: A Double-Blind Randomized Trial.** *Gastroenterology* 82(3): 401-405, March 1982.

Fifty-nine outpatients with endoscopically proven duodenal ulcers were evaluated for 4 to 8 weeks in a randomized, double-blind trial comparing sucralfate, a sulfated disaccharide (1 g, 0.5 hour before each meal and at bedtime), with cimetidine (300 mg, 0.5 hour before each meal and at bedtime). Ulcer symptoms and their relief were recorded by patients in a diary, along with data on cigarette, alcohol, coffee, and drug intake. Duodenoscopy was performed after 4 weeks to assess healing, and was repeated after 8 weeks if healing had not occurred by the 4-week evaluation. Twenty-four of 30 patients taking sucralfate (80.0 percent) and 22 of 29 patients taking cimetidine (75.9 percent) had their ulcers completely healed after 4 weeks. The overall healing rates after 8 weeks for the sucralfate and cimetidine groups were 90.0 percent (27 of 30 patients) and 86.2 percent (25 of 29 patients), respectively. There were no significant differences between the two treatment groups in ulcer healing, symptom relief, and side effects. No differences between groups were found in smoking habits. All but nine patients were regular smokers. The mean numbers of cigarettes/patient/day for the sucralfate and cimetidine groups were 18.8 and 17.5, respectively. No conclusions could be drawn regarding the levels of smoking and the healing results. (Auth. Abs. Mod.)

82-1402. Mattison, D. R. **The Effects of Smoking on Fertility From Gametogenesis to Implantation.** *Environmental Research* 28(2): 410-433, August 1982.

The literature concerning the effects of cigarette smoking, treatment with nicotine, cigarette smoke condensates, or cigarette smoke constituents on reproductive events from gametogenesis to implantation is reviewed. Epidemiologic evidence, although scanty, suggests that cigarette smoking decreases fertility in women. Cigarette smoking is also associated with an increased frequency of menstrual abnormalities and a cigarette dose-related decrease in the age of spontaneous menopause. Similar studies have not been conducted among men who smoke, although several small studies have demonstrated decreased sperm counts and an increased frequency of abnormal sperm morphology among male smokers. Cessation of smoking in several of these reports has been associated with reversal of the abnormalities. Treatment of rodents with cigarette smoke or cigarette smoke constituents produces similar effects on sperm number and morphology. Experimental evidence from humans and rodents suggests that cigarette smoke or nicotine can alter hypothalamic—pituitary interrelationships, stimulating growth hormone, cortisol, vasopressin, and oxytocin release and inhibiting luteinizing hormone and prolactin release. These changes may alter hormonal interrelationships necessary for successful reproduction. Cigarette smoke or nicotine may also alter the motility of the sperm, ovum, or conceptus within the female reproductive tract, and may impair implantation of the embryo. The data imply an adverse influence on reproduction at several sites from gametogenesis to implantation. However, additional experimentation is necessary before it will be possible to make unequivocal statements concerning reproductive interference by cigarette smoke or its components. (Auth. Abs. Mod.)

82-1403. Mayes, R. W. **Lack of Allergic Reactions in Workers Exposed to Pruteen (Bacterial Single-Cell Protein).** *British Journal of Industrial Medicine* 39(2): 183-186, May 1982.

The smoking habit was not found to influence symptoms from industrial exposure to Pruteen. Men exposed to high concentrations of bacterial single-cell protein (Pruteen) dust have complained of "sticky eyes" and influenza-like symptoms. Over 4 years, surveillance of a work force of 70 men with a program that included a respiratory health questionnaire, skin prick testing, serum testing for precipitating antibodies, lung function measurements, and chest radiography has shown no evidence of allergic reaction or of exposure-related deterioration in lung function. Of the sample population, 18 men were heavy smokers and 15 were light smokers: the decreases in lung function found in this study were correlated with smoking rather than Pruteen exposure. The influenza-like symptoms experienced may be attributable to exposure to lipopolysaccharide. The results show that Pruteen can be produced and handled without adverse effects on health, provided that adequate measures are taken to prevent exposure to high concentrations of dust. (Auth. Abs. Mod.)

82-1404. Olshansky, S. J. **Is Smoker/Nonsmoker Segregation Effective in Reducing Passive Inhalation Among Nonsmokers?** *American Journal of Public Health* 72(7): 737-739, July 1982.

The segregation of smokers and nonsmokers in an experimental area did not appear to protect the nonsmokers from the possible effects of passive smoking. Expired carbon monoxide (CO) and a test of coordination were used as measures of tobacco smoke exposure in a natural environmental setting where smokers and nonsmokers were segregated. Results indicated that by comparison to a control group, subjects seated in adjacent smoking/nonsmoking environments were not only exposed to similar ambient levels of CO, but also showed similar physical and physiological reactions to their exposure in the form of coordination test scores, expired CO, and blood carboxyhemoglobin. While the results may not be generalized to other tobacco smoke constituents or other environmental settings, they raise questions about the health benefits of smoker segregation which future research must address. (Auth. Abs. Mod.)

82-1405. Petitti, D. B.; Klein, R.; Kipp, H.; Kahn, W.; Siegelaub, A. B.; Friedman, G. D. **A Survey of Personal Habits, Symptoms of Illness, and Histories of Disease in Men With and Without Vasectomies.** *American Journal of Public Health* 72(5): 476-480, May 1982.

Using questionnaire information provided by 4,385 vasectomized and 13,155 matched, nonvasectomized men, no significant differences were found for a large number of symptoms and diseases, including those of the cardiovascular system. Of the vasectomized group, 38.5 percent were current smokers and 37.4 percent of the controls smoked. Vasectomy was strongly associated with marital status, stomach or duodenal ulcer, kidney or bladder infection, back pain, joint pain or swelling, and serious marital problems. However, stomach or duodenal ulcer was strongly associated with ever having smoked cigarettes: after stratifying by smoking habit, the differences between the two groups were small, the probability values large, and the rate ratios near one. These findings are inconsistent with the occurrence of large increases in the risks of many important diseases in vasectomized humans. (Auth. Abs. Mod.)

82-1406. Roed-Petersen, B. **Effect on Oral Leukoplakia of Reducing or Ceasing Tobacco Smoking.** *Acta Dermato-Venereologica* 62(2): 164-167, 1982.

Cessation of smoking was found to be the most effective method in reducing or eliminating oral leukoplakia. Over a 3-year period, new leukoplakia patients who were smokers were asked to give up their smoking habit. A total of 138 patients (63 females and 75 males) responded to the advice. The patients were followed once a month for the ensuing 3 months. Results indicated that a reduction of the daily tobacco consumption to less than half the normal was associated with disappearance of leukoplakias in two patients (6.3 percent). A reduction of smoking, therefore, is of less clinical significance when the treatment is aimed at total disappearance of the leukoplakias. Over the same timespan of 3 months, quitting smoking was far more effective, increasing the number of total disappearances to 19 (27.1 percent), while the combined group of regression and disappearance increased from 56 to 63 percent. It is noted that leukoplakias in nonsmokers are idiopathic (which merely signifies that they are caused by factors so far not identified), and as these unknown etiologic factors must affect smokers too, a certain percentage of smokers would probably not be helped by a reduction in or abstinence from smoking.

82-1407. Rossiter, C. E. **Passive Smoking. (Letter).** *Lancet* 1(8285): 1356, June 12, 1982.

The conclusion (*Lancet* 1(8275): 791, April 3, 1982) that there is no valid evidence that passive smoking is deleterious to healthy adults is criticized for its omissions. It is argued that the conclusion must have been based on the rejections of studies which provided evidence of passive smoking's adverse effects on mortality and lung function. Though there is general agreement that passive smoking is harmful to the fetus, children, and unhealthy adults, it is difficult to accept that passive smoking—which is accepted as having serious effects on the "unhealthy"—should have no effect at all on the "healthy." A more balanced view is that despite the shortcomings of many studies, there is ample clinical, epidemiological, and physiological evidence to conclude that passive smoking is a serious health hazard. See also 82-1072.

82-1408. Schiodt, M.; Andersen, L.; Shear, M.; Smith, C. J. **Leukoplakia-Like Lesions Developing in Patients With Oral Discoid Lupus Erythematosus.** *Acta Odontologica Scandinavica* 39(4): 209-216, 1981.

In a long-term followup of 52 patients with oral lesions of discoid lupus erythematosus (DLE), 8 patients showed a gradual transition of the typical DLE lesions to lesions which closely resemble leukoplakia. Of the 8 who developed leukoplakia, 7 were smokers, and 40 of the remaining 44 were also smokers. The lesions generally showed features similar to those in leukoplakias not preceded by DLE, including the presence of cytoplasmic tubular structures in vascular endothelium in two out of three patients examined. The leukoplakia-like stage may be the mucous membrane counterpart of the atrophic scars following DLE lesions of the skin. Though some leukoplakias are caused by tobacco, smoking does not seem to be a factor in whether DLE lesions develop into leukoplakia-like lesions. Careful diagnosis of leukoplakias is recommended in the presence of DLE. (Auth. Abs. Mod.)

82-1409. Stock, S. **The Perils of Second-Hand Smoking.** *New Scientist* 88(1221): 10-13, October 2, 1980.

The claim of the Freedom Organization for the Right to Enjoy Smoking Tobacco (FOREST) that there is no evidence that passive smoking is harmful to health is refuted, citing constituents of mainstream and sidestream smoke which could be as damaging to the passive smoker as to the smoker. Results of studies of the problem, although sparse, also confirm the conclusion that passive smoking is harmful, particularly to persons suffering from allergies, or chronic heart or lung diseases. The most hazardous constituents of tobacco smoke and their carcinogenic properties are listed, and the actions of polycyclic aromatic hydrocarbons and nitrosamines are described. Occupational exposure to passive smoking and its consequences are also discussed, as are the higher incidence of bronchitis and pneumonia in children of parents who smoke, and the effects of maternal smoking on the fetus.

82-1410. Strom, M.; Gotthard, R.; Bodemar, G.; Walan, A. **Antacid/Anticholinergic, Cimetidine, and Placebo in Treat-**

ment of Active Peptic Ulcers. *Scandinavian Journal of Gastroenterology* 16(5): 593-602, 1981.

A 12-week double-blind trial was undertaken with 72 patients with duodenal ($n=54$) or prepyloric ($n=18$) ulcers. Twenty-four patients (21 smokers) received cimetidine, 1 g/day; 24 patients (16 smokers) received 10 ml of an antacid suspension (buffering 84 mmol acid) 1 and 3 hours after every meal and at bedtime and 0.6 mg L-hyoscyamine in sustained-release tablets twice a day; and 24 patients (14 smokers) received placebo. The healing rate after 3 weeks' treatment was 67 percent ($p<0.005$ compared with placebo) with cimetidine, 50 percent ($p<0.01$) with antacid/anticholinergic, and 13 percent with placebo. After 6 weeks' treatment 83 percent were healed with cimetidine ($p<0.005$ compared with placebo), 96 percent with antacid/anticholinergic ($p<0.005$), and 33 percent with placebo. 'A further 6 weeks' treatment gave healing rates of 96 percent for cimetidine, of 100 percent for antacid/anticholinergic, and of 50 percent for placebo. Compared with placebo, cimetidine but not antacid/anticholinergic caused a faster relief of nighttime ulcer pain ($p<0.05$). There was a significant correlation between healed ulcers and complete relief of ulcer symptoms ($p<0.05$). In the placebo group the ulcers of nonsmokers healed to a higher extent than those of smokers ($p<0.05$). Because the healing rates in the two treated groups were so high, calculations of the influence of smoking could not be performed. There was no difference in smoking habits of the patients who relapsed and those who did not. Time to relapse was, however, significantly shorter after treatment with cimetidine than after antacid/anticholinergic ($p<0.05$). Recurrences occurred more often ($p<0.05$) after slow healing (6-12 weeks) than after fast healing (3 weeks). (Auth. Abs. Mod.)

82–1411. Sulochana, G.; Arunagiri, R. **Smoking and Ascorbic Acid (Vit. C) Excretion.** *Clinician* 45(5): 198-201, May 1981.

It is known that vitamin C deficiency is connected with atherosclerosis and associated diseases; the incidence of atherosclerosis is greater in smokers. The immediate effect of smoking on the excretion of ascorbic acid in urine was studied in 50 healthy habitual smokers, and results showed that smoking significantly elevates the excretion of ascorbic acid in urine. The study was conducted after 12 hours of fasting to nullify the dietary influence. After the first urine collection, the subjects smoked five cigarettes within 1 hour, when a second sample was collected. All 50 subjects smoked the same brand of cigarette. The vitamin C content was significantly higher in the second sample of all the subjects. It is concluded that smoking causes tissue depletion of ascorbic acid, leading to increased risk of atherosclerosis and associated diseases. (Auth. Abs. Mod.)

See also, **82–1220, 82–1337, 82–1339**

82–1412. Taha, A.; Ball, K. P.; Illingworth, R. D. **Smoking and Subarachnoid Haemorrhage.** *Journal of the Royal Society of Medicine* 75(5): 332-335, May 1982.

Subarachnoid hemorrhage (SAH) due to cerebral aneurysm should be considered another smoking-related disease. The smoking habits of 198 patients who had survived an SAH were determined by questionnaire. Those with a cerebral aneurysm had on average smoked more per day than those with arteriovenous malformations, or those with normal angiographic findings whose smoking habits were similar to the United Kingdom population as a whole. This group also showed a higher prevalence of smoking. The men, but not women, had started smoking at an earlier age than the nonaneurysm patients. Increased risk from smoking was estimated at 3.7 for women and 3.9 for men. However, as SAH due to cerebral aneurysm is more common in women than men, other causative factors must be involved.

82–1413. Wanner, H. U. **Luftqualitat in Wohn- und Arbeitsraemen. [Air Quality in Residences and Places of Work.]** *Sozial- und Praventivmedizin* 25(6): 328-332, 1980, German.

Indoor air pollutants are reviewed and possibilities for their elimination are presented. The pollutants which measurably affect air quality are considered, specifically tobacco smoke components such as carbon monoxide, aldehydes, and particulate matter. In insufficiently ventilated rooms, these pollutants may attain concentrations which could have long-term injurious health effects, particularly for people with heart and circulatory troubles and children. Carbon monoxide concentration may reach 9 ppm, which is the threshold value for long-term effects. The nuisance criteria must also be considered in evaluating permissible concentrations for nonsmokers. It is advisable to limit smoking in public buildings to certain rooms. Elimination of tobacco smoke from indoor air requires extensive ventilation; various methods are reviewed. Air purification devices may also be helpful in eliminating cigarette smoke pollutants from heavily contaminated indoor air.

82–1414. Weber, A. **Passivrauchen-ein Risiko fur die Gesundheit? [Is Passive Smoking a Health Risk?]** *Deutsche Medizinische Wochenschrift* 107(9): 323-325, March 5, 1982, German.

Epidemiological surveys and research on risks of passive smoking to children, people with allergies, asthma, and chronic bronchitis, and healthy adults are reviewed. It is concluded that passive smoking can adversely affect the health of children and sick persons; that passive smoking presents an increased risk of lung cancer or respiratory symptoms in healthy adults remains undetermined; and that irritating effects and other subjective troubles, e.g., olfactory, may be expected in healthy persons at a carbon monoxide (CO) concentration of 1.2 to 2.3 mg/m^3 of air. Measures are suggested to protect passive smokers from higher CO concentrations.

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

82–1415. Aitken, P. P.; Davies, J. K.; Leathar, D. S. **Smoking Prevalence and Beliefs Among Adult Scots: Implications for Health Education.** *International Journal of Health Education* 24(3): 164-175, 1981.

A smoking survey of 918 adults in two areas of Scotland have yielded five major patterns of findings: (1) The gap between male and female smokers is decreasing. Comparisons with previous findings indicate that this is due to a decrease in the proportion of male smokers, whereas among female smokers little change has occurred. (2) There are more ex-smokers among males than among females. Although the proportion of ex-smokers among males increased with age, there were no age differences among females in this respect. Furthermore, the proportion of ex-smokers was lower than comparable estimates for the United Kingdom as a whole. (3) There were proportionally more smokers in the Glasgow/Edinburgh areas. However, this was almost entirely due to differences between respondents in socioeconomic groups. (4) There were relatively more smokers among lower socioeconomic groups. The social class gradient of smoking prevalence may be attenuated in areas outside the major urban areas. (5) Most of the respondents thought that there is a link between smoking and ill health. However, some groups differed with respect to the strength with which this belief is held. For example, respondents in the higher socioeconomic groups tended to express this belief more strongly. (Auth. Abs. Mod.)

82–1416. Aitken, P. P.; Leathar, D. S. **Adults' Attitudes Towards Drinking and Smoking Among Young People in Scotland.** Edinburgh, Her Majesty's Stationery Office, 1981, 144 pp.

Results of individual interviews of 918 adult Scots to determine adult and parental attitudes toward smoking and drinking in Scottish youth are presented and analyzed. Areas of inquiry included: (1) the visibility of underage smoking and drinking, (2) adult's perceptions of children's understanding of nonalcoholic vs. alcoholic drinks and drinkers, (3) when and by whom health education should begin, (4) perceived peer and parental influences, and (5) when and by whom children should be introduced to cigarettes and alcohol. Underage smoking and drinking are not considered major concerns relative to other concerns in adult daily living. Nine out of ten respondents felt parents should take the responsibility for telling youngsters about the effects of alcohol/smoking, followed by schools in order of preference. Most people had a pessimistic view of parental influence on children's future smoking or drinking habits: peer influences were deemed far more important. Most adults also felt children should not be allowed to try alcohol or cigarettes in the home, an attitude inconsistent with actual practice. The questionnaire responses are subdivided by age, sex, and socioeconomic status.

82–1417. Alcoholism and Drug Addiction Research Foundation. **Statistical Supplement to the Annual Report 1979-1980.** Toronto, Alcoholism and Drug Addiction Research Foundation, 1981, 295 pp.

Surveys conducted in the civilian, noninstitutionalized Canadian population aged 15 and over, show an increase in 1979 of the number of self-reported nonsmokers and ex-smokers and a decline in the number of smokers from 43 percent (1965) to 34 percent. Accompanying the decline in the percentage of regular smokers was a trend among smokers, other than those consuming over 25 cigarettes/day, to smoke fewer cigarettes/day. Statistical data indicate that the level of education is inversely related to smoking, particularly for males for whom 43.5 percent with partial or a completed elementary education were self-reported smokers compared with 24 percent of respondents with a university degree. Based on provincial government tobacco tax revenue, an estimated 60.1 billion cigarettes were sold in Canada during 1977-1978, representing an annual per capita consumption of 2,576 cigarettes or a daily consumption of 9.4 cigarettes for every person aged 15 and over. In addition to the tobacco data, the Supplement provides an overview of recent trends in the use of alcohol, psychotropic drugs, and caffeine in Canada, and in Ontario in particular.

82–1418. Baettig, K.; Buzzi, R.; Nil, R. **Smoke Yield of Cigarettes and Puffing Behavior in Men and Women.** *Psychopharmacology* 76(2): 139-148, 1982.

Puffing behavior (number of puffs, puff interval, puff duration, peak pressure, latency to peak pressure, average and total puff volume) was measured in 67 dependent male and 43 dependent female smokers when they smoked two cigarettes of their habitual brand under laboratory conditions. Test-retest reliability for the two cigarettes was high, and factor analysis showed that puff shape, puff volume, and puff frequency accounted for about 50 percent of variation obtained with the different puffing variables. Expiratory tidal carbon monoxide (CO) levels increased with the number of cigarettes smoked before the tests and with the intensity of the smoking habit, but pre- to postsmoking change in Δ tidal CO differences were similar for smokers of all types of cigarettes (0.1-1.7 mg standard machine smoking nicotine yield). Volume compensation for differences of smoke yield of the cigarettes was generally more pronounced in women than in men and, additionally, it was more pronounced for cigarettes with standard smoke nicotine yield below 0.9 mg than for cigarettes with standard smoke nicotine yield above 0.9 mg for both sexes. In women, partial correlation procedures suggested that nicotine might be more important in determining puffing behavior than CO and condensate yield, but there were also no women smoking the strongest cigarettes (1.3-1.7 mg nicotine yield). For both sexes, no compensation by adjusting the number of cigarettes smoked daily was obtained. Personality ratings, pulmonary functions, and cardiovascular functions were not, or were only inconsistently, correlated with puffing behavior or type of cigarette. (Auth. Abs.)

82–1419. Cameron, P.; Boehmer, J. **And Coffee Too.** *International Journal of the Addictions* 17(3): 569-574, 1982.

In a study of motives for trying and using coffee, tobacco, liquor, and marihuana, most tobacco users offered motives that indicated continued guilt/shame, while the majority of other users expressed contentment with their habits. In 272 interviews with people aged 22 to 80, to determine whether and why they had used any or all of the four substances, the attracting and capturing powers of tobacco and marihuana registered as lower than those associated with coffee and liquor. A guilt/shame index was derived from avowed motives, and it was found that trying liquor and tobacco was associated with greater guilt/shame than that associated with coffee and marihuana use. Since the psychological mechanisms associated with the trying and use of the substances apparently are different, it would appear that effective counterpropaganda should be tailored for each substance. Given the "capturing" effect of each, efforts directed toward getting persons (usually adolescents) not to try a substance might well prove the most effective in keeping them from becoming users. For "triers," clues to effective strategy might be found by interviewing those who tried but did not become users, and for "users," the reasoning of "quitters" might be useful in cessation strategies.

82–1420. Chapman, S.; Fitzgerald, B. **Brand Preference and Advertising Recall in Adolescent Smokers: Some Implications for Health Promotion.** *American Journal of Public Health* 72(5): 491-494, May 1982.

A survey on brand preference and cigarette advertising recall in 1,195 schoolchildren (220 smokers, 975 nonsmokers), aged 11 to 14, was conducted in Sydney, Australia. Four of the 130 available brands accounted for the cigarettes consumed by 78.7 percent of those who smoked. Smokers were nearly twice as likely to correctly identify edited cigarette advertisements and slogans than were nonsmokers. Young smokers who smoke "any" brand tend more toward the light-medium range of smoking rates than do smokers who are brand loyal. This suggests that part of the "career" of becoming a regular smoker is to adopt a preferred brand. Following from this, important implications may exist for antismoking education programs tailored to meet not simply the pressures to be a smoker, but those to be a certain sort of smoker. Health promoters would do well to incorporate the same principles of audience and market research used by tobacco companies to sell cigarettes, in the planning of their efforts to *unsell* smoking. (Auth. Abs. Mod.)

82–1421. Chassin, L.; Presson, C. C.; Sherman, S. J.; Corty, E.; Olshavsky, R. W. **Self-Images and Cigarette Smoking in Adolescence.** *Personality and Social Psychology Bulletin* 7(4): 670-676, December 1981.

Adolescents' self-perceptions (real or ideal), perceptions of their ideal date, and perceptions of stereotypic male and female smokers and nonsmokers are related to their current smoking status and their intentions to smoke. This conclusion results from an assessment of these perceptions in 175 9th and 10th grade students (69 males, 106 females). Subjects who smoked differed from nonsmokers in having real self-concepts and ideal dates that more closely matched the stereotypic smoker. Nonsmokers' real self-concepts, ideal dates, and aspects of their ideal self-concepts all predicted behavioral intentions concerning smoking, with nonsmoking subjects whose perceptions most closely matched the smoker stereotype being most likely to intend to smoke. It is noted that subjects' self-concepts (both real and ideal) were unrelated to their intentions to "try" a cigarette. Only ratings of an ideal date were related to such intentions. (Auth. Abs. Mod.)

82–1422. Cliff, K. S.; Grout, P.; Machin, D. **Smoking and Attitudes to Seat Belt Usage.** *Public Health, London* 96: 48-52, 1982.

Observations made during a pilot study of seat belt use suggested that nonusers were more commonly smoking cigarettes than those using seat belts. A questionnaire survey which evaluated a seat belt education program asked respondents for information on seat belt use, cigarette consumption, age, sex, driver or nondriver of a motor vehicle, opinion on legislation to enforce seat belt wearing, and opinion on a reduction in car insurance premiums for seat belt wearers. Allowing for age and sex among drivers, self-reported seat belt wearing declined as cigarette consumption rose ($p < 0.01$). Analysis of the data related to "opinions on legislation to enforce seat belt wearing" by means of logit-linear models indicated that age was the only variable which, when considered alone, was found to influence the response ($p < 0.05$). If the two variables, cigarette consumption and drivers, were added in an interactive mode, then there was a significant improvement in the fit as compared to age alone ($p < 0.05$). This improvement was not found with respect to "opinions relating to insurance premium reduction," age—sex being the only interaction which was significantly associated with the response ($p < 0.05$). (Auth. Abs. Mod.)

82–1423. Coe, R. M.; Miller, D. K.; Wolff, M.; Prendergast, J. M.; Pepper, M. **Attitudes and Health Promoting Behavior of Medical and Law Students.** *American Journal of Public Health* 72(7): 725-727, July 1982.

An entering class of medical students (n=152) is compared with an entering class of law students (n=217) on attitudes toward self-responsibility for health and reported health behaviors. Data on sociodemographic descriptors, physical activities, safety (use of seat belts), smoking habits, drinking behavior, and attitudes toward health promotion were collected by self-administered questionnaires and compared with results of a survey of practicing physicians and lawyers (*New England Journal of Medicine* 303(2): 104-107, July 10, 1980). Three times more law students (19 percent) than medical students (7 percent) reported being current smokers. However, the rate for law students is less than half of that for the general population in this age group. The prevalence of cigarette smoking was 14 percent for practicing physicians and 20 percent for lawyers. Results indicated that medical students were stronger on self-responsibility and also reported more often engaging in health promoting behavior than law students. (Auth. Abs. Mod.) See also 81-0580.

82-1424. Fishbein, M. **Social Psychological Analysis of Smoking Behavior.** In: Eiser, J. R. (Editor). *Social Psychology and Behavioral Medicine*, New York, John Wiley and Sons, 1982, pp. 179-197.

A social psychological theory, identified as a theory of reasoned action, is summarized and then applied to an analysis of smoking and nonsmoking behavior of young women. Results of some research on the smoking decision are also presented. The theory shows the causes of behavior as a series of successive steps. Behavior is assumed to be determined by intentions which are explained in terms of attitudes toward the behavior and subjective norms which are further explained in terms of beliefs about the consequences of performing the behavior. According to the theory, it is the person's belief about, and attitude toward, his or her own performance of the behavior that are directly relevant to the formation of intentions. Through surveys on smoking status, it was found that personal smoking intentions were strongly correlated with smoking behavior. The relative importance of personal attitudes and social norms varied across populations. Survey results suggest that normative pressure is effective in preventing the onset of smoking in grade school students, but not in young women. However, normative pressure is effective in encouraging cessation among young women, but not in convincing grade schoolers to quit. In exploring attitudes of women who planned to continue smoking (intenders) and women who did not (nonintenders), all the subjects acknowledged the negative health effects of smoking. However, intenders saw the health consequences as less negative, thus health education may still play a role in cessation. Also, intenders viewed the positive effects of smoking (easing social interaction, something to do with hands) as outweighing the negative health consequences, whereas the nonintenders viewed these same positive effects as a result of not smoking. Nonintenders were significantly more likely than intenders to comply with their doctors than with any other referent.

82-1425. Fox, B. H. **Endogenous Psychosocial Factors in Cross-National Cancer Incidence.** In: Eiser, J. R. (Editor). *Social Psychology and Behavioral Medicine*. New York, John Wiley & Sons, 1982, pp. 101-141.

Endogenous psychosocial factors—psychological factors (emotional, attitudinal, or ideational phenomena within an organism) or social factors (external factors which produce or influence these psychological factors) which affect biological states or responses such as cancer induction or cancer protection within the body—are examined. The differences between these endogenous psychosocial factors (EPF) and exogenous psychological and social factors—those factors that do not have their effect on psychic processes—are also discussed. The EPF are further divided into genetic and imposed factors (biological vs. behavioral). Imposed factors can be events such as presence or loss of family or social support, bereavement, psychosis, job security, interpersonal family friction, or cultural shock associated with migration. Cancer incidence rates within and between countries show that female rates differ less between countries, indicating men's greater exposure to local carcinogens: oropharyngeal (smoking and drinking), esophageal (smoking and drinking), lung (smoking), and bladder (smoking and occupational exposures). Where exposures tend to be the same, few differences are noted. The role of tobacco use in cancer incidence is evident in such places as India, where mouth cancer rates caused by chewing betel with tobacco and lime constitute 46 percent of all cancers. Lung cancer rates, where the excess of smoking in males has led to a male:female ratio of 4:1 to 6:1 over the last 40 years, are additional evidence. An extensive literature review of the subject is included, leading to the conclusion that EPF do exert small effects, both in promoting and protecting; that many people adapt to stress; and that the low level of EPF contributions to cancer in general explains the observed outcomes in ethnic and national groups.

82-1426. Goldberg, S. R.; Gardner, M. L. **Second-Order Schedules: Extended Sequences of Behavior Controlled by Brief Environmental Stimuli Associated With Drug Self-Administration.** In: Thompson, T.; Johanson, C. E. (Editors). National Institute on Drug Abuse Research Monograph 37, Department of Health and Human Services, Public Health Service, DHHS Pub. No. (ADM) 81-1137, July 1981, pp. 241-270.

In experiments with monkeys, the complexities involved with environmental conditioning related to drug abuse were demonstrated using fixed-ratio injections of morphine, cocaine, and nicotine. Second-order schedules of drug injections, requiring response patterns paired with periodic stimuli (amber lights) plus drug injection after a predetermined number of responses, were rapidly learned by the monkeys. The same schedules, presented without the secondary associated stimuli, resulted in a decline in the response rate. Stimulus presentations during extinction (of the drug injections) using a saline solution resulted in a rapid decline in response rates; however, if responding was first allowed to decline during a period of saline substitution, without stimulus presentation, the subsequent reintroduction of the stimulus temporarily restored response rates and patterns. Thus, previously drug-paired stimuli can retain their response-maintaining characteristics for a long period of time when they are not repeatedly presented during extinction. Such processes could play a significant role in human relapse to drug self-administration after prolonged periods of abstinence. Direct analogies to the control of human behavior are tenuous; nevertheless, second-order schedules resemble the conditions under which human behavior is maintained by drug self-administration. Drug injections are not continuously available, but are administered intermittently only after progression through a number of sequential stimulus changes and component behavior patterns. These complex patterns are extremely sensitive to manipulations of environmental variables, which is considered a promising field for future research in the treatment of drug addiction.

82–1427. Gredler, B.; Kunze, M. **Impact of a National Campaign on Smoking Attitudes and Patterns in Austria.** *International Journal of Health Education* 24(4): 271-279, 1981.

The impact of a recent antismoking campaign in Austria was evaluated by surveying a representative sample of 2,004 people aged from 16 to 69. The data show that the campaign had a widespread effect: 99 percent of those interviewed were aware of the campaign (83 percent referred to it spontaneously, and 15 percent on being questioned), and all Austrians knew the slogan "Life without smoking can be just as nice!" (88 percent mentioned it spontaneously). Furthermore, some 66 percent of respondents knew the campaign's symbol. Results show that the reduction in the number of smokers (34 percent) has been much greater than in 1979 when a similar survey indicated a decrease of 27 percent. Currently, 27 percent of the population describe themselves as smokers. Moreover, for the first time the number of women who smoke (22 percent) is diminishing. There also appears to be a trend toward smoking cigarettes with a low tar content. Finally, a considerable increase was recorded in the number of smokers who would like to quit. (Auth. Abs. Mod.)

82–1428. Griffiths, R. R.; Brady, J. V.; Bigelow, G. E. **Predicting the Dependence Liability of Stimulant Drugs.** In: Thompson, T.; Johanson, C. E. (Editors). National Institute on Drug Abuse Research Monograph 37, Department of Health and Human Services, DHHS Pub. No. (ADM) 81-1137, July 1981, pp. 182-196.

A review of animal drug self-administration methods and their usefulness in studying the dependence liability of psychomotor stimulant drugs is provided. In one study with baboons, nicotine, caffeine, saline, and various stimulant drugs were in turn substituted for cocaine. Mean levels of nicotine self-infusion were within the saline control range. A survey of other animal studies on nicotine maintenance via self-administration reveals equivocal results. Means of assessing drug dependence with animal studies, the applicability of these studies to humans, and the relationship between the reinforcing and anorectic properties of appetite suppressant drugs are also discussed.

82–1429. Hansen, W. B.; Evans, R. I. **Feedback Versus Information Concerning Carbon Monoxide as an Early Intervention Strategy in Adolescent Smoking.** *Adolescence* 17(65): 89-98, Spring 1982.

The efficacy of several programs using feedback about carbon monoxide (CO) as strategies for deterring experimentation with cigarettes was tested in sixth grade children. All experimental subjects viewed a film depicting elevated CO levels in smokers' breath. Subjects in three conditions then viewed a live demonstration of the detection process using smoking and nonsmoking adult subjects, followed by personal monitoring with either immediate, delayed, or withheld feedback. An additional group viewed only the film and the live demonstration, and one group viewed only the film. Compared to no-treatment controls, the only program to reduce experimentation with cigarettes was the information-only program. Contrary to what was predicted, several programs, including those using immediate and withheld feedback had increases in rates of experimentation. For young adolescent populations, the use of simple information about the imminent consequences of smoking is advocated. (Auth. Abs.)

82–1430. Hart, G.; Glover, J. **Smoking Behavior and Opinions of South Australians.** *World Smoking and Health* 7(1): 32-37, 42, Spring 1982.

The smoking behavior and opinions of South Australians above age 15 were surveyed. In middle-age groups (30-60 years) smoking is more common among males, who tend also to be heavier smokers than females. The similarity in rates at younger age levels results from similar proportions of men and women taking up smoking, whereas in older age groups the similar prevalence results from the high proportion of men who are ex-smokers. Cigarette use is more common among the unemployed, particularly females, and there is an inverse relationship between smoking and exercise. The most frequent reason given for quitting was social influence from friends. There was strong support for antismoking activities, even among smokers, with the greatest support occurring in issues affecting children. Despite awareness of the potential harm of smoking in pregnancy, few women smokers quit in their most recent pregnancy and about half did not change their smoking habits. (Auth. Abs. Mod.)

82–1431. Henningfield, J. E.; Griffiths, R. R.; Jasinski, D. R. **Human Dependence on Tobacco and Opioids: Common Factors.** In: Thompson, T.; Johanson, C. E. (Editors). National Institute on Drug Abuse Research Monograph 37, Department of Health and Human Services, Public Health Service, DHHS Pub. No. (ADM) 81-1137, July 1981, pp. 210-234.

Despite the fact that cigarette smoke and opioid drugs appear to produce vastly differing pharmacological and behavioral effects, certain functional similarities are shown to exist, adding further support to the notion that cigarette smoking is an instance of drug dependence. In comparing these similarities, relevant experimental data, clinical observations, and epidemiological findings are discussed relative to patterns of use, personality characteristics and social factors, physiological dependence, deprivation effects, tolerance, dose effects on drug intake, reinforcing efficacy and dependence liability, response requirement, conditioning factors, antagonist administration effects, preloading effects, relapse patterns following abstinence treatment, and feeding behavior effects. Although some of the common functional relationships reviewed are not uniquely shared by cigarette smoking and opioid dependence, others are less widely shared with most other forms of drug or substance abuse, including the similarities noted in the pattern of chronic daily use, receptor specificity, and the role of physical dependence.

82–1432. Hill, D. J.; Gray, N. J. **Patterns of Tobacco Smoking in Australia.** *Medical Journal of Australia* 1(1): 23-25, January 9, 1982.

For the first time in a national survey of Australian smokers, more individuals of an age-sex group (males aged 20 to 24 years) smoked cigarettes than did not smoke. A representative sample of 4,309 people (2,137 men and 2,172 women) aged 16 years and over was interviewed in the winter of 1980. As in previous surveys, cigarette smoking rates in both sexes reached a peak in young adulthood (twenties), fell during the thirties and early forties, peaked around 50 years of age, and fell to a low rate in old age. Overall, 40 percent of men and 31 percent of women smoked cigarettes, the same percentages as in 1976. The highest rate of cigarette smoking (56 percent, up 10 percent) was among men aged 20 to 24 years. There was a distinctly negative relationship between smoking and occupational and educational levels for men, but not for women. Special health education strategies are urgently needed for those age groups in the adult population in which smoking peaks—the twenties and late forties. (Auth. Abs. Mod.)

82–1433. Janzon, L.; Lindell, S.-E.; Trell, E. **Smoking and Disease: Attitudes and Knowledge in Middle-Aged Men.** *Scandinavian Journal of Social Medicine* 9(3): 127-133, 1981.

In a general health screening program in Malmo, Sweden, 1,037 men (517 smokers, 290 ex-smokers, 230 nonsmokers), 47 to 48 years old, were questioned on smoking habits, whether they believed in an association between smoking and cancer of the lungs, mouth, larynx, and bladder, and cardiovascular and respiratory diseases, and whether they believed that by giving up smoking they would reduce the risk. The association between smoking and disease was recognized by more ex-smokers than smokers. Less than half of the smokers believed in an association between smoking and the diseases listed in the questionnaire. The prevalence of dyspnea, chronic bronchitis, and asthma increased with increasing tobacco consumption as did the proportion using sleeping pills and the percentage with at least three episodes of disability the previous year. Fifty percent of all smokers wished to stop smoking but only 5 out of 10 were convinced of an improvement in health by quitting. The results show a need for well formulated information as to the health hazards of smoking and the benefits of giving it up. (Auth. Abs.)

82–1434. Jessor, R. **Problem Behavior and Developmental Transition in Adolescence.** *Journal of School Health* 52(5): 295-300, May 1982.

Problem behaviors in adolescents, such as smoking, drug and alcohol abuse, and aggression, should be accepted as purposeful, goal-oriented, and functional in the developmental transitions to adulthood. Such behavior can serve as a way of expressing identification with the youth subculture: cigarette and marihuana use often serve as a mark of peer group membership, and as a way to be "tough," "macho," "experienced," "sexy," or "cool." Such behavior may also be a means of affirming one's maturity and placing a claim upon occupying adult status. The prevalence of certain problem behaviors is changing, however, as sexual activity and alcohol and marihuana use are increasing while smoking is decreasing (21 percent of the class of 1980 smoked on a daily basis vs. 27 percent of the class of 1975). Also, the male/female differences in participation rates are disappearing, and in the case of smoking, are now reversed. It is recommended that nonproblem behaviors leading to the same goal be reinforced or substituted for the problem behaviors; or where prevention is not realistic, minimizing the involvement, or insulating against the negative effects of the behavior may help. A final strategy would be to delay the onset of such behaviors until the adolescent is more skilled to handle the activity.

82–1435. Johnston, L. D.; Bachman, J. G.; O'Malley, P. M. **Highlights From Student Drug Use in America 1975-1981.** U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHHS No. (ADM) 82-1208, 1981, 130 pp.

This is the fifth in an annual series reporting the drug use and related attitudes of high school seniors in the United States. This report covers the high school classes of 1975 through 1981, and supersedes the previous reports. Reported prevalence and trends in drug use among high school seniors show 71 percent had tried cigarettes, 29 percent had smoked in the past month, and 13.5 percent smoked a half-pack or more per day. Daily cigarette use was reported more often than any other drug, with slightly more females (13.8 percent) smoking than males (12.8 percent). The largest difference was related to college plans (8 percent college-bound vs. 21 percent noncollege-bound). No differences relating to urbanity were found, but regional differences were apparent; the Northeast (17 percent) was highest, followed by the North Central and South, with the West being lowest (7 percent). The decline in cigarette use is decelerating, showing only a 1.0 percent drop in the past year, with use among males dropping more than females. Fifteen percent of the seniors reported initial use prior to 10th grade; 9 percent began smoking in grades 10-12. Regular use of cigarettes ($\geq$one pack/day) is judged by 63 percent of the seniors as entailing a great risk of harm for the user. The majority (74 percent) perceived their friends as disapproving of smoking a pack or more daily, while 70 percent themselves disapproved of smoking, and 43 percent believe smoking should be prohibited by law. Other categories of drug use reported are alcohol, marihuana (including hashish), inhalants, hallucinogens, cocaine, heroin, other opiates, stimulants, sedatives, and tranquilizers. See also 82-0551.

82–1436. Kannas, L. **The Dimensions of Health Behavior Among Young Men in Finland.** *International Journal of Health Education* 24(3): 146-155, 1981.

In an analysis of the cumulative vs. multidimensional nature of health behavior, smoking was found to be a "key" habit and predictive of behavior in other health areas. A questionnaire survey of 880 19-year-old men of Finland was conducted and the relationships between the different health habits were studied. The findings revealed a multidimensional structure, with specific health behaviors correlated in five dimensions: (1) use of sugar and fat; (2) alcohol, smoking, and sexual activity; (3) active health-oriented behavior; (4) eating and resting habits; and (5) dental care. Cigarette use correlated with drug, alcohol, coffee, chewing gum, and excessive fat and sugar consumption. Smoking was also associated with irregular eating habits, minimal consumption of fruit and milk, and greater sexual activity. Smoking did not correlate, however, with physical exercise, interest in sports, good dental hygiene, or visits to a doctor. These findings indicate that emphasis should be placed on encouraging healthful lifestyles rather than attempting to change one habit at a time.

82–1437. Kellam, S. G.; Brown, C. H.; Fleming, J. P. **Social Adaptation to First Grade and Teenage Drug, Alcohol and Cigarette Use.** *Journal of School Health* 52(5): 301-306, May 1982.

First grade children, assessed for social adaptation, psychological well-being, learning problems, and aggressiveness, were reinterviewed 10 years later to study the relationships of these factors to teenage substance abuse. The subjects were from Woodlawn, a black Chicago ghetto; and data were collected from appropriate sources such as first grade teachers, clinical observations, interviews with mothers, and questionnaires administered to the teenagers. It was found that learning problems in first grade did not predict teenage substance abuse. However, aggressiveness in males in first grade leads to 1.5 times more use by teenagers of beer or wine, hard liquor, marihuana, and cigarettes compared to nonaggressive males. Shyness without aggression in first grade leads to an inhibition of both cigarette and marihuana use by teenagers. While shyness without aggression appears to decrease use, shyness in the presence of aggression seems to enhance use. In fact, the moderate/severe shy in combination with the moderate/severe aggressive first grade males show the highest substance use in three of the four substance categories 10 years later. This effect is most clear for cigarettes and marihuana. First grade ratings of psychiatric symptoms were unrelated to male substance use 10 years later but did predict teenage cigarette use among females. (Auth. Abs. Mod.)

82–1438. Kunzendorf, R. G.; Denney, J. **Definitions of Personal Space: Smokers Versus Non-Smokers.** *Psychological Reports* 50: 818, 1982.

Smoking serves to expand personal territory and people who smoke desire more personal territory than nonsmokers. This conclusion resulted from a study relating the behavior of smoking and the definition of personal space. Each of 47 university students (smokers and nonsmokers) sat on an isolated chair and was instructed to tell an approaching experimenter to stop when he came "too close." The experimenter approached each subject twice, once without a cigarette and once while smoking. Each student completed a questionnaire regarding his own smoking behavior. The results confirmed that individually tested subjects desire more personal space in the company of a smoking experimenter than in the company of a nonsmoking experimenter, and that subjects who were smokers, in general, desire more personal space than subjects who were nonsmokers.

82–1439. Lambert, C. A.; Netherton, D. R.; Finison, L. J.; Hyde, J. N.; Spaight, S. J. **Risk Factors and Life Style: A Statewide Health-Interview Survey.** *New England Journal of Medicine* 306(17): 1048-1051, April 29, 1982.

To determine choices of lifestyle identified as risk factors for serious illness, a survey was made of the health-related behavior of 1,091 adults in Massachusetts. Four basic lifestyle factors are analyzed: exercise, smoking, weight, and alcohol use. Overall, the smoking prevalence rate is 33 percent with smoking being most pronounced among the 25- to 44-year-old group and least pronounced among persons 65 to 90. Women 25 to 44 smoked at the highest rate (40.8 percent) of any age and sex category. Results of the analysis showed women reporting more high-risk behavior than men in all areas except alcohol consumption, although the difference in smoking prevalence was not considered significant. Older persons reported less smoking and alcohol use as did persons of higher educational attainment, and to a lesser extent those with higher incomes.

82–1440. Lotecka, L.; Lassleben, M. **The High School "Smoker": A Field Study of Cigarette-Related Cognitions and Social Perceptions.** *Adolescence* 16(63): 513-526, Fall 1981.

A field study of the cigarette smoking behavior of adolescents was conducted. Both intrapersonal and interpersonal communications were examined utilizing individual and group investigations of free, cued, and group stimulated responses. A quasi-phenomenological approach (emphasizing eidetic and experiential perceptions) was used to elicit, describe, and summarize thematic content of the perceptions of high school smokers, ex-smokers, and nonsmokers. This content was used in group interchanges with smokers to generate disclosure of intentional and situational aspects of smoking decisions. Cognitive, imaginal, affective, and social differentiations emerged presenting a profile of the identified smoker as more anxious and problematical. Prevalent self-statements were explicated. Complex requirements were implied for individualistic approaches to personal coping/control skills development. It is concluded that tobacco is a form of drug dependence no less strong than that of other addictive drugs. Further studies of personal coping/control strategies and choice points in decisionmaking among core group smokers are recommended. (Auth. Abs. Mod.)

82–1441. Metzner, H. L.; Harburg, E.; Lamphiear, D. E. **Residential Mobility and Urban—Rural Residence Within Life Stages Related to Health Risk and Chronic Disease in Tecumseh, Michigan.** *Journal of Chronic Diseases* 35(5): 359-374, 1982.

Residential history, diagnosis of three chronic diseases, five clinical measurements, and histories of smoking and drinking were obtained from a sample of 615 women and 529 men, aged 35-69, randomly selected from respondents of the Tecumseh Community Health Study. Two measures of residential mobility and one of urban—rural residence, during early life stages and over the entire lifetime, were compared to subsequent adult health traits. Both length of stay in a residence and age during stay were associated with greater prevalence of coronary disease and with behavior patterns, namely cigarette smoking and drinking, that are risk factors for certain chronic diseases. Long stays in rural places were associated with both lower cigarette consumption and fewer women and older men who had ever smoked. Greater residential mobility, particularly in childhood but also in later life, was significantly associated with increased cigarette and alcohol consumption, greater prevalence of hypertension, and higher mean diastolic blood pressure in older persons. Greater duration of urban residence was associated with greater prevalence of chronic bronchitis. (Auth. Abs. Mod.)

82–1442. Murphy, D. C. **A Realistic Method of Health Hazard Appraisal for the Occupational Health Nurse.** *Occupational Health Nursing* 30(1): 11-16, January 1982.

To improve the services of the occupational nurse in a factory, approximately 300 employees were surveyed for smoking and drinking habits, demographic factors, and occupational chemical exposure. A sample of 38 employees who had at least 5 years' worth of laboratory data in company medical files were further analyzed with respect to blood parameters. In the small sample, hematocrit increased as tobacco exposure increased with a dose—response effect. A comparison of smoking habits with salary level revealed a higher rate of smoking among blue collar workers than white collar workers. Smoking also showed a positive association with accident rate. Implications of the work force profile, and the epidemiological approach to health care for the occupational nurse, are discussed.

82–1443. Nellis, M. J. **An Interresponse Time Analysis of Smoking in the Natural Environment.** Doctoral Dissertation, University of Chicago, Chicago, Joseph Regenstein Library Department of Photoduplication, Thesis No. T27976, June 1981, 138 pp.

Results of a smoking intervention study suggest that the differences in individual patterns of smoking may reflect differences in the variables controlling smoking in the subject's natural environment. Previous behavioral treatment programs have assumed smoking behavior to be a unitary phenomenon with treatment directed toward eliminating the behavior. Such treatments have generally attained immediate behavior change through a wide variety of intervention procedures, but the majority of subjects resume smoking at some point following completion of treatment. The present study of 16 smokers assumed that smoking, like any other complex pattern of behavior, might be related to different variables at different times, depending on events in the subject's natural environment. Following 2-week baseline reports of smoking which included the time each cigarette was smoked and the interval between smoking, subjects were individually counseled on behavioral intervention strategies. A quantitative index examined the change in rate of smoking from one cigarette to the next, and calculated a proportion of the time during which little or no change took place. Post hoc inferences were made on the basis of the nature of the observed smoking patterns and the nature of differences in strategies employed in the individual intervention programs. It is suggested that noting the differences in the variables controlling individual smoking patterns may prove helpful in suggesting specific interventions. The implications of these conclusions for smoking research and intervention are discussed.

82–1444. Newman, I. M.; Martin, G. L.; Irwin, R. P.; Ang, J.; McNulty, L. J. **Adolescent Cigarette Smoking in Three Societies: An Approach to Educational Planning.** In: *Man, Drugs, and Society—Current Perspectives.* Proceedings of the First Pan-Pacific Conference on Drugs and Alcohol, Canberra, Australia, February 26-March 5, 1980. Canberra, Australian Foundation on Alcoholism and Drug Dependence, 1981, pp. 248-255.

Fishbein's Model of Behavioral Intention was applied in questionnaire studies to define attitudinal and normative factors associated with adolescent cigarette smoking. Questionnaire A was developed on the basis of current smoking literature, and sampled populations in the United States and Australia. Questionnaire B was modeled after Fishbein's theory and administered to U.S. and New Zealand students. The subjective norm was prominent in the New Zealand sample: nonsmokers perceived pressure from peers and relatives not to smoke, whereas smokers did not perceive such pressure. In the United States both smokers and nonsmokers described parents as being opposed to the habit. Smokers in the United States wanted more strongly to comply with their friends than nonsmokers. Both U.S. and Australian smokers and nonsmokers differed in their beliefs about the consequences of smoking. All smokers were more likely to believe that smoking was enjoyable and relaxing than nonsmokers. No differences in the components of subjective norm occurred between smokers and nonsmokers in either the U.S. or Australian sample.

82–1445. Office of Population Censuses and Surveys. **General Household Survey. Preliminary Results for 1980.** U.K. Government Statistical Service, Office of Population Censuses and Surveys, GHS 81/1, June 16, 1981, 16 pp.

Smoking data from the 1980 General Household Survey (Great Britain) show that cigarette consumption between 1978 and 1980 has continued to decline among men (from 45 percent to 42 percent) but remained stable at 37 percent for women. The declines reflect fewer light smokers (< 20 cigarettes/day) for the period between 1972 and 1980, and fewer heavy smokers among men since 1974. The proportion of women who are heavy smokers has remained fairly constant. Figures for 1980 confirm a higher smoking prevalence in the manual than in nonmanual occupations, and while the drop in smoking between 1972 and 1980 occurred in most socioeconomic groups, it was most pronounced among professionals and least pronounced among unskilled women. Other areas covered in this report of preliminary survey results include: household composition, color, one-parent families, home ownership, consumer durables, labor participation (and education), working mothers, and unemployment. The results are based on personal interviews in about 15,000 private households throughout Great Britain.

82–1446. Peters, R.; McGee, R. **Cigarette Smoking and State-Dependent Memory.** *Psychopharmacology* 76(3): 232-235, March 1982.

Results of experiments clearly indicate that cigarette smoking can produce a state-dependent learning (SDL) effect on recall performance in humans. First, an experiment was conducted to select an appropriate control cigarette for use in an SDL design. A low-nicotine content (0.2 mg) cigarette was chosen as it did not appear to affect the physiological arousal of the subjects. In the second experiment, performed to determine cigarette smoking's effect on SDL, recognition memory was poorest when the material had been learned after smoking the 1.4 mg nicotine cigarette; second day recall was highest among those smoking the low-nicotine cigarettes. Nicotine content had no effect on immediate memory. It is concluded that the most likely basis for the results is the arousal produced by the nicotine content of the cigarette. (Auth. Abs. Mod.)

82–1447. Pihl, R. O.; Marinier, R.; Lapp, J.; Drake, H. **Psychotropic Drug Use by Women: Characteristics of High Consumers.** *International Journal of the Addictions* 17(2): 259-269, 1982.

A sample of 1,187 Montreal women were interviewed concerning their use of psychotropic medication, health and visits to physicians, use of tobacco and alcohol, and lifestyle and general satisfaction. Subjects were categorized as high, moderate, and nonconsumers of psychotropes: respectively, 5.5 percent, 22.6 percent, and 71.9 percent of the sample. High drug consumers typically were older, reported being in poorer health, visited physicians more frequently (and more frequently for undefined reasons), rated themselves as more unhappy, were less well educated, were more likely unemployed, smoked more but drank less alcohol, and were more likely to have children than the moderate users or abstainers. The high degree of smoking in the high drug use group may be an indication of the contribution of smoking to illness. The high users visited physicians primarily for hypertension, nervousness, and fatigue—the symptoms reported by heavy smokers. The findings tend to support the hypotheses that social and psychological problems of women are expressed vaguely, and thus are more likely medicalized than those of men. The implications of these findings for drug prevention programs and future research are discussed. (Auth. Abs. Mod.)

82–1448. Roberts, E. B.; Homer, J.; Kasabian, A.; Varrell, M. **A Systems View of the Smoking Problem: Perspective and Limitations of the Role of Science in Decision-Making.** *International Journal of Bio-Medical Computing* 13(1): 69-86, 1982.

The complex issues and relationships surrounding the smoking problem indicate the desirability of a system dynamics computer simulation model for policy development and analysis. An initial model-building effort is described, including results of initial policy and sensitivity testing. The model focuses on the interactions which affect the initiation and quitting of smoking. The population is divided into three levels: potential smokers (all nonsmokers above age 12), smokers, and ex-smokers. The key variables measured are the proportions of the population in each level, the rate of mortality, and the rate at which individuals leave one level and join another. The quit and initiation rates are governed by interactions such as antismoking campaigns, societal approval of smoking, perceived health hazards of smoking, and tobacco industry spending. For instance, the smoking initiation rate is decreased by restrictions on smoking, perceived hazards to health, antismoking campaigns, and the price of cigarettes. It is increased by increases in societal approval of smoking and tobacco industry promotions. The lack of scientific research on most of the relationships and parameters forced heavy reliance upon intuition in the model development. The sensitivity of simulated model outcomes to many of these assumptions demonstrates the need for more research if forecasting and policy determination are to be carried out with confidence. (Auth. Abs. Mod.)

82–1449. Ruznisky, S. A.; Thauberger, P. C.; Cleland, J. F. **Rejection and the Use of Chemical Agents.** *Social Science and Medicine* 15E(3): 205-210, August 1981.

The Avoidance of Rejection Scales (of self, by others, and of others) were specifically developed to assist the psychometric documentation of choice of an avoidance vs. confrontation strategy in the issue of rejection. Three kinds of rejection were examined concurrently with respect to the use of seven chemical agents related to health and social behavior: tobacco, alcoholic beverages, tranquilizers, medication, stimulants, soft drugs (such as marihuana), and hard drugs (such as heroin). An analysis of 300 individuals revealed that low scorers (confronters) reported less use of chemical agents in 15 of the 18 cases where a posteriori comparisons among high, medium, and low scorer groups indicated statistically significant differences. Rejection scales for tobacco use did not show significant levels in any of the groups—of self, by others, or of others. Some indication of curvilinearity was indicated in five of the statistically significant comparisons. In addition, the strongest linearity between the Rejection and Use of Chemical Agents scales appeared in the following descending order: of others, of self, and by others. These results suggest a value to awareness strategies, though not in all cases. (Auth. Abs. Mod.)

82–1450. Spencer, J. **Why Nurses Smoke.** *Nursing Mirror* 154(1): 26-29, January 6, 1982.

A pilot survey of 171 nurses (75 smokers and 96 nonsmokers) shows that the percentage defined as being present smokers (44 percent) seems to conform to the high level of smoking behavior generally found within the nursing profession. Results reveal that more present smokers worked during the night, had smoked at least one cigar, lived with other smokers, and had parents who presently smoked. In relating stress to the nurses' smoking behavior, eight questions were asked concerning: patient's deaths; inadequate staffing levels; contact with doctors; eating in the canteen; shift work; night duty; lack of time; and working with other nursing colleagues. No differences were found between smokers and nonsmokers. Also discussed are age factors, religious factors, type of nursing, cigarette consumption per day, and age when respondents began smoking.

82–1451. Stachnik, T. J.; Stoffelmayr, B. E. **Is There a Future for Smoking Cessation Programs?** *Journal of Community Health* 7(1): 47-56, Fall 1981.

Given the marginal success rates of formal smoking cessation clinics and the small number of smokers who have participated in such clinics, it must be concluded that their impact on the public health problem of smoking has been negligible. To improve the performance of antismoking programs, the following changes are suggested: (1) clinics should be offered at existing organizational units, e.g., workplaces, where all the smokers can be readily informed about the program and also provide mutual support for nonsmoking; (2) an immediate inducement (money) should be made available to smokers for successful participation in the clinic; (3) a pervasive social support system should be employed to help the smoker with the difficult problem of maintaining abstinence after it has been achieved; (4) because of the large number of smokers, highly trained personnel should not be necessary (programs should be designed to be administered by employers, local clubs, or churches); and (5) duration of the programs should be extended until the habit has weakened considerably, and provisions made for maintenance of nonsmoking after the program has ended. A proposed program is discussed which meets these criteria, and which may also apply to other health behavior factors, such as obesity, hypertension, and serum cholesterol levels. (Auth. Abs. Mod.)

82–1452. Stewart, P. J.; Rosser, W. W. **The Impact of Routine Advice on Smoking Cessation From Family Physicians.** *Canadian Medical Association Journal* 126(9): 1051-1054, May 1, 1982.

In a controlled trial to measure the impact of physicians' advice to cigarette smokers during a routine office visit, no significant effects were noted. Physicians had been asked to advise all their patients on the hazards of smoking. However, after two followup periods, no significant differences were found in the three measures used to determine outcome—desire to stop smoking, an attempt to stop, and success in stopping—between the control and intervention groups. This suggests that routine advice may not be the most effective way for a physician to promote nonsmoking. Although the advice was effective in increasing motivation, it did not increase the rates of success. As many smokers lack the resources they need to change their behavior, physicians should not rely solely on a few words of advice; self-help kits, referral to cessation programs, or continuous followup may also be warranted. (Auth. Abs. Mod.)

82–1453. Stransky, M. **Tabak- und Alkoholkonsum bei Jungen Maennern. [Tobacco and Alcohol Consumption of Young Men.]** *Ernaehrungs-Umschau* 28(11): 376-378, November 1981, German.

One hundred and sixty-six Swiss male draftees, 20 years old, were interviewed about their smoking habits. The results show that: (1) 46.4 percent of the subjects smoked cigarettes and only 1.8 percent consumed other tobacco products; (2) 23.4 percent of the smokers consumed over 20 cigarettes/day (heavy smokers) and 91 percent of cigarette smokers inhaled; (3) half of the smokers had begun smoking at the age of 16 or 17, i.e., right after graduation from school; and (4) 57 percent of the draftees considered smoking dangerous to health, while another 31 percent agreed with this statement only under certain conditions. These results generally agree with those of two previous Swiss surveys except that in those surveys more users of tobacco products other than cigarettes were found. Also, the percentage of heavy smokers was higher. A shift toward starting smoking at a younger age was noted in comparison to a survey taken 8 years ago.

82–1454. Streja, D. A.; Boyko, E.; Rabkin, S. W. **Predictors of Outcome in a Risk Factor Intervention Trial Using Behavior Modification.** *Preventive Medicine* 11(3): 291-303, May 1982.

In both univariate and multivariate analysis, attrition from a weight loss program was significantly associated with cigarette smoking, higher body weight, larger desired weight loss, and psychiatric history. To evaluate factors related to successful completion of a behavior modification program aimed primarily at eating habits, 100 volunteers (82 women and 18 men), 19 to 64 years in age, took part in a 20-week program. Smoking was highly correlated with attrition from the program, with present smokers having a higher rate than former smokers. Smoking history also differed with onset of obesity: more smokers were in the adolescence-onset and adult-onset groups than the childhood-onset group. In the 55 subjects who completed the program, significant ($p<0.05$) decreases in weight, skinfold thickness, systolic and diastolic blood pressure, total serum cholesterol, and uric acid were observed. Changes in HDL cholesterol and triglycerides were not significant. Actual weight loss in subjects who completed the program was significantly greater in those with a modest weight loss goal, and in married subjects perceiving their spouses as supportive or overweight. The findings also suggested that former smokers are more likely to be concerned about their future health and to comply with health behavior change programs. (Auth. Abs. Mod.)

82–1455. Sutton, S. R. **Fear-Arousing Communications: A Critical Examination of Theory and Research.** In: Eiser, J. R. (Editor). *Social Psychology and Behavioral Medicine.* New York, John Wiley and Sons, 1982, pp. 303-337.

Theories and findings on the effect of fear-arousing communications in health behavior change are reviewed. Theories discussed include the fear-drive model, nonmonotonic models, and Leventhal's parallel response model. Findings from studies published between 1953 and 1980, are summarized. Areas examined are the main and nonmonotonic effects of fear, efficacy of the recommended action, the role of specific instructions, communicator and recipient factors, and other elements. Some of these findings show that heavy smokers tended to be less frightened of health risks than light smokers, though there were no differences in intention to stop smoking between the two groups. Also, resistance is often aroused when individuals perceive a threat they feel unable to avoid: a high-fear approach combined with low reassurance resulted in disbelief that smoking causes lung cancer. Conversely, subjects given high reassurance/high fear presentations were smoking less after 5 weeks than the low reassurance group. A cognitive decisionmaking model based on SEU (subjective expected utility) theory is proposed as an alternative to these models. Studies based on this theory show that when stopping is perceived as highly effective in removing the health threats, intentions to stop/reduce smoking were increased.

82–1456. Thomas, E. M. **How Do Pharmacists Feel About Smoking? The Preliminary Results of a 1979 Survey.** *Canadian Pharmaceutical Journal* 113(12): 402-404, December 1980.

Smoking rates among 963 Canadian pharmacists surveyed in 1979 were very low (19 percent) and those who did smoke used fewer cigarettes and lighter cigarettes than the general public. Responses to the survey questionnaire showed that as a group, the respondents were highly motivated to act as health promoters and educators. Smoking was not permitted by employees in the pharmacy but customers could smoke. Pharmacists sold and displayed tobacco products to attract customers rather than for direct profit. Though respondents were aware of some smoking-related problems, such as lung cancer (78 percent), heart disease (67 percent), and pregnancy complications (59 percent), in other areas (e.g., bladder cancer) awareness was low. Few claimed at least some knowledge of the economic aspects of smoking (56 percent), smoking and the nonsmoker (55 percent), smoking habits of Canadians (30 percent), and smoking and the law (25 percent). Most were unaware that it is illegal to sell tobacco to children. Responses identified possible topics for information and education projects.

82–1457. Vener, A. M.; Krupka, L. R.; Climo, J. J. **Drugs (Prescription, Over-the-Counter, Social) and the Young Adult: Use and Attitudes.** *International Journal of the Addictions* 17(3): 399-415, 1982.

A study of the total drug use (prescription, over-the-counter (O-T-C), and social), of 600 college students (310 males, 290 females), 18-21 years of age, demonstrated that 75 percent had ingested alcohol, 91 percent caffeine, 41 percent analgesics, 28 percent vitamins, and 17 percent prescription medicines, and 39 percent had smoked marihuana and 21 percent ordinary cigarettes during the week prior to interview. Women used more prescriptions, O-T-C, and social drugs than men, with women averaging 5.1 percent and men 4.5 percent. Relative to cigarette smoking, a greater percentage of women (27 percent) were smoking than were men (16 percent). Less than 1 percent of the students selected nicotine from five selected drugs as the most dangerous to society and only 4 percent selected it as the most dangerous to the biological system. Responses to a question regarding their willingness to marry a user of drugs showed that 41 percent of the men and 29 percent of the women were unwilling to marry cigarette smokers. The findings suggest that drug education programs need to place more emphasis upon the hazards associated with O-T-C and social drugs. (Auth. Abs. Mod.)

82-1458. Wheatley, G. **Smoking Habits of Canadians 1965 to 1979.** Health and Welfare Canada, Health Services and Promotion Branch, Health Promotion Directorate, Technical Report Series No. 9, December 1980, 50 pp.

The smoking habits of Canadians aged 15 and over, based on the results of the Health and Welfare Canada December 1979 survey, are reported. Data from 1965 to 1979 indicate a continuation of earlier trends, with an almost 2 percent decline in the number of regular cigarette smokers since 1977 (9 percent since 1965). Only one in three Canadians are now regular smokers, and nonsmokers now constitute 60 percent of the population. The number of ex-smokers has risen to 15 percent; occasional smokers remain at less than 4 percent; pipe and/or cigar smokers, about 2 percent. The most significant finding was the level of tar exposure of regular cigarette smokers, down 10 percent since 1977 (8 percent for males, 12 percent for females). Smokers of the high-tar and -nicotine brands declined from 43 percent in 1977 to only 18 percent in 1979; low yield brand smokers increased from 18 percent in 1977 to 31 percent in 1979. Since 1965, twice as many males as females have quit smoking and regular male smokers have decreased 16 percent compared to only a 1 percent decrease for females. In the 15- to 19-year-old age group, male regular cigarette smokers have increased slightly while females have increased so that now both are at similar levels; 26.8 percent for males, and 26.4 percent for females.

82-1459. Wilson, R.; Iverson, D. **Federal Data Bases for Health Education Research.** *Health Education* 13(3): 30-34, May-June 1982.

Health education research and resources useful to educators are reviewed. An outline for sampling general research areas is suggested, consisting of predisposing factors, enabling factors, reinforcing factors, behavior problems, and health status. Within this plan, a health behavior such as smoking could be researched fully as to onset of smoking, frequency, type of cigarette smoked, pattern of smoking, and the associated health data. Federally sponsored sources of data discussed include the Hypertension Detection and Follow-Up Program (HDFP), the Primary Prevention of Hypertension study, the Multiple Risk Factor Intervention Trial (MRFIT), Health Services Administration, National Heart, Lung, and Blood Institute (NHLBI), National Center for Health Statistics (NCHS), Food and Drug Administration, National Institute on Alcohol Abuse and Alcoholism, Centers for Disease Control, National Institute on Drug Abuse (NIDA), Department of Agriculture, and the Office of Smoking and Health.

See also, **82-1264, 82-1474, 82-1523**

SMOKING PREVENTION AND INTERVENTION

82-1460. Abelin, Th.; Beck, R. **Preventing Smoking in Switzerland.** *World Smoking and Health* 7(1): 22-26, Spring 1982.

Peculiarities of Switzerland's history and governmental structure serve as an obstacle to smoking prevention and education, and protect the tobacco industry. In 1973, the AT (AT-Swiss Interagency Council on Smoking and Health) was founded and is now fighting tobacco use with a five-step concept, involving information, publicity, motivating individuals to stop or not start smoking, assistance in quitting, and political activity. The AT compiles all smoking information and publishes an index for educators; the Swiss information media publicizes smoking information. Doctors and health service personnel are encouraged to influence quitting, and smoking is treated as a physical dependence. Politically, the AT balances the influence of the tobacco industry, and recently, restrictions on advertising, warnings on packages, declaration of content of cigarettes, and nonsmoking areas have been implemented. Smoking in Switzerland cost 560 to 800 million Swiss francs in 1976: when a 37-year-old man quits, an economic benefit of 23,000 Swiss francs results. A tobacco tax for financing the antismoking campaign is proposed.

82-1461. Association for Operating Room Nurses Journal. **Cancer Society Sees Reduction in Smokers.** *Association for Operating Room Nurses Journal* 35(1): 151, January 1982.

The American Cancer Society (ACS) reports a smoking reduction of 12 percent among adults and 27 percent among teenagers as a result of a 5-year program. Begun in 1976, the society's "Target 5" program is halfway to its goal of a 25 percent reduction in adult smokers, and a 50 percent decrease among teenagers. This has meant reversing the trend of increased smoking among women. During the last 5 years, ACS volunteers have conducted approximately 18,000 free quit-smoking clinics for 20 million adults and 50 million teenagers, with a success rate of 25 to 30 percent. The society has also produced a variety of films, booklets, pamphlets, and self-help kits, and has developed cooperative antismoking programs with hospitals, industries, and schools. Each year on the day of the Great American Smokeout, millions of smokers try giving up cigarettes for 24 hours; a significant number quit for good. The program has cost the ACS 1 million dollars above its annual budget of 7 to 8 million, to counteract an industry which spends 1 billion yearly to advertise its products.

82–1462. Bates, L. H. Smoking Control From the Oncologist's Point of View. *Journal of the Indiana State Medical Association* 75(5): 322-324, May 1982.

The single most common reason given by patients who have stopped smoking is "my doctor told me to quit." When physicians advise their patients to quit smoking, initial results are not as successful as those of organized programs. However, the 6-month to 1-year rate is nearly identical. The magnitude and pathogenesis of smoking-related disease is generally unknown by the public, and many physicians are not fully aware of smoking's association with coronary artery disease, cerebrovascular disease with subarachnoid hemorrhage, chronic bronchitis, emphysema, black lung disease, peptic ulcer, reduced birth weight and infant mortality, and various cancers. Physicians are advised to record the smoking history of every patient, and advise every smoker to quit, explaining the benefits of quitting and hazards of continuing. In addition, patients should be given printed material, advice on how to quit or referral to a program, and support and encouragement if the patient is at first unable to quit completely.

82–1463. Benfari, R. C.; Ockene, J. K.; McIntyre, K. M. **Control of Cigarette Smoking From a Psychological Perspective.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 3. Palo Alto, California, Annual Reviews Inc., 1982, pp. 101-128.

In a review of the extensive number of strategies and programs developed to facilitate smoking cessation, it is concluded that little progress has been made over the last 10 years in the control of smoking behavior. One of the main reasons for this impasse has been a lack of focus on factors that contribute to maintenance of the habit, such as mass advertising. Furthermore, studies linking psychological variables to treatment variables have been limited. Instead, much of the existing research has focused on more effective intervention techniques or pursuing the relationship between antecedent variables and smoking behavior. It is suggested that a large-scale smoking program designed to fit the person with the proper treatment regimen can be accomplished by (1) targeting resistant groups for more specific intervention, (2) choosing the most cost-effective regimens for specific population groups, (3) devising preventive measures for the initiation stage, and (4) improving the maintenance of abstinence rate in smokers who attain cessation.

82–1464. Breslow, L. **Control of Cigarette Smoking From a Public Policy Perspective.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health.* Volume 3. Palo Alto, California, Annual Reviews Inc., 1982, pp. 129-151.

Among all present-day disease-causing agents, cigarette smoking does the greatest harm to the health of people in industrialized society. The conflict that public policy must resolve is between the long-term health effects and the short-term economic interests involved in cigarette smoking. In this overview of public policy on cigarette control, both the health and economic pressures are explored. A brief history of public policy on cigarette smoking control between 1950 and 1980 is given. The aims and objectives of smoking control are presented, and possible alternatives discussed, including prohibition, restrictions on smoking, reduction of the hazardous substances in cigarette smoke, restrictions on advertising, public information, education, aid to those who want to quit, taxation and other economic measures, international aspects of the problem, and research.

82–1465. Choi-Lao, A.T.H.; Keon, J.; Laurin, N.; Shand, J. **Smoking and Health: A Canadian Resource Guide of Public Education Materials.** Ottawa, University of Ottawa, School of Nursing, September 1982. 276 pp.

The many types of public education materials available on smoking and health, and the many health organizations concerned about the problem, are brought together in this Canadian resource guide. Designed for easy access to the information, the guide is divided into: (1) instructions for use; (2) listing by title, which also includes the medium (e.g., film, poster, book, pamphlet), description, audience level, publisher, distributor, and price; (3) indices by distributing agency, material medium, publisher/producer, and subject; (4) a bibliography of books on smoking cessation; and (5) directories of distributors and publishers/producers. The guide is expected to be of help to voluntary health organizations, public health units, health educators, and resource libraries.

82–1466. Cooreman, J.; Perdrizet, S.; Burghard, G. **La Lutte Anti-Tabagique a l'Ecole Primaire. [Anti-Smoking Measures at the Primary School.]** *Archives Francaises de Pediatrie* 39(2): 113-117, February 1982, French.

A retrospective study was made from a self-administered questionnaire distributed to 4,166 pupils aged 15 to 18, attending Bas-Rhine high schools, in spring 1979. The results demonstrate the relative effectiveness of educational antismoking measures when information is given to specific age groups. Information given at the primary school level resulted in a proportionately and significantly lower number of smokers; there was also a tendency both to smoke fewer cigarettes per day, and to choose brands having lower tar and nicotine levels. These pupils also left a greater portion of the cigarette unsmoked, i.e., they left longer cigarette butts. In addition, the number of pupils who had never smoked was greater in those groups who received information at the primary school level; and the number of ex-smokers (those who had given up smoking for at least 1 year) was also greater. The results emphasize the effectiveness and importance of public health measures carried out in young children. (Auth. Abs.)

82–1467. Evans, R. I. **Training Social Psychologists in Behavioral Medicine Research.** In: Eiser, J. R. (Editor). *Social*

Psychology and Behavioral Medicine. New York, John Wiley and Sons, 1982, pp. 15-35.

In a discussion on the application of social psychology to prevention and control of heart disease, a smoking prevention program for adolescents is described. A strategy of "inoculation against pressures to smoke" was used. If students can be brought through the period of greatest vulnerability to these influences, then later addiction to smoking will be less likely to occur. Thus this program focuses on training adolescents to resist these pressures. The pilot study with 750 seventh-grade students consisted of a series of short videotapes featuring adolescents presenting information and role-playing situations where social pressure to smoke is encountered. In some classrooms, feedback was given and posters dealing with peer pressure were present. All students were surveyed by questionnaire following the videotapes. Evidence suggests that peer pressure, parental modeling, and media messages may outweigh the belief that smoking is dangerous. Also, fear is not an effective deterrent to smoking, and antismoking messages with a "time trap"—overly focused on future consequences—are ineffective. Immediate hazards should be emphasized, and the skills needed to resist peer pressure taught for positive results. An example of these concepts applied to preventive dentistry is also given.

82-1468. Hay, D. R. **Drinking and Smoking in the Land of the Long White Cloud. (Letter).** *British Medical Journal* 284(6325): 1338, May 1, 1982.

Although the conclusion of a previous article (*British Medical Journal* 284(6321): 1029-1031, April 3, 1982) that New Zealand is slow in smoking control measures is agreed with, it is noted that the situation is not accurately pictured: where smoking is concerned, New Zealand is comparable to most European countries. New Zealand's smoking control actions include numerous advertising bans; smoking restrictions on public transportation; educational, economic, and legislative antismoking efforts; phasing out of tobacco agriculture and manufacturing; and questions on smoking habits in a national census. Both smoking rates and age-standardized death rates dropped between 1976 and 1981. The Cancer Society and National Heart Foundation actively promote nonsmoking, and the achievements of the newly formed pressure group ASH are anticipated. See also 82-1113.

82-1469. Homel, P. J.; Daniels, P.; Reid, T. R.; Lawson, J. S. **Results of an Experimental School-Based Health Development Programme in Australia.** *International Journal of Health Education* 24(4): 213, 263-270, 1981.

The Schools Project, an attempt to bring about positive changes in the health knowledge, attitudes, and behavior of students, was introduced to a total of 1,850 children from a working class area of Northern Sydney. A group of 1,350 children were involved as controls. A curriculum for kindergarten through grade 12 was developed and tailored to the local peculiarities and health demands of the area by a "cluster" of teachers. Comprehensive measures of the students' health knowledge, attitudes, and behaviors were taken by use of an anonymous self-administered multiple choice questionnaire, completed by all students between year 5 and year 12 before the program was introduced, and then again 2 years later. The most significant results were recorded in the secondary schools where students in the program made clear and significant gains over and above the control students. Where increases in the scores of the control secondary students occurred, these were, without exception, significantly less than in the experimental group. In terms of daily smoking rates, no significant change for girls was noted from either group. However, smoking rates of the boys in the experimental secondary school dropped by 13 percent while a 5 percent increase was noted among the boys in the control group. Highly significant drops in the rates of alcohol consumption among both the boys ($p<0.005$) and the girls ($p<0.05$) in the experimental secondary school occurred, in contrast to increases as high as 9 percent in the daily drinking rates for the controls.

82-1470. Horn, D. **National Approaches to Smoking Control.** *World Smoking and Health* 7(1): 18-20, Spring 1982.

In any given nation the following factors will either promote or retard an effective antismoking effort: (1) magnitude of the health problem (nations with high incidence of smoking-related disease are more likely to take strong measures against the habit); (2) segment of the population which smokes (cigarette use in a culture usually begins with young upper class males and spreads to lower classes, females, and older age groups; smoking cessation tends to be highest in business and professional males); (3) the place of the tobacco industry in the economy; (4) level of other risk factors for smoking-related diseases; (5) social acceptance of illness induced by personal health behavior; and (6) the relative emphasis in disease control on formal management vs. individual responsibility resulting from health education.

82-1471. Kim, S. **Feeder Area Approach: An Impact Evaluation of a Preventive Project on Student Drug Abuse.** *International Journal of the Addictions* 17(2): 305-313, 1982.

To determine the impact of a primary prevention project at the community level, a feeder area approach was implemented for a period of 2 years. One of the 10 feeder areas, a division of Mecklenburg County, North Carolina, where the elementary schools of one area "feed" into one of two predesignated junior high and then one senior high school was chosen as the target area. This concentrated the various primary prevention programs in one small area rather than scattering resources throughout the county. The feeder area therefore served as an experimental group with the rest of the county serving as a control group in a natural social setting. Through countywide drug surveys implemented before and after the project, it was determined that there is a significant reduction in the current users of various drugs in the selected feeder area when compared to the rest of the county. The largest reductions were found among the social drugs: cigarettes, marihuana, and alcohol. (Auth. Abs. Mod.)

82–1472. King, K. **Selected Behavioral Strategies for the Health Educator.** *Health Education* 13(3): 35-37, May-June 1982.

Behavioral strategies effective in changing smoking habits, poor eating habits, toothbrushing, and other health behaviors are described. The acquisition of health knowledge and traditional educational methods do not necessarily lead to changes in health behavior. Thus behavioral change techniques particularly applicable to health education are recommended. These include positive reinforcement of good health behavior, modeling, self-control and self-monitoring (found to reduce smoking rates), and stimulus control (not lighting a cigarette after a cup of coffee).

82–1473. Kuebler, T. W. **Managing the Cigarette-Smoking Patient in Your Practice.** *Journal of the Indiana State Medical Association* 75(5): 326-328, May 1982.

It is argued that physicians who have a clear-cut strategy toward smoking cessation when dealing with patients who smoke may produce a quit rate of 19 percent. It is recommended that physicians diagnose, record, and follow a patient's smoking habit in their medical record as a disease. Also, patients should be questioned about their understanding of how smoking hurts them, and how quitting will help them. Their information should be corrected if necessary, and quitting clearly recommended. Those unable to stop on their own should be referred to outside sources. The physician's office environment should be utilized as a no-smoking area, in which educational pamphlets and posters are prominently displayed.

82–1474. Miller, G. H.; Pearson, L. J.; Schumaker, J. A. **Some Fallacies of the Gori Report on Less Hazardous Cigarettes.** *Journal of the Indiana State Medical Association* 75(4): 264-267, April 1982.

A critical analysis of a report (*Journal of the American Medical Association* 240(12): 1255-1259, September 15, 1978) on low-tar, low-nicotine "less hazardous" (LT-LN) cigarettes, and of the handling of prepublication publicity which apparently misled the public, is presented. The study implied, through the use of a Critical Value, that there were levels of cigarette consumption at which the risk of disease for smokers is equivalent to that for nonsmokers. Although in a rebuttal (*Journal of the American Medical Association* 241(19): 2005, May 11, 1979) the original author stated that the intent was only to suggest a procedure for gradually reducing the hazards for smokers who are unable to quit "cold turkey," and not to specify a "safe" level of cigarette smoking, the media coverage had already stressed the "tolerable" limits shown in the study (and average smokers do not read medical journals). The media impact is shown in reports that physicians have been instructing patients to switch to LT-LN cigarettes, rather than urging them to quit smoking. Deficiencies of the original study are discussed, one being the complete disregard of the potentially harmful compounds that are formed from the combustion of the new flavoring additives and the new organic compounds used in place of tobacco. Physicians are strongly urged, in light of cancer studies which have shown no "safe" levels of cigarette consumption, to advise their patients to quit smoking, rather than switch to LT-LN cigarettes. See also 79-0369 and 79-0720.

82–1475. Parkinson, R. S.; Beck, R. N.; Collings, G. H., Jr.; Eriksen, M.; McGill, A. M.; Pearson, C. E.; Ware, B. G. **Managing Health Promotion in the Workplace. Guidelines for Implementation and Evaluation.** Palo Alto, California, Mayfield Publishing Co., 1982, 322 pp.

Comprehensive information to help businesses and other organizations set up health promotion programs is presented. The fitness programs have been designed to help employees deal with alcoholism, hypertension, smoking, weight control, and stress. Although the emphasis is on guidelines for implementation and evaluation, examples of health promotion programs in 17 corporations such as AT&T, Ford, Metropolitan Life, IBM, TWA, and Xerox are included as well as articles by experts on health at the worksite. The chapter on smoking cessation programs (first published in *Public Health Reports* 95(2): 149-157, March-April 1980) discusses behavioral trends, physician counseling, proprietary vs. nonprofit programs, examples of current programs, and includes recommendations. See also 80-1249.

82–1476. Perry, C. L.; Murray, D. M. **Enhancing the Transition Years: The Challenge of Adolescent Health Promotion.** *Journal of School Health* 52(5): 307-311, May 1982.

A conceptual model for adolescent health behavior development, the Environmental Influences System, and a correlate model, the Personal Influences System, are described. The models consist of four structures arranged on a distal—proximal continuum, which exert influence on the adolescent, and thus serve as targets for antismoking messages. The most proximal is the model structure, consisting of parents, best friends, siblings, teachers, and media figures. Children with parents and/or best friends who smoke are far more likely to smoke themselves; the reason most often cited for smoking is cigarette use by a best friend. Next on the continuum is the network structure; interacting groups such as families, neighborhoods, and peer groups. A peer group gathering after school is the situation in which smoking is most often learned and reinforced. Social systems are the next influence, and include formal groups such as school, church, and the workplace. In making the transition to adult roles, smoking attitudes at college or on the job become increasingly important. The most distal influence is the community message structure—government regulations, media messages, and private health organizations. The corresponding structures of the Personal Influences

System are: (1) behavior repertoire (smoking vs. getting a job as an adult action), (2) perceived environment (*everyone* smokes vs. *everyone* jogs), and (3) personality structure (accepting a cigarette for lack of self-esteem to refuse). In the trial programs described, behavior change techniques are developed around the considerations inherent in adolescent transitions, e.g., smoking is disassociated with adulthood, and healthful activities are substituted.

82–1477. Purcell, I.; Lloyd, D.; Hardes, G.; Alexander, H.; Leeder, S. R. **Hunter Region Childhood Smoking Prevention Study and the Evaluation of Preventive Education.** In: *Man, Drugs and Society—Current Perspectives.* Proceedings of the 1st Pan-Pacific Conference on Drugs and Alcohol, Canberra, February 26-March 5, 1980. The Australian Foundation on Alcoholism and Drug Dependence, Canberra, Australia, 1981, pp. 256-258.

Although methodological and design problems have been shown to exist in evaluation research, it is felt that they can and must be overcome to give drug education the chance to prove its credibility. The importance of evaluation in preventive health education; the problems of evaluation of drug education programs; and examples of some of the problems that occur in evaluation research, based on the Hunter (New South Wales) region Childhood Prevention Study, are discussed. Evaluation should be concerned with program efficacy, effectiveness, and efficiency. Specific problems in drug education are outlined, and the Hunter region study, which involved 6,000 primary schoolchildren, is used to illustrate how some aspects of an education program, which focused on prevention of smoking, were evaluated. Criteria for evaluation of the program included whether it increased the child's knowledge, influenced the children's attitudes, discouraged the children from smoking, or prevented the adoption and/or continuation of smoking. The importance of baseline data, study timetable, teacher effects, timing in the measurement of outcome, defining objectives, measuring the processes involved as well as the outcomes, and sampling, are all detailed. It is noted that nonparticipation and attrition are potential sources of bias in such research. Also, all of the components should be evaluated in any multicomponent program. The Hunter study was composed of nine activities, but the sample size and limited resources made it impossible to do individual evaluations. Therefore, there is no way to establish which components were the most effective.

82–1478. Syme, S. L.; Alcalay, R. **Control of Cigarette Smoking From a Social Perspective.** In: Breslow, L.; Fielding, J. E.; Lave, L. B. (Editors). *Annual Review of Public Health,* Volume 3, Palo Alto, Annual Reviews Inc., 1982, pp. 179-199.

Ways in which social and cultural forces influence the initiation and establishment of smoking behavior and suggested methods of intervention and prevention are reviewed. Several components of the social environment that influence smoking at the macrolevel include (1) the cultural component that associates smoking with relaxation, adulthood, sexual attractiveness, and emancipation; (2) the socioeconomic structure of the tobacco industry; (3) explicit advertising utilizing cultural values that favor smoking; and (4) implicit endorsements by influential persons such as film stars, TV personalities, teachers, and doctors. At the microlevel, parents, siblings, and peers are the influential components. From a public health perspective, the most sensible strategy of control is prevention. Three suggested strategies are (1) prohibition and/or regulation of smoking, (2) regulation of production and/or distribution of tobacco products, and (3) health education. It is concluded that the same social milieu that encourages smoking behavior can also be used constructively to discourage it. It is also necessary to better understand and neutralize the many forces generated by the cigarette industry that encourage consumption.

82–1479. Wagner, E. H.; Beery, W. L.; Schoenbach, V. J.; Graham, R. M. **An Assessment of Health Hazard/Health Risk Appraisal.** *American Journal of Public Health* 72(4): 347-352, April 1982.

A state-of-the-art review of the health hazard/health risk appraisal (HHA/HRA) health promotion technique was conducted. This method consists of measurement, by self-report questionnaire of clients' behaviors and personal characteristics (e.g., smoking habits, alcohol intake, family history of cancer and heart disease, exercise, frequency of checkups) viewed as predictive of an increased or decreased chance of dying from one of the leading causes of death. Risk estimates are based on data from the Framingham Study and the American Cancer Society. Interviews with developers and users of the HHA/HRA and consultation with experts in epidemiology, biostatistics, and behavioral science indicated that the scientific basis for HHA/HRA risk predictions is problematic because of arithmetical imprecision, insufficiency of the scientific evidence for certain behavioral recommendations, and inaccuracies in client-supplied data. It is concluded that the HHA/HRA's potential for motivating behavioral change cannot be substantiated from available evidence, nor can the method be assumed absent of adverse effects.

See also, **82–1342, 82–1434, 82–1452**

SMOKING CESSATION METHODS

82–1480. Agne, C. **The Effects of Follow-Up Procedures on the Maintenance of Non-Smoking Behavior of the Participants in a Smoking Cessation Program.** Doctoral Dissertation, Texas Woman's University, College of Health, Physical Education, and Recreation, Denton, University Microfilms International GAX82-08752, December 1981, 98 pp.

The effect of weekly vs. twice per month vs. monthly telephone followup in maintaining nonsmoking behavior of participants in a quit-smoking program was examined. The subjects were 22 participants in the American Cancer Society's 3-day "I Quit" program. Though the difference in effectiveness between the three followup frequency intervals was not significant, the data show that there were no successful quitters in the monthly group, two in the twice-monthly group, and three in the weekly group. An additional four in the twice-monthly group and two in the weekly group were partially successful. Telephone followup also appeared to add a humanizing element to the program: 84 percent of the subjects indicated appreciation and benefits from the contact. Success was unrelated to daily cigarette consumption, age, weight fluctuations, education, marital status, need for additional help, or reason for quitting, but was negatively associated with the presence of other smokers at home.

82–1481. Fuller, J. A. **Smoking Withdrawal and Acupuncture.** *Medical Journal of Australia* 1(1): 28-29, January 9, 1982.

Acupuncture treatment appeared to help reduce the effects of smoking withdrawal among at least some subjects in a smoking cessation study. A study examined the relapse rate of 194 people (118 men and 76 women) who were given acupuncture treatment to help them stop smoking. Ninety-five percent of patients quit smoking after three acupuncture treatments. Fifty-five (32 percent) of the 174 patients who replied to a mailed questionnaire said they had not smoked since treatment; the success rate was: 1 week, 86 percent; 6 months, 41 percent; 12 months, 34 percent; and 24 months, 30 percent. There were no further relapses among those patients who abstained for more than 24 months. Eighty-five percent of those who responded reported that acupuncture had eased the symptoms of smoking withdrawal. However, if the patient's motivation is weak, subsequent relapse will occur. (Auth. Abs. Mod.)

82–1482. Hills, A. A. **Target-Setting Self-Control for Smoking.** *Psychological Reports* 50(1): 68-70, February 1982.

In an effort to identify aspects of smoking cessation treatment programs most acceptable to subjects, 60 volunteers (all smokers) were recruited for an 11-week smoking modification program. All agreed to participate in all aspects of the program. The mean age of participants was 39.2 years, length of smoking 21.6 years, and number of cigarettes smoked/day was 25.7. All subjects recorded consumption for the initial week of the program, after which each was telephoned weekly to establish a reduction target for the following week: "self-paced" subjects set their own target; "therapist-paced" subjects had a target set for them. Guidance information was the same for both groups and included three home visits. No significant differences between the groups were found for age, duration of smoking, and baseline smoking rate, but therapist-paced subjects showed greater reductions at up to 3 months followup. A significant decrease in compliance (increase in target deviation) with time in treatment, supported the view that a "stuck" point at about 10 to 12 cigarettes/day occurs with this type of program. The locus of control data did not reveal a clear relationship with outcome, although therapist-paced subjects tended to lower consumption more consistently. Positive aspects of the program need to be investigated on a longer followup period with a larger group to establish the importance of a therapist in smoking cessation/reduction programs.

82–1483. Kozlowski, L. T.; Rickert, W. S.; Pope, M. A.; Robinson, J. C. **A Color-Matching Technique for Monitoring Tar/Nicotine Yields to Smokers.** *American Journal of Public Health* 72(6): 597-599, June 1982.

A technique that enables individuals to detect the number of puffs taken on a filter cigarette (and hence their tar yield) is described. The "color" of the spent filter is compared to a color scale that simulates the appearance of filters exposed to low-, standard-, or high-yield smoking-machine regimens. Average ratings of filters by 11 subjects correlated almost perfectly with the number of standard puffs to which the filters had been exposed. (Auth. Abs.)

82–1484. Linberg, S. E.; Horan, J. J.; Hodgson, J. E.; Buskirk, E. R. **Some Physiological Consequences of the Rapid Smoking Treatment for Cigarette Addiction.** *Archives of Environmental Health* 37(2): 88-92, March-April 1982.

Physiological changes produced by rapid smoking were not found to be due to a breathing method artifact in a group of 36 male and female volunteers in a smoking reduction program. The effects of rapid smoking, casual smoking, and rapid breathing on heart rate, blood pressure, cardiac output, and rectal-forearm-finger temperature in the younger group ($n=26$) of subjects, age 19 to 24 years, were compared with those of subjects in the older group ($n=10$), age 29 to 45 years. The older subjects smoked low-nicotine cigarettes only, while the younger subjects smoked either low- or high-nicotine cigarettes. Carboxyhemoglobin, end-tidal carbon monoxide, plasma nicotine, electrocardiogram, and subjective symptom data were also obtained. In addition to the conclusion on breathing method, results show that: (1) changes are greater than those produced by casual smoking; (2) the effect of varying the nicotine level during rapid smoking is not fully predictable; (3) firm conclusions on the hazard potential of the physiological changes produced by rapid smoking cannot be advanced; and (4) the evidence for the wisdom of excluding older age subjects from participating in the rapid smoking treatment is at best indirect (older subjects displayed no ECG abnormalities, but they were restricted to low-nicotine cigarettes).

82–1485. Schneideman, I. **Acupuncture and Smoking. (Letter).** *New Zealand Medical Journal* 94(693): 276, October 14, 1981.

A discussion of acupuncture as an aid to smoking cessation (*New Zealand Medical Journal* 93(686): 421-423, June 24, 1981) is criticized relative to (1) volunteer screening procedures, (2) acupuncture's role, (3) the usefulness of electroacupuncture, and (4) the placement points. Volunteers should be screened thoroughly, and only those anxious to quit should be selected. It is emphasized that acupuncture functions to control withdrawal symptoms after cessation, but not in preventing resumption of the habit. Further, attributing therapeutic effects to a psychological component based on the poor response to additional electroacupuncture is questioned. Finally, the omission of any mention of Dr. H. L. Wen, the first doctor to successfully use acupuncture to treat withdrawal symptoms, is noted. For many patients, only one treatment is necessary to prevent withdrawal symptoms. Subjects are screened for motivation, and a 75 percent success rate in smoking cessation is claimed. See also 82-0217.

See also, **82–1229**

TOBACCO PRODUCT ADDITIVES

82–1486. Boden, R. M. **1-N-Butoxy-1-Ethanol Acetate and Uses Thereof for Augmenting or Enhancing the Aroma or Taste of Consumable Materials.** United States Patent No. 4,333,481, June 8, 1982, 15 pp.

The organoleptic properties and uses of 1-N-butoxy-1-ethanol acetate are described. When added to foodstuffs, toothpastes, chewing gum, perfumes, tobacco products, and other consumables, this acetate displays acetaldehyde-like, acetic acid-like, and ethereal properties, but is more stable and profound than acetaldehyde. In smoking tobacco, sweet, fruity, winey, fresh, and berry characteristics are imparted in the mainstream and in the sidestream smoke. It is also an appropriate flavorant in chewing tobacco.

82–1487. Hale, R. W. **Cigarette.** United States Patent No. 4,331,166, May 25, 1982, 4 pp.

A smoking article is described which improves the sensory effect of cigarette smoke by using a disc to shape the flow of exiting smoke into a divergent pattern, increasing the perceived flavor of the cigarette. The article is most useful with cigarettes having relatively low delivery of particulate matter and a related deficiency in flavor. While the exact mechanism is not understood, it is believed the expanding cone shape of exit smoke causes particulate matter in the smoke to impact on a larger number of sensors in the smoker's mouth than the narrow column of exit smoke from a conventional cigarette. The larger number of sensory receptors contacted give increased sensory response or flavor to the smoker. In comparing low delivery cigarettes, modified according to the described invention, with unmodified cigarettes of the same brand, analytical tests showed no significant change in resistance to draw, total particulate matter, nicotine, and dilution.

82–1488. Houminer, Y.; Sanders, E. B. **Smoking Compositions.** United States Patent No. 4,259,969, April 7, 1981, 10 pp.

Tobacco and nontobacco smoking compositions which incorporate a substituted pyrazine or pyridine compound as a flavorant additive, and which are characterized by low volatility and low pack aroma, are described. Novel hydroxyethyl-substituted pyrazine and pyridine compounds are subjected to pyrolysis conditions to yield constituents that enhance the flavor and aroma of the mainstream and sidestream smoke under smoking conditions. The smoking compositions can be prepared by admixing natural tobacco and/or reconstituted tobacco and/or a nontobacco substitute with between about 0.0005 and 2 weight percent based on the weight of the smoking composition of the flavorant additive.

82–1489. Marmo, D.; Rocco, F. L. **Chewing Gum Containing Flavor Composition and Flavor Composition Therefor.** United States Patent No. 4,259,355, March 31, 1981, 31 pp.

Flavoring compositions for chewing tobacco are described. The formula comprises 85 percent Wisconsin leaf, 15 percent Pennsylvania leaf, and a casing sprayed at a rate of 30 percent. The resulting product is then redried to a moisture content of 20 percent. Ingredients of the casing include corn syrup, licorice, glycerine, fig juice, prune juice, and flavor materials described by the patent. Nuances produced by the compositions include sustained and consistently strong orange, cherry, and fruity notes.

82–1490. Mussinan, C. J.; Mookherjee, B. D.; Vock, M. H.; Vinals, J. F.; Kiwala, J.; Schmitt, F. L. **Use of Mixture Containing Caryophyllene Alcohols for Augmenting or Enhancing the Aroma or Taste of Foodstuffs.** United States Patent No. 4,250,201, February 10, 1981, 18 pp.

An organoleptically improved smoking tobacco product and additives, which impart specific desired woody, cigar-box-like, and Oriental tobacco-like flavor characteristics of natural "Turkish" tobacco (prior to smoking and, on smoking, in the mainstream and in the sidestream), are described. The characteristics can be readily controlled and maintained at the desired uniform level regardless of variations in the tobacco components of the blend. An aroma and flavor additive containing as an active ingredient one or more caryophyllene alcohols or dihydrocaryophyllene alcohol isomers is added to the smoking tobacco material, to the filter, or to the leaf or paper wrapper. The smoking tobacco material may be shredded, cured, cased, and blended tobacco material or reconstituted tobacco material substitutes. Satisfactory results are obtained if the proportion by weight of the sum total of caryophyllene alcohol or dihydrocaryophyllene alcohol isomer mixture used to flavoring material is between 1,500 and 15,000 ppm (0.15 percent-1.5 percent).

82–1491. Renold, W.; Skorianetz, W.; Schulte-Elte, K.-H.; Ohloff, G. **Spirane Derivatives.** United States Patent No. 4,174,327, November 13, 1979, 13 pp.

A method of producing spirane compounds to reproduce the full character of patchouli oil is disclosed. Owing to their particular organoleptic properties, these derivatives can be used for the preparation of artificial flavors or for the aromatization of foodstuffs, animal feeds, beverages, pharmaceutical preparations, and tobacco products. The compounds suitable for flavoring tobacco or tobacco products impart a woody, amber-like, and cedar wood-like note reminiscent of Oriental tobaccos. The various nuances are achieved by the replacement of various isomers of 2,6,10,10-tetramethyl-1-oxa-spiro[4.5]decan-6-ol.

82–1492. Ross, W. S. **What's Been Added to Your Cigarette?** *Reader's Digest* 121(723): 111-114, July 1982.

The advent of the low-yield cigarette with its altered flavor has brought about a new concern that manufacturers might be putting in more harm with additives than they are removing. Representatives of the Office of the Assistant Secretary for Health have been negotiating with the tobacco industry for the release of lists of additives used in U.S. cigarettes. Although many of these substances are derived from compounds considered safe to humans, alterations may occur upon burning. Animal skin tests have shown tumorigenic activity for the following substances: coumarin, caramel, invert sugar, eugenol, guaiacol, angelica root extract, cocoa, licorice, glycerol, glycol, dodecan-5-olide, and nonan-4-olide. Acrolein, formed by the pyrolysis of glycerol, suppresses the action of microscopic cilia that force irritants from the lungs. As a result, the risk of chronic bronchitis and emphysema is increased and the lungs are exposed to toxins and carcinogens. It is concluded that if additives pose no threat, that fact should be known; if there is a threat, the substances should not be used.

82–1493. Rowsell, D. G.; Spring, D. J.; Hems, R. **Acyclic Carboxamides Having a Physiological Cooling Effect.** United States Patent No. 4,230,688, October 28, 1980, 10 pp.

Tobacco products containing certain organic compounds, which can be readily synthesized and which have been found to have a physiological cooling effect similar to that obtained with menthol, but without the strong minty odor, are described. The compounds are amides which may readily be prepared by conventional techniques, i.e., by the reaction of an acid chloride of the formula $R_1R_2R_3$ COCl with an amide of the formula HNR'R" in the presence of a hydrogen chloride acceptor. Typical amides used are listed, with an indication of their cooling activity. The compounds can be incorporated into cigars, cigarettes, pipe and chewing tobaccos, and snuff, as well as tobacco filters. The pad of filter material, which may be of cellulose acetate, paper, cotton α-cellulose, or asbestos fiber, is simply impregnated with an alcoholic solution of the coolant. The effect is to give a pleasant, cool sensation in the mouth when the cigarette is smoked.

82–1494. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Cyclohexenemethanols and Their Lower Esters in Perfume Compositions.** United States Patent No. 4,247,423, January 27, 1981, 46 pp.

A method for manufacturing an improved tobacco product containing, as an active ingredient, one or more of the oxabicyclooctane derivatives and/or the cyclohexene alkyl and alkenyl carbinols and/or esters is described. With this method desirable hay-like notes may be imparted to smoking tobacco products and may be readily varied and controlled to produce the desired uniform flavoring characteristics. In addition to the oxabicyclooctane derivatives and the cyclohexene alkyl and alkenyl carbinols and esters, other flavoring and aroma additives may be added to the smoking tobacco material or substitute. The additives may be added to the smoking tobacco material, to the filter or to the leaf or paper wrapper, or to a filter which is part of the smoking article. The smoking tobacco material may be shredded, cured, cased, and blended tobacco material or reconstituted tobacco material or tobacco substitutes (e.g., lettuce leaves) or mixtures thereof. The proportions of flavoring additives may be varied in accordance with taste. Satisfactory results are obtained if the proportions by weight of the sum total of oxabicyclooctane derivatives and/or cyclohexene alkyl and alkenyl carbinols and/or esters used to flavoring material are between 0.05:1 and 0.50:1. This is one of a series of patented tobacco additives based on oxabicyclooctane derivatives.

82–1495. Sprecker, M. A.; Trenkle, R. W.; Mookherjee, B. D.; Vock, M. H.; Vinals, J. F.; Kiwala, J.; Schmitt, F. L. **Process for Preparing 6-Hydroxy-2,6-Dimethylheptanal and Intermediates Thereof.** United States Patent No. 4,242,281, December 30, 1980, 22 pp.

An organoleptically improved smoking tobacco product and additives which provide specific desired sweet, woody, piney, and fruity flavor characteristics of natural tobacco (prior to smoking and, on smoking, in the mainstream and in the sidestream) as well as cooling effects are described. These effects are created, enhanced, modified, or augmented and may be readily controlled and maintained at the desired uniform level regardless of variations in the tobacco components of the blend. The additive contains, as an active ingredient, 6-hydroxy-2,6-dimethylheptanal (or stereoisomer thereof). In addition, other flavoring and aroma additives which are listed may be added to the smoking tobacco material either separately or in admixture, by any convenient method. Only a portion of the tobacco needs to be treated; thus, it can be blended with other tobaccos before forming a product.

82–1496. Wilson, R. A.; Schreiber, W. L.; Mookherjee, B. D.; Kiwala, J.; Vinals, J. F.; Vock, M. H.; Stork, G.; Schmitt, F. L. **2,6,6-Trimethyl-α-(Iso)Propenyl-1-Cyclohexene-1-Methanols and -1,3-Cyclohexadiene-1-Methanols and Organoleptic Uses Thereof.** United States Patent No. 4,243,823, January 6, 1981, 38 pp.

The preparation, uses, and organoleptic properties of the cyclohexene methanols and cyclohexadiene methanols are described. In tobaccos, these compounds provide a sweet-musty, hay-tobacco-like, fruity-raspberry, ionone-like aroma prior to smoking, and a fruity, Virginia tobacco-like aroma on smoking both in the mainstream and sidestream smoke.

82–1497. Yoshida, T.; Hall, J. B. **Ketones and Organoleptic Uses Thereof.** United States Patent No. 4,234,518, November 18, 1980, 31 pp.

A process for the production of alpha, beta unsaturated ketones by reaction of an aldehyde with a ketone in the presence of a catalyst consisting essentially of either zinc acetate or zinc acetate dihydrate is described. The ketones obtained are suitable for augmenting or enhancing the organoleptic properties of perfumes, foodstuffs, and tobaccos. In the case of tobacco, the ketones derived by this process are capable of supplying and/or potentiating certain flavor and aroma notes usually lacking in many tobacco flavors. The invention also provides improved tobacco additives and methods whereby various Virginia-type tobacco notes may be imparted to smoking products by readily varying and controlling the compound for the desired uniform flavor characteristics. In addition to the ketone(s), other flavoring and aroma additives, which are listed, may be added to the tobacco material or substituted either separately or in mixture with the produced ketone(s). The described ketones can also be incorporated with materials such as filter tips, seam paste, and packaging materials which are used along with tobacco.

TOBACCO MANUFACTURING AND PROCESSING

82–1498. Baier, A. **Apparatus for Perforating Webs of Wrapping Material for Rod-Shaped Smokers Products.** United States Patent No. 4,247,754, January 27, 1981, 8 pp.

An apparatus is described which can form holes or perforations in a cigarette wrapper to admit atmospheric air into the column of tobacco smoke and thus influence the nicotine- and/or condensate-content of the smoke. The apparatus, which makes holes in a running web of wrapping material for cigarettes or in a running web of uniting band material which connects filter plugs with plain cigarettes, has one or more groups of series-connected electrode pairs whose electrodes are disposed at the opposite sides of the web. Several sources of high-voltage impulses transmit impulses seriatim to one electrode of the foremost electrode pair of each group whereby the resulting spark discharge across the gaps between the electrodes of each pair entails the formation of holes. The size and/or the number of holes can be varied to ensure that the permeability of each unit length of the web will equal or approximate a selected value, by changing the frequency of application of high-voltage impulses or by changing the number of series-connected electrode pairs. The polarity of impulses is reversed, either regularly or at random intervals, to remove scraps of paper which accumulate at the tips of the electrodes. The electrode pairs of each group are staggered, as considered transversely and/or in the longitudinal direction of the web. (Auth. Abs. Mod.)

82–1499. Bodai, C. A.; Bodai, B. I. **Selective Filtering of Tobacco Smoke by Enhanced Filtration Efficiency.** United States Patent No. 4,253,508, March 3, 1981, 17 pp.

A sonic filter providing enhanced filtration for separating and retaining particles, vapor phase constituents, and gaseous components from tobacco smoke by stimulated coagulation and adsorption is described. A sonic generating disc is disposed in the flowstream of tobacco smoke capable of generating an ultrasonic field having frequencies up to about 1 MHz. The disc includes a ring of vibrojet whistles facing into the flow, driven by the flow to produce high frequency vortices therein. Each of the vibrojet whistles includes a resonating cavity dimensioned to resonate in phase with the others to create a disturbance which constructively interferes with the disturbances produced by the other cavities to provide a high energy field in the flow. The generating disc is also contoured to provide collision surfaces whereby all particles vibrate in phase as they cross each of the anterior and posterior surfaces thereby inducing coagulation and adsorption. When coagulation of particles of different sizes is desirable, a spectrum of frequencies can be generated which contains the optimum processing frequency of the various particles in tobacco smoke. However, it can also be used at selective frequencies for filtering out some particular constituent or constituents, such as the major carcinogenic constituent of tobacco smoke, benzopyrene (BaP).

82-1500. Bryant, H. G., Jr.; Bullock, J. F.; Honeycutt, R. G. **Stability of Tobacco Casing Systems Containing Palladium.** United States Patent No. 4,235,251, November 25, 1980, 4 pp.

A method is described which uses a polysaccharide protective colloid to improve the stability of dispersions of palladiums in casing systems used to case, flavor, and deposit palladium on tobacco. The incorporation of palladium in a smoking tobacco composition reduces the proportion of polyclic aromatic hydrocarbons (PCAH) normally present in natural leaf tobacco smoke. Further, palladium combined with a nitrate salt is even more efficient in reducing PCAH; the smoke condensate collected from the smoke of cigarettes composed of tobacco treated with palladium and a nitrate salt exhibits a substantially reduced biological activity when evaluated on experimental animals. This invention provides (1) an improved method for the manufacture of smoking tobacco compositions containing palladium; (2) a more efficient method for depositing palladium on a smoking tobacco; (3) a method for improving the stability of aqueous dispersions, including but not limited to tobacco casing systems, of palladium and its various salts; and (4) a method for improving the stability of metallic palladium dispersions in conventional tobacco casing systems.

82-1501. Debrois, P. **Method for Manufacturing Filter-Tipped Cigarettes.** United States Patent No. 4,237,908, December 9, 1980, 3 pp.

A manufacturing method is described for filter-tipped cigarettes which allows the admission of outside air into the body of the filter, thereby ensuring a dilution and cooling of the primary smokestream, and improved condensation of the tars on the filtering material. A band is wound on the end of the cylinder of tobacco and on the filter tip so as to encircle and connect them. Prior to winding and connection, the paper is slit longitudinally on the path followed by the band and the two edges of the slit are spaced according to the desired dilution of the smoke on the longitudinal axis of the cigarette. (Auth. Abs. Mod.)

82-1502. de la Burde, R. Z.; Aument, P. E.; Utsch, F. V. **Tobacco Product.** United States Patent No. 4,333,483, June 8, 1982, 11 pp.

A tobacco expansion process is described which utilizes gaseous carbon dioxide in an amount of at least 1 part carbon dioxide per 100 parts of tobacco. The filling power of the leaf is increased, reducing the tobacco content of the final product. Expansion of the tobacco is accomplished in the following sequence of steps: (1) contacting tobacco with carbon dioxide gas at a pressure of at least 250 pounds per square inch gauge (psig) for a time sufficient to impregnate the tobacco with the gas, (2) releasing the pressure, and (3) subjecting the treated tobacco to rapid heating conditions to remove the carbon dioxide and thereby expand the tobacco. (Auth. Abs. Mod.)

82-1503. Gengtao, C. **China Produces a "Medicinal" Cigarette.** *Tobacco International* 184(10): 16, 18, May 14, 1982.

The Beijing Cigarette Factory of China is manufacturing a "medicinal" cigarette containing 1.0 to 1.5 percent bluish dogbane extract. Bluish dogbane is a perennial herb used in traditional Chinese medicine to treat coughing and asthma, lower blood pressure, and increase the volume of blood in the coronary arteries. When added to cigarettes, bluish dogbane relieves coughing and helps offset the negative effects of nicotine. The "medicinal" cigarette, however, is not helpful to one's health: rather, it contains less toxic substances than ordinary cigarettes and the bluish dogbane extract has a "slight curative effect on bronchitis and similar ailments." Of chronic bronchitis sufferers who smoked 10 to 20 bluish dogbane cigarettes per day, 65.9 percent experienced less coughing, less phlegm, and normal lung function compared to 23.8 percent who smoked regular cigarettes. This is due to less nicotine and 15 to 20 percent less carbon monoxide, as well as the bluish dogbane content of the cigarettes.

82-1504. Heitmann, U.; Buchegger, J. **Apparatus for Increasing the Permeability of Wrapping Material for Rod-Shaped Smokers' Products.** United States Patent No. 4,240,448, December 23, 1980, 11 pp.

A method and apparatus for the perforation of webs which are to be converted into wrappers for filter rod sections, cigars, cigarillos, or cigarettes are described. Perforation is done by means of one or more laser beams in the machine wherein the articles are produced. The permeability of wrappers can be changed by increasing or reducing the number of holes and/or by increasing or reducing the size of holes in each wrapper. The permeability of the wrappers is monitored and the beam intensity is automatically adjusted when the monitored permeability deviates from a desired value. The perforations are formed for the purpose of admitting cool atmospheric air which influences the quantity of nicotine and condensate in the column of smoke. The perforations are disposed close to or in the filter plugs of filter cigarettes, cigars, or cigarillos, or close to one or both ends of each filter rod section or plain cigarette, depending on the length of such articles. (Auth. Abs. Mod.)

82–1505. Keritsis, G. D. **Modified Cellulosic Smoking Material and Method for Its Preparation.** United States Patent No. 4,333,484, June 8, 1982, 25 pp.

An improved process is disclosed for producing a cellulosic smoking material which incorporates a metal salt from the group consisting of calcium, magnesium, iron, and aluminum salts of various organic or inorganic acids, and affords reduced particulate matter and puff count yet has the flavor and aromatic qualities of natural tobacco. An aqueous slurry of the cellulosic material is formed, preferably of loose and slightly beaten cellulosic fibers, and the metallic salt is added to the slurry which is then cast. After drying and conditioning, the resultant sheet is slit or cut to produce a low-tar filler material. A blowing or foaming agent is contacted with the slurry in the form of loose and slightly beaten fibers to insure a more uniform casting or extrusion of slurry and to prevent the premature rupture of the foam cells at the die or casting knife.

82–1506. Naylor, D. B.; Baker, R. R. **Smoking Articles.** United States Patent No. 4,328,817, May 11, 1982, 4 pp.

Smoking articles, such as cigarettes, in which the smoke composition can be controlled to meet requirements, i.e., to reduce and/or otherwise control deliveries of total particulate matter and carbon monoxide, are described. The article consists of a rod of smoking material with packing density that varies continuously along at least a major portion of the overall length but is constant across any section. Preferably, the packing density decreases in the direction away from that end of the rod intended for lighting in such a manner that there is between a 5 percent and a 40 percent weight difference between the more dense half length and the less dense half length of the rod. The density at the less dense end of the rod may be in the range of 170 to 240 mg cm^{-3} and that at the denser end in the range of 290 to 350 mg cm^{-3}.

82–1507. Newman, M. A. **Cigarette Fire Extinguisher.** United States Patent No. 4,226,249, October 7, 1980, 4 pp.

An item inside a cigarette is described, which allows the smoker to extinguish the cigarette by squeezing the band. A reservoir inside, upon being squeezed, will release a liquid through a collapsed wall and into a tube which extends through the tobacco of the cigarette. The liquid will extinguish the fire cone at the end of the tube. This mechanism will also extinguish the cigarette should it be discarded without the smoker rupturing the reservoir, thus preventing fires and eliminating sidestream smoke from smoldering cigarettes left in ashtrays. The tube will burn with the tobacco until the cigarette is discarded. The cigarette will self-extinguish within 3 seconds.

82–1508. Samfield, M. **The Solution Is Dilution.** *Tabak Journal International* 2: 116-117.

The vapor phase of cigarette smoke is important for taste, aroma, and also health effects as many of the components are pharmacologically active. Hydrogen cyanide and acrolein, for instance, inhibit epithelial ciliary motility. The particulate phase of smoke has long been reduced with fibrous filters. The gas phase, however, is not effectively absorbed by filters; and dilution and ventilation are used for reducing gas components such as carbon monoxide (CO). This is accomplished with either porous paper, or perforated filters and plug wrap. Porous paper is considered inadequate because the burn rate increases with the porosity of the paper. Also, as the cigarette burned, less paper surface was available for ventilation; thus the delivery of smoke components was continually changing. This was solved with perforated filters and perforated plug wrap. With this combination, CO reduction is greater than expected, as the perforations allow outward diffusion of the CO, as well as ventilation. Thus far, catalysts which prevent formation of CO during combustion have been commercially unsuccessful.

82–1509. Stevens, P. M. **Device for Perforating Packaged Cigarettes.** United States Patent No. 4,231,378, November 4, 1980, 13 pp.

Because perforating cigarettes reduces the toxicity of smoke inhaled and earlier devices for this process were inconvenient as the perforation was performed on single cigarettes after removal from the package, an improved method was devised. The cigarette perforating device of the present invention comprises an inner housing, and an outer housing member which is slidable relative to the inner housing. The outer housing has a generally rectangular receiving area which is dimensioned to receive a package of cigarettes of any of the presently available sizes. Two coil springs are mounted internally of both housing members so as to bias the housings apart from each other. The outer housing has a support or partition member which has elongated piercing needles fixedly mounted. As the housings are squeezed together the needles puncture all of the cigarettes in the package which is placed in the housing area. Optionally provided is a locking mechanism for preventing relative movement of the housing portions to prevent the needles from inadvertently protruding from a nested position, thereby increasing the safety of the device.

82–1510. Sykes, L. M.; Snow, R. G.; de la Burde, R. Z.; Aument, P. E. **Process for Expanding Tobacco.** United States Patent No. 4,336,814, June 29, 1982, 8 pp.

An improved process for expanding tobacco, utilizing carbon dioxide, is described. Tobacco expansion serves to reduce both cost and the tar delivery in the smoke. This process involves impregnating the tobacco with liquid carbon dioxide and draining all excess. The saturated tobacco is subjected to pressure or temperature conditions which convert the liquid carbon dioxide to solid. It is then exposed to a heat source which vaporizes the solid carbon dioxide to gas, thus expanding the tobacco and removing the carbon dioxide. Ideally, the tobacco to be processed has a moisture content between 17 and 25 percent and an output moisture content no higher than 6 percent.

82-1511. Zilkey, B. F.; Court, W. A.; Binns, M. R.; Walker, E. K.; Dirks, V. A.; Basrur, P. K. **Chemical Studies on Canadian Tobacco and Tobacco Smoke. 1. Tobacco, Tobacco Sheet, and Cigarette Smoke Chemical Analysis on Various Treatments of Bright and Burley Tobacco.** *Tobacco International* 184(10): 83-89, May 14, 1982.

Data indicate that the toxic components in cigarette smoke produced from Canadian tobacco may be reduced by the modified processed cigarette leaf (PCL) cast sheeting procedure described, by blends of chopped whole plant (CWP) tobacco sheet or a tobacco substitute, and by high efficiency cellulose acetate and charcoal filters. A set of 25 test cigarettes was manufactured from various treatments, including cast tobacco sheeting of bright and Burley tobacco grown in Ontario, and a tobacco substitute, each tipped with either a high efficiency cellulose acetate or charcoal filter. The contents of the cigarettes were analyzed for tobacco filling value, total alkaloids, reducing sugars, ash, total nitrogen, true protein nitrogen, fiber, lignin, and cellulose. Cigarette parameters measured were weight, pressure-drop, puff number, total particulate matter (TPM), nicotine, tar, phenols, cresols, neophytadiene, nitromethane extractibles, carbon monoxide (CO), carbon dioxide (CO_2), hydrogen cyanide (HCN), acetaldehyde, acrolein, total aldehydes, and pH. Tobacco sheeting tended to reduce filling value, total alkaloids, reducing sugars, total and protein nitrogen, and ash, and to increase fiber, cellulose, and lignin. In cigarette smoke, sheeting tended to decrease nicotine, nitromethane extractibles, phenols, cresols, neophytadiene, acetaldehyde, acrolein, and total aldehydes, but increased CO and total HCN deliveries. The general reduction in condensate nitromethane extractibles in modified PCL tobacco sheeting treatments suggests that this procedure potentially reduced the biologically active components of smoke condensate. (Auth. Abs. Mod.)

TOBACCO ECONOMICS

82-1512. Ahmad, M. **Spotlight on Pakistan: Diverse Deterrents Slow Progress.** *Tobacco Reporter* 109(6): 30-32, 34, 36-37, June 1982.

During 1981, Pakistan experienced a short tobacco crop and a slowed pace in the cigarette market. Among the reasons mentioned are increased costs of production and procurement of raw materials for tobacco manufacture, inflation, and antismoking campaigns. The antismoking campaign launched in Pakistan last October is considered one of the most aggressive ever conducted anywhere. The campaign is under the direction of a committee formed by the Federal Health and Social Welfare Minister. Other possible factors include import restrictions on cigarettes for Pakistanis employed in other countries, the return of the bidi cottage industry, and the smuggling in of foreign cigarettes. In 1981, Pakistan's exports of unmanufactured tobaccos continued to fall, although this decrease was partly countered by higher shipments of cigarettes. Imports of unmanufactured and manufactured tobacco continued to be low in 1981, although Pakistan's filter market share increased. For 1982, indications are that all types of tobacco except flue-cured and Burley will be grown in sufficient amounts to meet requirements.

82-1513. Barford, M. **Year of Trauma for Britain's Tobacco Industry.** *Tabak Journal Internationcl* 2: 122, 124, April 1982.

Due to huge tax increases and other price rises, cigarette sales in Britain slumped by 11 percent in 1981. Many of these increases came as the cigarette price war was ending, which meant the end of large discounts, thus magnifying the price jumps. As a result, many smokers changed to hand rolling; but most reduced their cigarette consumption, buying in packs of 10 instead of 20. The industry's new focus on profits rather than turnover has rekindled interest in exports. In addition, jobs and advertising and promotional expenditures are being cut, and the industry is tackling the long-neglected problem of low efficiency and overstaffing in manufacture.

82-1514. Harris, J. E. **Taxing Tar and Nicotine.** *American Economic Review* 70(3): 300-311, June 1980.

Mathematical solutions are presented in this cost—benefit analysis of taxing cigarettes according to their tar and nicotine (TN) content. The design of a TN-based tax hinges critically on the health tradeoffs between smoking frequency and TN delivery: (1) if smoking a few high-TN cigarettes produces more health damage than smoking more low-TN cigarettes, a prohibitively high tax on brands delivering more than 13-15 mg tar should be considered; (2) if smoking many low-TN cigarettes is more damaging, a relatively high uniform tax on all brands would be more beneficial; but (3) if health damage depends only on total TN uptake, the choice would be to tax all brands with a moderate price differential at 16-17 mg tar. Such health tradeoffs would also determine the incremental welfare gains to be achieved by additional gradations in the tax scheme; but these gains must be balanced against the cost of maintaining an array of price gradations for different TN deliveries. The annual net benefit from a simple step-function tax would have ranged from 0.1 to 2.8 billion dollars in 1975, based on a conservative interpretation of the unperceived health costs of smoking. The welfare gains could be substantially greater if other health effects, such as those from passive smoking, or the synergistic effects with certain occupational exposures, are taken into account. Other variables, such as the differences in the carbon monoxide content of the brands chosen, in addition to the effects on the supply side of the problem (the magnitude and distribution of cigarette advertising expenditures would be a critical issue) are also discussed.

82-1515. Huebner, A. **Tobacco's Lucrative Third World Invasion.** *Business and Society Review* (35): 49-53, Fall 1980.

Stagnation in the annual growth of tobacco consumption in the industrialized world, attributable to strong antismoking campaigns, has spurred the international tobacco companies to step up their sales efforts in the Third World. Because the governments of Third World nations are unlikely to spend scarce funds on antismoking campaigns and farmers are easily persuaded to switch to the economically lucrative high-quality tobacco crop, conditions are ideal for inhabitants of these nations to succumb to advertising which reinforces the image of the smoker as "sophisticated, young, attractive, healthy, and sexy." In addition to the obvious catastrophic consequences to health attributable to tobacco consumption, tobacco growing as a way to economic development is self-defeating. Conversion to a tobacco crop diminishes the land available for food requiring importation of these products. When the foreign exchange rate is unfavorable, widespread malnutrition and even starvation result. Tobacco curing requires large quantities of heat, aggravating the energy shortage in many of these countries. People of good will in the industrialized world are faced with the challenge of demanding an end to the export of smoking to the Third World.

82-1516. Rizio, D. **Cigarette Advertising Seeks Novelty.** *Tabak-Journal International* 2: 92-93, April 1982.

The U.S. cigarette industry, despite negative warnings and antismoking drives, has increased by 2.5 percent or 17.97 billion units in 1981 with R. J. Reynolds (RJR), Philip Morris (PM), and Brown and Williamson (B&W)'s shares increasing 8.87, 16.20, and 2.26 billion units, respectively. With competition so intense, especially in the low-tar 100 mm market, these three leading companies are entering the new year with extraordinary marketing strategies. Going beyond conventional, often hackneyed, advertisements proclaiming lowest tar and best taste, RJR, PM, and B&W will pour millions of promotional/advertising dollars into sponsorships of namesake sporting, artistic, and music events. Three such innovative campaigns have commenced, featuring top selling brands: MERIT REPORT—a national public opinion survey (PM); CAMEL SCOREBOARD—an advertisement that frames sports page results (RJR); and KOOL JAZZ FESTIVALS—a series of 20 concerts (B&W). These subtle advertising approaches are a means by which RJR, PM, and B&W can proliferate their brand names on radio and television, circumventing the 1971 Federal airways ban, while seeking once again to establish cigarettes in a positive manner. (Auth. Abs. Mod.)

82-1517. Ruskov, D. **Proizvodstvo, Potreblenie i Iznos na Tyutyun i Tyutyunevi Izdeliya ot Kuba. [Cuban Production and Consumption and Export From Cuba of Tobacco and Tobacco Products.]** *Bulgarski Tyutyun* 27(1): 39-45, 1982, Bulgarian.

Official Cuban statistics on tobacco grown by State and private farms show that private farms produced about 78 percent of all Cuban tobacco in the 1977-1979 period. Dark tobacco for cigarettes and cigar wrappers accounts for over 85 percent of total production. The total area under tobacco culture varied from 593,000 ha in 1971-1972 to a maximum of 721,000 ha in 1975-1976. The average tobacco yield varied from 57.6 kg/ha in 1978-1979 to 69.7 kg/ha in 1976-1977. The 1981-1985 agricultural plan projects a 671,000 ha area for tobacco culture and an 85 to 89 kg/ha yield in 1985. About 60 percent of tobacco is processed into cigarettes and cigars. Cigarette production increased by 70 percent from 1971 to 1979, or by 40 percent in relation to population (from 1,180 to 1,770 cigarettes/person). Both filter and filterless cigarettes have been produced. In contrast, the manufacture of cigars remained nearly constant through the same period. The domestic consumption, mainly of cigarettes, has tended to increase. About 60 percent of tobacco products are exported, mainly cigars to Spain (40 to 50 percent of total exports) and cigarettes to the Soviet Union (over 90 percent of total exports).

82-1518. Sumner, M. T.; Ward, R. **Tax Changes and Cigarette Prices.** *Journal of Political Economy* 89(6): 1261-1265, December 1981.

An analysis of the difference between specific and ad valorem taxes found that the excise tax induces a substitution of quality for quantity, resulting in a greater increase in the price per unit than predicted by the constant-quality model; the ad valorem tax induces a reduction in quality and lower after-tax price than predicted. Another study found that prices rise significantly less than the tax in New Hampshire where the State tax was ad valorem, and significantly more in other States, which impose specific taxes. The present study interprets the data differently. An explanation suggested is that price changes are discontinuous because of the cost of change itself, the uncertainty of competitors' reactions, and the indivisibility of the unit of account. When there is a major change in costs, the retail margins eroded by continuous changes in minor cost components will be restored, so that retail price rises by more than the initiating factor; the latter therefore appears to exert a disproportionate influence on prices, unless appropriate allowance is made for the backlog of small increases which were not worth implementing in isolation. Correcting for the influence of general inflation on price changes and allowing for the effect on wholesale price changes through interstate competition, it is shown that there is no difference between the effects of specific and ad valorem taxes on cigarette prices and that the impact of taxes per se on prices is significantly less than one for one.

82-1519. World Tobacco. **Was Canadian Cigarette Trade Buoyant in 1981?** *World Tobacco* 76: 133-134, March 1982.

The Canadian cigarette industry appeared to do well in 1981, with the sales trend still rising. It is estimated that the Canadians purchased about 67,000 million cigarettes in 1981. Indications point to a flattening of consumer demand due to many adverse factors such as unemployment, high interest rates, general national economic weakness, excessive taxes, and a strong antismoking campaign. For all age groups, discretionary spending power is shrinking and smoking is becoming increasingly expensive. The sales of regular-size (72 mm) cigarettes are increasing, while king sizes lost a bit of ground. This shift in cigarette type preferences appears to be age-related since those most accustomed to king size are 50 or more years old, which is the main age group in which smoking is declining. The market's growth area is the lighter cigarettes, especially brands with moderate- instead of low-tar levels. The economic outlook for the immediate future appears difficult.

See also, **82-1520**

LEGISLATION

82-1520. Garner, D. W. **The Cigarette Industry's Escape From Liability.** *Business and Society Review* (33): 22-25, Spring 1980.

Legislation to increase the tax on cigarettes to fund increases in social welfare costs attributable to smoking is proposed. It is argued that, according to two Surgeon Generals, over a quarter million deaths per year in the United States are caused by smoking. Nevertheless, the cigarette business remains the only industry immune against liability. Cigarette warning labels mandated by Congress have produced a paradox: while the warning may have done very little to cut down consumption, it has relieved any legal anxiety felt by the cigarette defense counsel. Though the smoker is warned on the label that cigarettes are "dangerous," he has not been warned of habituation. Dependency adds a new dimension to the situation as it increases the likelihood of long-term use, which in turn increases the risks of early death, disability, and disease. If smokers, in particular adolescents, are advised of the risk of dependency, prospective consumers may properly evaluate risks vs. benefits of smoking. The irrationality and unfairness of having the public subsidize smoking injury through social welfare and private insurance guarantees should provide a basis for legislatively compromising the liability of cigarette manufacturers. A "safety tax" structured to fall more heavily on cigarettes containing the highest levels of tar and carbon monoxide would alleviate some of the public burden.

82-1521. Hay, D. R. **Children, Smoking and the Law.** *New Zealand Medical Journal* 95(706): 348, May 26, 1982.

From February 1, 1982, a New Zealand law prohibiting the sale of cigarettes to minors ceased to exist. In defense of the change, a new Minister of Health argued that smoking is a matter of education, not legislation. He further claimed that the old law was unenforceable, and the existence of legislation restrictive to minors encourages smoking for rebellion and to achieve adult status. However, smoking is a public health problem of enormous proportions, and though smoking education in the schools can influence adolescents' behavior, these programs will be ineffective without changes in adult examples and legislative reinforcement. Also, easy availability encourages experimentation, which in turn leads to dependence. Laws are statements of social concern, and should themselves be viewed as educational and not punitive devices. The World Health Organization recommends legislative action to complement educational activities and to indicate how important the issue is to society. Lack of legislation will hinder, not help, the cause of improved health.

82-1522. Lewit, E. M.; Coate, D.; Grossman, M. **The Effects of Government Regulation on Teenage Smoking.** *Journal of Law and Economics* 24: 545-573, December 1981.

The impact of the Fairness Doctrine and advertising ban policy on the demand for cigarettes by U.S. teenagers is estimated and the extent to which an increase in the Federal excise tax on cigarettes would reduce teenage smoking is examined. The Fairness Doctrine of the Federal Communication Commission resulted in the airing of antismoking messages on radio and television from July 1, 1967 to January 1, 1971; the Public Health Cigarette Smoking Act of 1970 banned prosmoking cigarette advertising on radio and television after January 1, 1971. Regression results suggest that the Fairness Doctrine had its largest impact in the first year of its existence and was associated with a 3.4 percentage point reduction in smoking. It is further suggested that the advertising ban was not a particularly effective policy instrument to curtail teenage smoking in the post-Fairness Doctrine environment. Results indicate that if future reductions in youth smoking are desired, an increase in the Federal excise tax is a potent policy to accomplish this goal. Comments concerning the interpretation of the results and their implications are provided by L. Benham.

82-1523. Mello, N. K. (Editor). **Advances in Substance Abuse. Volume 2: Behavioral and Biological Research.** Greenwich, Connecticut, JAI Press Inc., 1981, 358 pp.

Common processes in the genesis and maintenance of substance abuse patterns are explored and the development of ideas and concepts about addictive behaviors are traced in a series of eight papers. In a discussion of legal restrictions that might be employed to discourage the use of alcohol, tobacco, and other drugs, four modes are presented: (1) establish the conditions under which a potentially harmful substance or commodity is available (prohibitory vs. regulatory schemes); (2) regulate the flow of information and messages regarding the target behavior (governmental information efforts, mandatory warnings, or proscriptions of certain types of messages); (3) influence consumer behavior through the imposition of sanctions or punishments for undesired behavior (i.e., unauthorized possession and use of marihuana) or through other devices which establish incentives for desired behavior or noncoercive disincentives for undesired behavior (i.e., lower insurance premiums for nonsmokers); and (4) symbolize and express the official governmental view of the behavior and generate derivative effects on behavioral patterns for influencing attitudes and beliefs. Other papers examine the effects of alcohol on human sexual behavior, overeating as substance abuse, conditioning factors in opiate use, the intentional inhalation of volatile substances, animal models of genetic factors in alcoholism and of drug self-administration, and statistical stability in random brain systems—possible implications for polydrug abuse in the borderline syndrome.

82-1524. Roemer, R. **Legislative Action To Combat the World Smoking Epidemic.** Geneva, World Health Organization, 1982, 131 pp.

To assist government and health officials in developing effective legislation to combat what has been described as the "smoking epidemic," a study of national smoking control measures from various countries throughout the world is presented. The subject is examined in three parts. First, the role and evolution of legislation in smoking control is reviewed. Second, the existing legislation is summarized and analyzed according to 10 objectives: advertising control, health warnings on packaging, disclosure of tar/nicotine levels, control of the toxic components in tobacco, restrictions on sales, restrictions on smoking in public places and the workplace, prevention of smoking onset in youth, mandatory health education, economic measures, and multipurpose statutes. Third, a challenge to developing nations is outlined, and final conclusions drawn for the legal potential in smoking control. Because the focus is on legislation, an analysis of the full range of smoking control programs is beyond the scope of this study. Where available, evaluations of specific legislation are included, and six appendices are provided.

GENERAL

82-1525. Blackburn, J. D. **Legal Aspects of Smoking in the Workplace.** *Labor Law Journal* 31(9): 564-569, September 1980.

The issue of whether an employee has an enforceable right under the law to a smoke-free environment is discussed in relation to four bodies of law that touch on the subject: constitutional law, Federal statutory and administrative law, State statutory and administration law, and common or case law. On the Federal level, the U.S. Supreme Court's decision in *Gasper v. Louisiana Stadium and Exposition District* has dismissed any constitutional claims on the subject. Problems of applying Federal law to the smoking in the workplace issue, particularly the Occupational Safety and Health Act of 1970, are examined. Efforts have been made by lobby groups to apply State laws prohibiting smoking in public areas and State buildings to the workplace, to classify the employee injured by cigarette smoke as a handicapped employee, and to apply workers' compensation laws to employees "injured" by sidestream smoke. These efforts have not been successful. Although the decision in *Shimp v. New Jersey Bell Telephone* , which was based on common law, resulted in an injunction banning all smoking from work areas, its application to other cases in other jurisdictions may be limited. It is concluded that it is up to legislatures to balance the interest of the nonsmoker against the smoker. To accomplish this, a Federal Indoor Clean Air Act and a Model State Smoking Pollution Prevention Act are proposed.

82-1526. Cenci, L. **Smoking and the Workplace.** *Bulletin of the New York Academy of Medicine* 58(5): 471-479, June 1982.

The cost of employee smoking to industry and company established smoking control programs are summarized in this review of related reports on the subject. The annual cost of smoking to American industry, calculated in 1980 dollars, has been estimated to be 47.5 billion dollars—11 billion dollars for excess medical care of smokers and 36.5 billion dollars for various indirect costs such as absenteeism, premature retirement, and early death. Results of a survey of 3,000 U.S. companies (30 percent responded) revealed that 15 percent of the responding companies had a smoking program for employees, and almost 50 percent restricted or prohibited smoking in the workplace. The growing awareness by nonsmokers that cigarette smoke is a hazard to their health is also examined in terms of management's and government's responses.

82–1527. Charlton, A. **Galsworthy's Images of Smoking in the Forsyte Chronicles.** *Social Science and Medicine* 15A(5): 633-638, September 1981.

The social history of smoking is discussed as it has been portrayed in novels, using as a prime example the works of John Galsworthy. Many of the novels reflect the age in which they were written and tell us much about the social development of the smoking habit—from being a leisure class indulgence (1880s) to an accepted social practice (particularly for relieving stress) by the 1920s. It is noted that smoking was mentioned as possibly related with the deaths of several characters, suggesting that some relationship with disease may have been suspected as early as 1892. Women were also smoking, and while not considered socially acceptable behavior, even Queen Victoria admitted to smoking in an 1877 journal entry. Events of the period, including two wars, the general strike, the emancipation of women, and changes in the tobacco industry, are all used in connection with the development of smoking during this era in England. The works of several contemporaries are introduced for comparison: Galsworthy was a smoker, but Thomas Hardy (who rarely mentions smoking) did not smoke. Conan Doyle, who also mentioned it often, was a smoker. Thus, in reading such novels, an image of smoking as largely associated with "good" characters, relaxation, and the release from tension is projected.

82–1528. Dale, K. C. **ACSH Survey: Which Magazines Report the Hazards of Smoking?** *ACSH News and Views* 3(3): 1, 8-10, May-June 1982.

In a survey of 18 magazines, only one-third reported the hazards of smoking both frequently and accurately. Those surveyed have a large circulation and, with the exception of women's magazines, represent a varied audience. Despite the prevalence of newsworthy information during the survey period, few magazines covered the smoking topic extensively. However, the quality of those that did was excellent. Some magazines never discussed the hazards of smoking, or else used editorial smoke screens to downplay the role of smoking in causing disease. Many editors claimed that smoking was "old news," and did not fit their audience's interest or their editorial style. However, other health issues received extensive reporting: *Cosmopolitan* published 155 articles on dieting (vs. 8 on smoking) between 1971 and 1981. Magazines which refused tobacco ads provided the best coverage of the smoking and health issue: the quality of reporting in those with more than 5 percent of their advertising revenue from tobacco was consistently weak, the exceptions being large-circulation magazines like *Time* and *Newsweek*.

82–1529. Fynn, A. E. **Cigarette Advertising and Health Education: 8 Questions.** In: Deeley, T. J. (Editor). *Communication and Cancer Education.* Cardiff, Wales, Tenovus Cancer Information Centre, 1980, pp. 21-27.

Contrary to tobacco industry claims, it is argued that cigarette advertising does encourage the onset of smoking. Further, a total ban on tobacco promotion is encouraged based on the causal relationship between smoking and serious illness. Cigarette manufacturers claim that they cannot influence the public to begin smoking, or to smoke more, and deny that advertising expenditures have any effect on consumption; their ads are meant only to influence brand loyalty. However, tobacco sponsorship of sport events links smoking and sports in impressionable fans; and media images of the successful, young, "cool" smoker are influential to the young. The companies cannot claim they aim their promotional attempts only at smokers. Cigarettes are readily available for sale to the public, and smokers should not be prevented from buying them; but nonsmokers and the young should be protected. The current British code of advertising is insufficient to control the tobacco industry; the health warnings present in ads have no effect on the consumption of 82 percent of smokers surveyed. An advertising ban would not immediately reduce the number of smokers, but would have a great effect in the years to come. Additionally, large expenditures should be made on antismoking advertising to prevent onset and encourage quitting. Over time, a complete advertising ban combined with antismoking campaigns is a cost-effective way of preventing tobacco-related disease.

82–1530. Gray, N. J.; Hill, D. J. **Smoking: The Role of Government.** *Medical Journal of Australia* 1(1): 6-7, January 9, 1982.

The Federal and State governments of Australia are criticized for apathy toward smoking as a public health threat. Although older males are smoking less, rates among women and young men are increasing, eventually resulting in avoidable deaths for the next generation. Though doctors treat smoking-related disease, the responsibility for primary prevention rests in the political arena. The tobacco industry is wealthy, carries much political influence, has a union, is free to promote its products in all media except television and radio, and exploits all major Australian sports. Though the pharmaceutical industry must carefully label its products, tobacco companies only recently were given a voluntary code for labeling tar and nicotine. A cigarette containing 14 milligrams of tar may be misleadingly called "super mild" in a market which includes brands yielding one-third that amount. Legislation should correct these discrepancies, and it is recommended that the government remove all tobacco promotions, stop subsidizing the industry, and sharply increase taxes on tobacco products.

82-1531. James, T. **Smoking, Health and Money. (Letter).** *South African Medical Journal* 61(13): 462, March 27, 1982.

Life insurance companies in the United Kingdom are offering inducements to render smokers into nonsmokers. These companies are advertising discounted premiums (about 10 percent) for nonsmokers who have abstained during the 12 months preceding the issuance of the policy, and sales have increased by as much as 25 percent. One company offers a 30 percent reduction and another is issuing "with-profits contracts" to enable holders to qualify for preferential bonus rates if they are nonsmokers, reflecting their higher life expectancy. However, adjustments have not been made reflecting the evidence that more women than men are smokers. It is proposed that South African insurance companies advertise similar adjustments.

82-1532. Secker-Walker, R. H.; Ayres, S. M.; Cohen, J. D. **Cigarette Smoking or Your Health.** *Medical Times* 110(6): 37-44, June 1982.

Since the turn of the century, chronic illnesses such as arteriosclerosis, obstructive pulmonary disease, and cancer have replaced acute illness as the major threat to health: smoking is identified as the largest single preventable cause of chronic disease. In this review of the health consequences of smoking, it is noted that smokers have a much higher mortality from cancers of the lung, prostate, esophagus, buccal cavity, bladder, and stomach than nonsmokers, as well as pneumonia, emphysema, vascular conditions, coronary disease, ulcers, and cirrhosis of the liver. The major constituents of cigarettes are nicotine, carbon monoxide, tars, various noxious gases, and particulates. Small airways dysfunction occurs even among passive smokers. Infants of smoking mothers are more likely to be underweight and suffer from pneumonia or bronchitis in the first year of life. Smokers have twice the mortality from cardiovascular disease: the habit lowers exercise duration, accelerates or aggravates coronary artery disease, increases pulse rate and blood pressure, and lowers plasma concentrations of high density lipoproteins. The best approach to smoking-related mortality is primary prevention, aimed at those in their early teens. School education programs administered by medical students taught the health effects of smoking, but focused on negating social, peer, and advertising pressures. Another strategy offered a reward (a trip to an amusement park) to the class with the lowest smoking rates: after a year of followup, program participants had a much lower smoking incidence.

82-1533. Wigle, D. T.; Johansen, H. **Parental Smoking and Infant Morbidity. (Letter).** *Canadian Medical Association Journal* 126(11): 1277, June 1, 1982.

A study which attributed the high rate of respiratory tract infection among Canadian Indian infants to infant feeding patterns and parental attitudes toward medical care is criticized for not mentioning parental smoking habits. Findings that relate infant morbidity and mortality to parental smoking are summarized. It is noted that although there are scant data on the smoking habits of specific Canadian subgroups, a survey of Canadian Inuit Indians in 1966 revealed that 86.7 percent of the women and 95.2 percent of the men smoked cigarettes compared with 34 percent and 56.7 percent of all women and men, respectively. Further, data from the Nutrition Canada Survey conducted during 1970-1972 showed a prevalence of cigarette smoking of 59.5 percent among Indian men and 56.4 percent among Indian women as opposed to 43 percent and 34 percent for non-Indian men and women, respectively. Indian households also have more members, possibly providing more exposure to children who are susceptible to air pollution owing to their greater lung ventilation rate per unit of body weight.

AUTHOR AND ORGANIZATIONAL INDEX

Aaronson, S. A., 82-1273
Abelin, Th., 82-1460
Abelmann, W. H., 82-1349
Aceto, M. D., 82-1232, 82-1245
Achord, J. L., 82-1392
Agne, C., 82-1480
Ahmad, M., 82-1512
Aitken, P. P., 82-1415, 82-1416
Akbarkhanzadeh, F., 82-1303
Alcalay, R., 82-1478
Alcoholism and Drug Addiction Research Foundation., 82-1417
Alexander, H., 82-1477
Ali, B., 82-1280
Andersen, L., 82-1408
Anderson, A. J., 82-1345
Anderson, K. E., 82-1237
Andreasen, H., 82-1330
Ang, J., 82-1444
Angelkort, B., 82-1343
Anzar, U. T., 82-1324
Arany-Fuzessery, K., 82-1213
Arias, M. E., 82-1385
Arkwright, P. D., 82-1344
Armstrong, B., 82-1251
Armstrong, B. K., 82-1344
Aromaa, A., 82-1365
Aronson, M. D., 82-1304
Arrendal, H., 82-1274
Arunagiri, R., 82-1411
Ashley, M. J., 82-1252
Association for Operating Room Nurses Journal., 82-1461
Atkins, N., 82-1312
Aument, P. E., 82-1502, 82-1510
Aurell, M., 82-1377
Austen, J., 82-1390
Ayers, M., 82-1321
Ayres, S. M., 82-1532
Ayromlooi, J., 82-1380

Bachman, J. G., 82-1435
Badawy, A.A.-B., 82-1222
Baettig, K., 82-1418
Baier, A., 82-1498
Baig, M.H.A., 82-1250
Bake, B., 82-1320
Baker, R. R., 82-1506
Ball, K. P., 82-1412
Barboriak, J. J., 82-1345
Barchas, J. D., 82-1242
Barchuk, A. S., 82-1286, 82-1287
Barford, M., 82-1513
Barnes, R., 82-1305
Barrat, J., 82-1388
Barrett-Connor, E., 82-1346
Bartley, M. J., 82-1351
Basrur, P. K., 82-1511
Bates, L. H., 82-1462
Bauer, L. A., 82-1306
Beaty, T. H., 82-1307
Beck, R., 82-1460
Beck, R. N., 82-1475
Beery, W. L., 82-1479
Beilin, L. J., 82-1344
Ben, R. L., 82-1304
Benfari, R. C., 82-1463
Bennet, K. R., 82-1391
Benowitz, N. L., 82-1347
Bensemana, D., 82-1401
Bianchi Porro, G., 82-1392
Bidet, J. M., 82-1348
Bigelow, G. E., 82-1428
Bing, O.H.L., 82-1349
Binns, M. R., 82-1511
Birch, S., 82-1312
Biryukova, N. E., 82-1215
Bjure, J., 82-1377
Blackburn, J. D., 82-1525
Blot, W. J., 82-1259
Blouin, R. A., 82-1306
Blum, J. K., 82-1214
Blumhardt, L. D., 82-1399
Bodai, B. I., 82-1499
Bodai, C. A., 82-1499
†Bodai Industries, Inc., 82-1499
Bodemar, G., 82-1410
Boden, R. M., 82-1486
Boehmer, J., 82-1419
Bone, R. C., 82-1218
Bottoms, S. F., 82-1386
Boyko, E., 82-1454
Brady, J. V., 82-1428
Breckenridge, A. M., 82-1238
Breslow, L., 82-1464
Bridbord, K., 82-1263
†British-American Tobacco Co., Ltd., 82-1506
Broekelmann, T. J., 82-1340
Brooks, W. W., 82-1349
Brophy, P., 82-1272
Broudy, M., 82-1312
Brown, C. H., 82-1437
Bryant, H. G., Jr., 82-1500
Buchegger, J., 82-1504
Bullock, J. F., 82-1500
Burghard, G., 82-1466
Burton, H. R., 82-1219
Buskirk, E. R., 82-1484
Butler, N., 82-1339
Buzzi, R., 82-1418
Bygdeman, S., 82-1318

Calverley, P.M.A., 82-1308
Cameron, P., 82-1419
Carpintero Avellaneda, J. L., 82-1329
Carroll, W. M., 82-1399
Cenci, L., 82-1398, 82-1526
Chamson, A., 82-1315
Chang, K.-J., 82-1249
Chapman, S., 82-1420
Charlton, A., 82-1527
Chassin, L., 82-1421
Chatterjee, T., 82-1226
Chen, C. J., 82-1307
Cheynel, J., 82-1348
Choi-Lao, A.T.H., 82-1465
Church, D. F., 82-1239
Clark, T.J.H., 82-1333
Cleland, J. F., 82-1449
Clement, J., 82-1309
Cleophas, T.J.M., 82-1350
Cliff, K. S., 82-1422
Climo, J. J., 82-1457
Coate, D., 82-1522
Coe, R. M., 82-1423
Coggins, C.R.E., 82-1310, 82-1311
Cohen, B. H., 82-1307
Cohen, H. I., 82-1276
Cohen, J., 82-1363
Cohen, J. D., 82-1532
Cohn, M. A., 82-1312
Colburn, N. H., 82-1260
Cole, P. V., 82-1247
Collings, G. H., Jr., 82-1475
Conney, A. H., 82-1237
Cook, D. G., 82-1351

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Cook-Mozaffari, P., 82-1261
Cooper, R., 82-1352
Cooreman, J., 82-1466
Corty, E., 82-1421
Costas, R., 82-1370
Court, W. A., 82-1511
Craighead, J. E., 82-1262
Crank, G., 82-1239
Cruz-Vidal, M., 82-1370
Cullen, K., 82-1253
Cummins, R. O., 82-1351

Dale, K. C., 82-1528
Dang, C. V., 82-1393
Daniels, P., 82-1469
Datta, B. N., 82-1279
Davidovich, I. M., 82-1369
Davies, J. K., 82-1415
Davis, B., 82-1312
Davis, D. L., 82-1263
Davison, G., 82-1264
Debrois, P., 82-1501
Decker, J., 82-1265
De Guire, L., 82-1373
Dehnen, W., 82-1223
de Klerk, N., 82-1251
de la Burde, R. Z., 82-1502, 82-1510
Denney, J., 82-1438
Dennison, D., 82-1379
Deshpande, T. V., 82-1381
Desiderio, D., 82-1380
Dhand, R., 82-1279
Diamond, E. L., 82-1307
Dimich, H., 82-1220
Dirks, V. A., 82-1511
Drabick, D., 82-1382
Drake, H., 82-1447
Duch, P., 82-1382
Duchene-Marullaz, J., 82-1348
Duffy, M., 82-1264
Dumont, E., 82-1364
Dunkley, G. C., 82-1266
Dzhioyev, F. K., 82-1286

Ehrly, A. M., 82-1358
Ekstrom-Jodal, B., 82-1377
Elwood, J. H., 82-1313
Elwood, P. C., 82-1313
Epstein, S. S., 82-1298
Erhard, P., 82-1386
Eriksen, M., 82-1475
Eschwege, E., 82-1388
Eskilsson, J., 82-1372
Evans, C. H., 82-1273
Evans, G., 82-1394
Evans, H. J., 82-1248
Evans, M., 82-1222
Evans, R. I., 82-1429, 82-1467
Evans, S. C., 82-1395

Fabiano, V. L., 82-1376
Fahning, A., 82-1236
Falkner, F., 82-1384
Farley, A., 82-1401
Fawer, R. F., 82-1314
Feinleib, M., 82-1363
Feldman, J. G., 82-1267
Fennis, J.F.M., 82-1350
Fernandez Sanchez, J., 82-1329
Filiatrault, S., 82-1327
Finison, L. J., 82-1439
Fink, M., 82-1273
†Firmenich, SA, 82-1491
Fischer, D. A., 82-1324
Fishbein, M., 82-1424
Fitzgerald, B., 82-1420
Flanders, W. D., 82-1268
Fleming, J. P., 82-1437
Flenley, D. C., 82-1308
Ford, J. M., 82-1283
Fortnagel, P., 82-1241
Fox, B. H., 82-1425
Fraumeni, J. F., Jr., 82-1259, 82-1269
Freestone, S., 82-1353
Frey, J., 82-1315
Fribourg, S., 82-1383
Fridriksson, H. V., 82-1316
Friedman, G. D., 82-1405
Friedman, L., 82-1229
Fuder, H., 82-1224
Fugedi-Nemeti, M., 82-1213
Fuller, J. A., 82-1481
Furchgott, R. F., 82-1230
Fynn, A. E., 82-1529

Gagnon, M., 82-1401
Ganguin, H.-G., 82-1254
Garcia-Palmieri, M. R., 82-1370
Gardner, A. W., 82-1314
Gardner, M. L., 82-1426
Garn, S. M., 82-1384
Garner, D. W., 82-1520
Gengtao, C., 82-1503
Geyssant, A., 82-1355
Gibel, W., 82-1270
Gilbert, M., 82-1348
Gillanders, E. M., 82-1364
Gingras, S., 82-1373
Ginter, E., 82-1396
Glover, J., 82-1430
Glover, M. U., 82-1354
Glueck, C. J., 82-1282
Godsey, F., 82-1364
Goldberg, R., 82-1375
Goldberg, S. R., 82-1426
Golding, J., 82-1339
Goldstein, J. C., 82-1265
Gorrod, J. W., 82-1227
Gortmaker, S. L., 82-1317
Gotthard, R., 82-1410
Govindan, C. K., 82-1239
Graham, R. M., 82-1479
Grant, I.W.B., 82-1271
Gray, N. J., 82-1432, 82-1530
Gredler, B., 82-1427
Green, J. P., 82-1272
Greenblatt, D. J., 82-1225
Greenspan, D., 82-1297
Greenspan, J. S., 82-1297
Greenwald, P., 82-1273
Griffiths, R. R., 82-1428, 82-1431
Grimby, G., 82-1377
Grossman, M., 82-1522
Grout, P., 82-1422
Gujral, J. S., 82-1279
Gupta, M., 82-1226
Gyuzelev, L., 82-1415

Hackney, J. D., 82-1324
Halac, E., 82-1385
Halac, J., 82-1385
Hale, R. W., 82-1487
Hall, J. B., 82-1497
Hallgren, R., 82-1274
Halliday, A. M., 82-1399
Hansen, L. S., 82-1297
Hansen, W. B., 82-1429
Harburg, E., 82-1441
Hardes, G., 82-1477
Harries, A. D., 82-1397
Harris, J. E., 82-1514
Hart, G., 82-1430
Hartley, J. W., 82-1273
Hartmann, D., 82-1396
Harwick, R. D., 82-1275
†Hauni-Werke Koerber and Co. KG., 82-1498, 82-1504
Havlik, R., 82-1370
Hay, D. R., 82-1468, 82-1521
Healy, J. C., 82-1355
Heatley, R. V., 82-1397
Hecht, S. S., 82-1217
Hedenstierna, G., 82-1318
Hedenstroem, H., 82-1316

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Heitmann, U., 82-1504
Heller, W. D., 82-1236
Hems, R., 82-1493
Henderson, B. E., 82-1291
Henius, U. M., 82-1330
Henningfield, J. E., 82-1431
Henson, D., 82-1273
Herberman, R. B., 82-1273
Hermann, T., 82-1355
Heymach, G. J., 82-1340
Hibberd, A. R., 82-1227
Hiesche, K., 82-1274
Higenbottam, T., 82-1333
Hill, D. J., 82-1432, 82-1530
Hiller, F. C., 82-1218
Hillerdal, G., 82-1316
Hills, A. A., 82-1482
Hinds, M. W., 82-1276
Hinton, G. G., 82-1381
Hirayama, T., 82-1277, 82-1278
Hjermann, I., 82-1356
Hodgson, J. E., 82-1484
Hoffmann, R. G., 82-1345
Holme, I., 82-1356
Homel, P. J., 82-1469
Homer, J., 82-1448
Honeycutt, R. G., 82-1500
Hopkin, J. M., 82-1319
Horan, J. J., 82-1484
Horn, D., 82-1470
Hornig, D., 82-1396
Houben, H., 82-1353
Houminer, Y., 82-1488
Huber, G. L., 82-1349
Huebner, A., 82-1515
Husum, B., 82-1228
Hyde, J. N., 82-1439

Illingworth, R. D., 82-1412
†International Flavors and Fragrances Inc., 82-1486, 82-1489, 82-1490, 82-1494, 82-1495, 82-1496, 82-1497
Irie, T., 82-1322
Irwin, R. P., 82-1444
Iverson, D., 82-1459

Jacob, P., III, 82-1347
Jacobs, F. H., 82-1317
Jaffe, J. H., 82-1229
Jagenburg, R., 82-1256
James, T., 82-1531
Janzon, L., 82-1433
Jarboe, C. H., 82-1233
Jarvholm, B., 82-1320
Jasinski, D. R., 82-1431
Jeffery, P. K., 82-1321
Jenkins, R. M., 82-1319
Jessor, R., 82-1434
Jindal, S. K., 82-1279
Johansen, H., 82-1533
Johanson, W. G., Jr., 82-1341
Johnson, G. S., 82-1273
Johnston, L. D., 82-1435
Jones, L., 82-1397
Jones, R. T., 82-1347
Jorfeldt, L., 82-1318

Kahn, W., 82-1405
Kallner, A., 82-1396
Kannas, L., 82-1436
Kanzler, M., 82-1229
Kaplan, T., 82-1229
Kappas, A., 82-1237
Kasabian, A., 82-1448
Katayama, S., 82-1217
Kaupass, R., 82-1214
Kaur, S., 82-1280
Kawakami, Y., 82-1322
Kellam, S. G., 82-1437
Kelley, D. G., 82-1340
Kelly, K., 82-1282
Kennedy, H., 82-1375
Kent, D. C., 82-1398
Keon, J., 82-1465
Keritsis, G. D., 82-1505
Khan, M. T., 82-1230
Khoury, P., 82-1282
Kiesewetter, H., 82-1343
Kim, S., 82-1471
King, K., 82-1472
Kipp, H., 82-1405
Kirshner, N., 82-1249
Kissin, B., 82-1267
Kiwala, J., 82-1490, 82-1494, 82-1495, 82-1496
Klein, R., 82-1405
Klima, H., 82-1323
Kobayashi, D., 82-1220
Koehler, B. A., 82-1260
Koek, W., 82-1231
Kohn, K. W., 82-1273
Kolonel, L. N., 82-1276
Komaroff, A. L., 82-1304
Kozlowski, L. T., 82-1483
Kriss, A., 82-1399
Krous, H. F., 82-1383
Krupka, L. R., 82-1457
Kuber, M. T., 82-1354
Kuebler, T. W., 82-1473
Kuhn, C., 82-1340
Kuhnert, B. R., 82-1386
Kuhnert, P. M., 82-1386
Kuller, L., 82-1255
Kunze, M., 82-1427
Kunzendorf, R. G., 82-1438

Lall, K. B., 82-1368
Lam, R., 82-1310
Lamb, D., 82-1281
Lambert, C. A., 82-1439
Lamphiear, D. E., 82-1441
Lan, S.-P., 82-1325
Lancet., 82-1357
Landgraf, H., 82-1358
Lapp, J., 82-1447
Laroche, G., 82-1373
Laskarzewski, P., 82-1282
Lassleben, M., 82-1440
Laurin, N., 82-1465
Lavenius, B., 82-1320
Lawson, J. S., 82-1469
Lazzaroni, M., 82-1392
Leathar, D. S., 82-1415, 82-1416
Lee, H. P., 82-1359
Lee, P. N., 82-1400
Leeder, S. R., 82-1477
Leggett, R. J., 82-1308
Lemarie, J. C., 82-1348
Leppanen, A., 82-1243
Leren, P., 82-1356
Levine, A. S., 82-1273
Lewis, B., 82-1360
Lewit, E. M., 82-1522
†Liggett Group Inc., 82-1500
Linberg, S. E., 82-1484
Lindell, S.-E., 82-1433
Linn, W. S., 82-1324
Linnman, L., 82-1288
Lloyd, D., 82-1477
Lorenz, R., 82-1367
Lotecka, L., 82-1440
Love, G. J., 82-1325
Love, R. G., 82-1326
Lowe, R. H., 82-1216, 82-1219
Lumsden, A., 82-1281

Machin, D., 82-1422
Madle, S., 82-1236
Maeda, H., 82-1244
Malik, A. K., 82-1279
Malik, S. K., 82-1279
Malmberg, P., 82-1316
Malo, J. L., 82-1327
Marinier, R., 82-1447
Marmo, D., 82-1489

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Martin, B. R., 82–1232, 82–1245
Martin, F., 82–1401
Martin, G. L., 82–1444
Martin, R. R., 82–1327
Martin, W., 82–1240
Mattison, D. R., 82–1402
May, D. C., 82–1233
Mayes, R. W., 82–1403
McAulay, I. R., 82–1313
McCarty, M. F., 82–1234
McCoy, G. D., 82–1217
McCredie, M., 82–1283
McCullagh, S. F., 82–1328
McCusker, K., 82–1218
McDonald, J. A., 82–1340
McElderry, L., 82–1308
McGee, D., 82–1363
McGee, R., 82–1446
McGill, A. M., 82–1475
McGregor, J. F., 82–1284
McIntire, S. L., 82–1242
McIntyre, K. M., 82–1463
McLeod, P., 82–1218
McMichael, A. J., 82–1285
McNulty, L. J., 82–1444
Medway, D. A., 82–1324
Meijler, F. L., 82–1361
Meilahn, E., 82–1255
Mellies, M., 82–1282
Mello, N. K., 82–1523
Mellstrom, D., 82–1256
Menkes, H. A., 82–1307
Merabishvili, V. M., 82–1287
Merino Berdugo, J., 82–1329
Metzner, H. L., 82–1441
Miller, B. G., 82–1326
Miller, D. K., 82–1423
Miller, G. H., 82–1474
Miralles Lozano, F., 82–1329
Mokhnachev, I. G., 82–1215
Mookherjee, B. D., 82–1490, 82–1495, 82–1496
Morazain, R., 82–1364
Morgan, K. T., 82–1310
Morrison, J. A., 82–1282
Mossman, B. T., 82–1262
Mulvey, J. J., 82–1295
Mulvihill, J. J., 82–1273
Murphy, D. C., 82–1442
Murray, D. M., 82–1476
Muscholl, E., 82–1224
Mussinan, C. J., 82–1490
Musy, C., 82–1311

Naeye, R. L., 82–1387, 82–1389
Nancy, N. R., 82–1332
Napalkov, N. P., 82–1286, 82–1287
Naylor, D. B., 82–1506
Nellis, M. J., 82–1443
Nelson, K. J., 82–1260
Netherton, D. R., 82–1439
Neverov, I. V., 82–1362
New Scientist., 82–1235
Newill, C. A., 82–1307
Newman, I. M., 82–1444
Newman, M. A., 82–1507
Niebuhr, E., 82–1228
Nil, R., 82–1418
Nilsson-Ehle, P., 82–1372
Norppa, H., 82–1243
Nou, E., 82–1274
Numa, R. Y., 82–1385

Oakes, D., 82–1314
Obe, G., 82–1236
O'Brien, J. R., 82–1364
Ockene, J. K., 82–1463
Office of Population Censuses and Surveys., 82–1445
Ohloff, G., 82–1491
Olshansky, S. J., 82–1404
Olshavsky, R. W., 82–1421
O'Malley, P. M., 82–1435
Oxhoj, H., 82–1330

Pantuck, C. B., 82–1237
Pantuck, E. J., 82–1237
Papoz, L., 82–1388
Park, B. K., 82–1238
Parker, C. M., 82–1337
Parkinson, R. S., 82–1475
Partridge, P., 82–1338
Pearson, C. E., 82–1475
Pearson, L. J., 82–1474
Peeva, S., 82–1215
Pepper, M., 82–1423
Pequignot, G., 82–1388
Perdrizet, S., 82–1466
Perry, C. L., 82–1476
Pershagen, G., 82–1288
Peters, R., 82–1446
Petitti, D. B., 82–1405
Peto, R., 82–1289, 82–1290
Petrillo, M., 82–1392
Petzold, A. S., 82–1384
†Philip Morris Inc., 82–1487, 82–1488, 82–1502, 82–1505, 82–1510
Pihl, R. O., 82–1447
Pike, M. C., 82–1291
Pope, M. A., 82–1483
Posner, B., 82–1331
Posner, V., 82–1331
Prada, A., 82–1392
Prendergast, J. M., 82–1423
Presson, C. C., 82–1421
Preston-Martin, S., 82–1291
Pryor, W. A., 82–1239
Punjani, N. F., 82–1222
Purcell, I., 82–1477

Rabkin, S. W., 82–1454
Rabson, A. S., 82–1273
Radford, E. P., 82–1292
Rai, U. C., 82–1332
Ramsay, L. E., 82–1353
Rao, A.S.V., 82–1312
Reade, P. C., 82–1391
Reader, K., 82–1394
Rebohle, E., 82–1335
Reed, D., 82–1363
Rees, P. J., 82–1333
Reid, T. R., 82–1469
Reif, A. E., 82–1293
Renaud, S., 82–1364
Renold, W., 82–1491
Reunanen, A., 82–1365
Rhodes, J., 82–1397
Richard, J. L., 82–1366
Richardson, D., 82–1240
Rickert, W. S., 82–1483
Ridella, S. A., 82–1384
Riebe, M., 82–1241
Riggan, W. B., 82–1325
Rimington, J., 82–1294
Rimpela, M., 82–1243
Rinehimer, D. A., 82–1337
Rizio, D., 82–1516
Roberts, E. B., 82–1448
Robinson, J. C., 82–1483
Rocco, F. L., 82–1489
Roed-Petersen, B., 82–1406
Roemer, R., 82–1524
Rogers, D. F., 82–1321
Rogers, W. R., 82–1341
Rolles, C. J., 82–1390
Rosenberg, J., 82–1347
Ross, S., 82–1389
Ross, W. S., 82–1492
Rosser, W. W., 82–1452
Rossiter, C. E., 82–1407
Roth, K. A., 82–1242
Roth, P., 82–1367
Rothman, K. J., 82–1268
Rothschild, H., 82–1295
Rouse, I., 82–1344
Rowsell, D. G., 82–1493

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Ruch-Ross, H., 82-1317
Rundgren, A., 82-1256
Ruskov, D., 82-1517
Russell, M.A.H., 82-1247
Ruznisky, S. A., 82-1449

Sackner, M. A., 82-1312
Saloojee, Y., 82-1247
Samet, J. M., 82-1334
Samfield, M., 82-1508
Sanders, E. B., 82-1488
Schatzkin, A., 82-1352
Schenker, M. B., 82-1334
Schiodt, M., 82-1408
Schmitt, F. L., 82-1490, 82-1494, 82-1495, 82-1496
Schneideman, I., 82-1485
Schneider, W. D., 82-1335
Schneiderman, M., 82-1263, 82-1290
Schoenbach, V. J., 82-1479
Schottenfeld, D., 82-1296
Schreiber, W. L., 82-1496
Schulte-Elte, K.-H., 82-1491
Schumaker, J. A., 82-1474
Schwartz, D., 82-1388
Searle, C. E., 82-1336
Secker-Walker, R. H., 82-1532
Sellers, E. M., 82-1225
†Service d'Exploitation Industrielle des Tabacs et des Allumettes, 82-1501
Shader, R. I., 82-1225
Shand, J., 82-1465
Shaper, A. G., 82-1351
Shear, M., 82-1408
Sheen, S. J., 82-1216, 82-1219
Sherman, S. J., 82-1421
Shevtsov, B. P., 82-1369
Shida, A., 82-1322
Shillitoe, E. J., 82-1297
Shy, C. M., 82-1325
Siebenborn, R., 82-1224
Siegelaub, A. B., 82-1405
Siess, W., 82-1367
Silverman, S., Jr., 82-1297
Simon, R. M., 82-1273
Sims, R., 82-1218
Singhi, P., 82-1368
Singhi, S., 82-1368
Sirotin, B. Z., 82-1369
Skorianetz, W., 82-1491
Slangen, J. L., 82-1231
Slepetis, R., 82-1249
Smith, C. J., 82-1408
Smith, G. G., 82-1337
Smith, N., 82-1390
Snow, R. G., 82-1510
Sorlie, P. D., 82-1370
Sorsa, M., 82-1243
Spaight, S. J., 82-1439
Speizer, F. E., 82-1334
Spencer, J., 82-1450
Spier, C. E., 82-1324
Sprecker, M. A., 82-1494, 82-1495
Sprince, H., 82-1337
Spring, D. J., 82-1493
Stachnik, T. J., 82-1451
Stamler, J., 82-1371
Steen, B., 82-1256
Stenhouse, N. S., 82-1253
Sterling, T. D., 82-1220
Stevens, P. M., 82-1509
Stewart, J. H., 82-1283
Stewart, P. J., 82-1452
Stille, W. T., 82-1257
Stock, S., 82-1409
Stoffelmayr, B. E., 82-1451
Stork, G., 82-1496
Stott, F. D., 82-1312
Stovin, P.G.I., 82-1338
Stransky, M., 82-1453
Streja, D. A., 82-1454
Strom, M., 82-1410
Stubbe, I., 82-1372
Suarez, L., 82-1346
Sulochana, G., 82-1411
Sumner, M. T., 82-1518
Sutton, S. R., 82-1455
Svanborg, A., 82-1256
Svardsudd, K., 82-1377
Swartz, J. B., 82-1298
Sykes, L. M., 82-1510
Syme, S. L., 82-1363, 82-1478
Symington, I. S., 82-1364
Szklo, M., 82-1375

Tabershaw, I. R., 82-1257
Taha, A., 82-1412
Takano, T., 82-1244
Takkunen, H., 82-1365
Taube, A., 82-1288
Taylor, B., 82-1339
Taylor, J. S., 82-1283
Thauberger, P. C., 82-1449
Theriault, G., 82-1373
Thien, Th., 82-1353
Thiringer, G., 82-1320
Thomas, E. M., 82-1456
Thompson, D., 82-1374
Tibblin, G., 82-1377
Tobias, M., 82-1380
Tomlinson, V. S., 82-1319
Tomori, Z., 82-1258
Tonascia, J., 82-1375
Townsend, M., 82-1255
Trell, E., 82-1433
Trenkle, R. W., 82-1495
Tripathi, H. L., 82-1245

U.S. Department of Health and Human Services., 82-1299
U.S. Department of Health and Human Services, National Toxicology Program., 82-1221
Utsch, F. V., 82-1502

Valencia, L. M., 82-1324
Valencia Rodriguez, A., 82-1329
VanBakel, A. B., 82-1233
Van De Woestijne, K. P., 82-1309
Vandeputte-Van Messom, G., 82-1246
Vandongen, R., 82-1344
van't Laar, A., 82-1350, 82-1353
Varrell, M., 82-1448
Vasselon, C., 82-1355
Vedin, A., 82-1378
Vena, J. E., 82-1300
Vener, A. M., 82-1457
Venet, T. G., 82-1324
Ventrone, R., 82-1311
Verbuecheln, J., 82-1223
Vesey, C. J., 82-1247
Vieweg, W.V.R., 82-1354
Vijayalaxmi, 82-1248
Villiger, B., 82-1340
Vinals, J. F., 82-1490, 82-1494, 82-1495, 82-1496
Viveros, O. H., 82-1249
Vock, M. H., 82-1490, 82-1494, 82-1495, 82-1496
Vogt, H.-J., 82-1236
Vokmann, R., 82-1320

Wadsworth, J., 82-1339
Wagner, E. H., 82-1479
Walan, A., 82-1410
Walker, D. K., 82-1317
Walker, E. K., 82-1511
Wall, S., 82-1288
Wallis, T. W., 82-1341
Wanner, H. U., 82-1413
Ward, R., 82-1518
Ware, B. G., 82-1475
Warren, S. E., 82-1354

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Watson, H., 82-1312
Wearne, K. L., 82-1253
Weber, A., 82-1414
Weber, P. C., 82-1367
Wedel, H., 82-1377
Weinberg, G., 82-1255
Weir, F. W., 82-1376
Weisburger, E. K., 82-1273
Weisburger, J. H., 82-1301
Weiss, S. T., 82-1304
Westphal, K., 82-1241
Wettengel, R., 82-1342
Wheatley, G., 82-1458
White, M., 82-1302
Wigle, D. T., 82-1533
Wilde, J., 82-1254
Wilhelmsen, L., 82-1377, 82-1378
Wilhelmsson, C., 82-1378
†Wilkinson Sword Ltd., 82-1493
Williams, G. M., 82-1301
Williams, W. M., 82-1233
Wilson, J. D., 82-1218
Wilson, R., 82-1459
Wilson, R. A., 82-1496
Wilson, S. P., 82-1249
Wolff, M., 82-1423
Wolfgang, J., 82-1379
World Tobacco., 82-1519
Wulf, H. C., 82-1228
Wynder, E. L., 82-1217

Yano, K., 82-1363
Yevtushenko, G. N., 82-1362
Yoshida, T., 82-1497
Yoshikawa, T., 82-1322
Yousef, A. A., 82-1250

Zahran, F., 82-1250
Zharsky, S. L., 82-1369
Zilkey, B. F., 82-1511

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

SUBJECT INDEX

ABSENTEEISM
- acute respiratory tract illness, smoking factor, 82–1304
- employee smoking relationship, 82–1526
- welders vs. nonwelders, smoking effect, 82–1314

ACCIDENTS
- automobile vs. smoking-related mortality in Australia, 82–1251
- factory workers, smoking relationship, 82–1442
- fire prevention, self-extinguishing cigarette, 82–1507

ACETYLCHOLINE
- nicotinic receptor mechanism in the cat, blocking with phenoxybenzamine, 82–1230
- renal water and salt excretion in goats, effect with nicotine, 82–1246

ACRIDENES
- National Toxicology Program, carcinogen study, 82–1221

ACROLEIN
- cigarette smoke ingredient, effect on lung cilia, 82–1492
- minute ventilation response in the rat, probable involvement, 82–1311

ADENOCARCINOMA
- nonsmoking Chinese women, incidence, 82–1272

ADOLESCENTS
- asthma incidence relationship to parental smoking, 82–1317
- blood pressure in males aged 8 to 16, smoking effects, 82–1368
- brand preference and advertising recall in Australia, implications for antismoking programs, 82–1420
- cardiovascular disease risk factor investigation, 82–1379
- Fishbein theory applied to smoking attitudes and behavior, 82–1444
- government regulation effect on smoking habit, 82–1522
- high school seniors, cigarette and drug use, 1975-1981, 82–1435
- initiation of smoking, possible deterrent of habituation warning on label, 82–1520
- legislation vs. education in discouraging smoking in New Zealand, 82–1521
- model for a health behavior change program, 82–1476
- perceptions related to smoking behavior, 82–1440
- prevention of smoking programs and review of health effects, 82–1532
- problem behavior, theoretical model and recommendations, 82–1434
- resisting peer pressure, smoking prevention program, 82–1467
- self-images and smoking status/intentions relationship, 82–1421
- smoking habits in Canada, 1965 to 1979, 82–1458
- substance abuse, personality factors in first grade as predictors, 82–1437
- underage drinking and smoking in Scotland, adult attitudes toward, 82–1416

ADRENAL GLANDS
- nicotinic receptors in the cat, blocking by phenoxybenzamine or acetylcholine, 82–1230

ADVERTISING
- brand preference and advertising recall among schoolchildren, implications for antismoking programs, 82–1420
- 1971 cigarette advertising broadcast ban, effect on teenage smoking, 82–1522
- cigarette tax based on tar—nicotine levels, effect on, 82–1514
- smoking habit maintenance factor, 82–1463
- social influence on smoking behavior, 82–1478
- tobacco advertising revenue vs. smoking/health articles in magazines, 82–1528
- tobacco company sponsorship of sporting, artistic, and music events, 82–1516
- tobacco consumption of Third World inhabitants, effect on 82–1515
- total ban on tobacco ads in Britain recommended, 82–1529
- worldwide legislative restrictions, analysis, 82–1524

AGE FACTORS
- alcohol use and smoking in Massachusetts adults, 82–1439
- bronchitis incidence in infants and children, maternal smoking vs. breastfeeding relationship, 82–1339
- elderly men in Sweden, smoking effects, 82–1256
- high density lipoprotein cholesterol in younger vs. older men, smoking factors, 82–1346
- lung cancer, age and early diagnosis in survival, 82–1302
- lung function, effect with smoking, 82–1318
- middle-aged men, health of unemployed vs. employed in Great Britain, 82–1351

  middle-aged men, recognition of smoking-related health hazards in Sweden, 82–1433
  myocardial infarct before age 36, smoking risk factor, 82–1354
  physiological effects of rapid smoking in younger vs. older subjects, 82–1484
  school antismoking campaigns, age-related efficacy, 82–1466
  smoking behavior and opinions of South Australians, 82–1430
  smoking patterns in Australia, 1980 survey, 82–1432
  theophylline clearance in obstructive pulmonary disease, smoking relationship, 82–1306
  therapeutic drug metabolism, aging and smoking effects, 82–1225
  ventricular fibrillation and cardiac arrest, sex, age, and smoking as predictors, 82–1375

AGING
  elderly men in Sweden, smoking effects, 82–1256

AIR POLLUTION
  ambient nitrogen dioxide and smoking relationship to respiratory illness in Chattanooga, Tennessee, 82–1325
  environmental chemicals, occupational exposure, and smoking in cancer etiology, 82–1263
  indoor levels of tobacco smoke, review of testing methods, 82–1220
  indoor smoke concentrations, review, 82–1413
  lung cancer in the Soviet Union and Europe, smoking relationship, 82–1287
  lung cancer risk, with occupational and smoking factors, 82–1300
  oil aerosol and smoking relationship to respiratory symptoms in machine shop workers, 82–1330
  ozone exposure responses, smoking effect, 82–1324
  passive smoking, review of studies of possible health hazards, 82–1400
  pulmonary asbestos and dust content in adults in East Anglia, England, 82–1338
  respiratory tract infections in Canadian Indian infants, parental smoking effect, 82–1533
  welding fumes vs. smoking in pulmonary function impairment, 82–1303
  workplace indoor pollution, health and economic consequences, 82–1398
  workplace, legal aspects of passive smoking, 82–1525

ALCOHOL CONSUMPTION
  adult attitudes toward underage drinking and smoking in Scotland, 82–1416
  amblyopia patients, smoking and drinking habits and effect of abstinence, 82–1399
  blood platelet function, smoking, calcium, diet, and alcohol effects, 82–1364
  blood pressure effect, smoking relationship, 82–1344
  chronic diseases, residential history and smoking and drinking relationship, 82–1441
  coronary artery occlusion, effects with smoking, 82–1345
  eye diseases, relationship with drug and tobacco use, 82–1395
  guilt/shame association with use, smoking habit similarities, 82–1419
  head and neck cancer risk factor, tobacco use relationship, 82–1265, 82–1267
  head and neck neoplasms in hamsters, synergistic effects with nitrosamines 82–1217
  high school seniors, 1975-1981, 82–1435
  morbidity and mortality association, tobacco and drug use interrelationship, 82–1252
  mortality from all causes in Busselton Australia, association and smoking relationship, 82–1253
  19-year-old males in Finland, smoking association, 82–1436
  oral and esophageal carcinomas, promoting action of smoking, 82–1301
  oral and pharyngeal cancer risk, smoking relationship, 82–1275
  predicting adolescent substance abuse from first grade personality factors, 82–1437

ALCOHOLS
  caryophyllene mixtures as tobacco flavorants, 82–1490
  cyclohexene and cyclohexadiene methanols as flavorants, 82–1496
  cyclohexene methanols as tobacco flavorants, 82–1494
  spirane derivatives for flavoring tobacco, 82–1491

ALICYCLIC HYDROCARBONS
  cyclohexene and cyclohexadiene methanols as flavorants, 82–1496

ALKALOIDS, TOBACCO
  mutagenic capacity, bacterial tests, 82–1241

ALLERGY
  bronchial reactivity to methacholine in smokers vs. nonsmokers, 82–1327

AMBLYOPIA, TOBACCO
  alcohol, drug, and tobacco use relationship, 82–1395
  biochemical basis for pathogenesis, proposal, 82–1393
  cyanide association, 82–1337
  patients' smoking and drinking habits and effect of abstinence, 82–1399

AMIDES
  tobacco additive, acyclic carboxamides as menthol substitutes, 82–1493

AMINES
  nicotine-enhanced 5-hydroxytryptamine synthesis, reversal by naloxone, 82–1222

SUBJECT INDEX

AMINO ACIDS
protein content of bright and Burley varieties, 82-1216, 82-1219

ANGINA PECTORIS
Paris men, incidence and relationship of leukocyte count and smoking, 82-1366
pathogenesis, smoking factor, 82-1369

ANTIBODIES
Herpes simplex virus type 1 neutralizing antibody in oral cancer patients, 82-1297

ANTISMOKING CAMPAIGNS
adolescents, health behavior change program model, 82-1476
advertising recommendations, 82-1529
age-related efficacy of school campaigns, 82-1466
American Cancer Society's "Target 5", 82-1461
Austria, effectiveness of recent campaign, 82-1427
brand preference and advertising recall among adolescents, implications, 82-1420
consequences for industrialized nations vs. Third World, 82-1515
East Germany, priorities, 82-1270
factors affecting efficacy, 82-1470
Fairness Doctrine effectiveness among teenagers, 82-1522
lung cancer risk reduction, recommendations of the Soviet Union, 82-1287
Pakistan's program, 82-1512
physician's advice to patients, recommendations, 82-1462
recommendations for cancer prevention 82-1286
Switzerland, national program, 82-1460
worldwide smoking restrictions in public places, analysis, 82-1524

ANTISMOKING MATERIALS
Canadian resource guide to materials and distributors, 82-1465

ANXIETY
adolescents, profile aspect of smokers, 82-1440

ARGENTINA
national fetal growth patterns, maternal smoking effect, 82-1385

AROMATIC HYDROCARBON CONTENT
palladium for reducing amount, 82-1500

AROMATIC HYDROCARBONS
hepatic enzyme induction relationship, 82-1238
National Toxicology Program, carcinogen study, 82-1221

ARRHYTHMIA
nicotine effect, 82-1376
pathogenesis, smoking factor, 82-1369

ARSENIC
National Toxicology Program, carcinogen study, 82-1221
occupational exposure and smoking interaction on lung cancer mortality in Sweden, 82-1288

ARTERIAL DISEASES
chronic occlusive, red cell filterability in smokers vs. nonsmokers, 82-1348
coronary artery occlusion, alcohol and smoking effects, 82-1345

ARTERIES
coronary artery occlusion, alcohol and smoking effects, 82-1345

ARTERIOSCLEROSIS
carbon monoxide in etiology, reevaluation, 82-1376
cessation of smoking, effect on risk, 82-1255
primary prevention of coronary disease via lifestyle change, 82-1371
smoking-elevated vitamin C excretion in etiology, 82-1411

ARYL HYDROCARBON HYDROXYLASE
tumor promotion, smoking relationship, 82-1284

ASBESTOS
lung cancer, synergistic effect with smoking, 82-1293
lung content of adults in East Anglia, England, 82-1338
National Toxicology Program, carcinogen study, 82-1221
occupational exposure, cancer incidence and death relationship, 82-1290
pathogenesis and related diseases, review, 82-1262
respiratory morbidity among Australian waterside workers, risks of smoking vs. asbestos exposure, 82-1328
solid-state carcinogen, tobacco smoke as a synergist in lung carcinoma development, 82-1301

ASTHMA
parental smoking relationship to incidence in children, 82-1317
salbutamol vs. ipratropium bromide in airways obstruction relief, smokers vs. nonsmokers, 82-1331

ATTITUDES
adolescents, smoking beliefs in New Zealand, Australia, and the United States, 82-1444
Australian students, effect of health education project, 82-1469
Austria, impact of antismoking campaign, 82-1427
avoidance of rejection, correlation to drug use, 82-1449

Canadian pharmacists, evaluation of smoking and health program, 82–1456
high school seniors, perceived acceptance of smoking, 82–1435
nurses' smoking behavior, 82–1450
personal space and smoking behavior relationship, 82–1438
Scotland, adult attitudes toward underage drinking and smoking, 82–1416
seat belt use in smokers vs. nonsmokers, 82–1422
smoking intent and behavior, correlation, 82–1424
South Australians, smoking behavior and opinions, 82–1430
Swiss male draftees, perceived smoking hazard, 82–1453

AUSTRALIA
adolescents, smoking attitudes, 82–1444
brand preference and advertising recall among schoolchildren, implications for antismoking programs, 82–1420
Busselton study, alcohol and smoking effect on mortality from all causes, 82–1253
critique of government antismoking measures, 82–1530
health education effectiveness on working class students in Northern Sydney, 82–1469
New South Wales, lung function in smoking vs. nonsmoking workers, 82–1305
premature mortality from cigarette smoking vs. road crashes, 82–1251
primary school smoking prevention program, evaluation, 82–1477
respiratory morbidity among Brisbane waterside workers, risks of smoking vs. asbestos exposure, 82–1328
smoking behavior and opinions, 82–1430
tobacco smoking patterns, 1980 survey, 82–1432

AUSTRIA
antismoking campaign effectiveness, 82–1427
chronic bronchitis in Vienna, smokers vs. nonsmokers, 82–1323

AUTONOMIC NERVOUS SYSTEM
bronchial reactivity to inhaled methacholine in baboons, nicotine effect, 82–1341

AVERSIVE THERAPY
rapid smoking, physiological effects, 82–1484

BACTERIA
mutagenicity tests of tobacco constituents, 82–1241

BEHAVIOR, ANIMAL
nicotine self-administration in baboons, 82–1428

BEHAVIOR, HUMAN
adolescent problem behavior, theoretical model, 82–1434
adolescents, self-images and smoking status/intentions relationship, 82–1421
attrition from health behavior modification program, smoking factor, 82–1454
Australian students, health education project effect, 82–1469
avoidance of rejection, correlation to drug use, 82–1449
drug-paired stimuli in maintenance of nicotine self-administration, 82–1426
endogenous psychosocial factors in cancer incidence, 82–1425
Fishbein theory applied to adolescent smoking, 82–1444
guilt/shame association with coffee, marihuana, tobacco, and alcohol use, 82–1419
health behavior change, effect of fear-arousing communications, 82–1455
health practices, smoking habit correlations, 82–1436
low-yield cigarette purchasing, money vs. health messages as incentives, 82–1229
lung cancer patients with long-term survival, smoking habits, 82–1264
Massachusetts adults, lifestyle, 82–1439
measurement by health hazard/health risk appraisal, smoking factor, 82–1479
medical vs. law students, health promoting and smoking habits, 82–1423
opioid and tobacco dependence, functional similarities, 82–1431
perceptions related to adolescent smoking behavior, 82–1440
personal space and smoking behavior relationship, 82–1438
physicians' advice, effect on smoking cessation, 82–1452
reduction of smoking, self- vs. therapist-paced program, 82–1482
residential history, relationship to smoking and drinking habits and chronic disease incidence, 82–1441
seat belt use, smoking correlation, 82–1422
smoking intent and behavior, correlation, 82–1424

BELGIUM
respiratory function in smokers vs. nonsmokers, obstructive airways disease, 82–1309

BENZ(*A*)ANTHRACENES
National Toxicology Program, carcinogen study, 82–1221

BENZO(*A*)PYRENE
metabolism and binding in the rat lung, 82–1223

metabolism stimulation by oral tobacco consumption in rats, 82–1280
National Toxicology Program, carcinogen study, 82–1221

BENZO(*A*)PYRENE CONTENT
measurement technique, 82–1213

BERYLLIUM
National Toxicology Program, carcinogen study, 82–1221

BIDI SMOKING
bronchogenic cancer relationship in India, 82–1279

BIOASSAY
benzo(*a*)pyrene metabolites in rat blood, lung induction study, 82–1223
tumor promoters and inhibitors, cell culture for detection, 82–1260

BIRTH WEIGHT
neurological status of newborn, relationship, 82–1381

BLADDER NEOPLASMS
aluminum vs. smoking as risk factors, 82–1266
risk in heavy smokers, 82–1293

BLOOD
arterial gas values of COPD patients vs. sons, 82–1322
benzo(*a*)pyrene metabolite concentrations, rat lung study, 82–1223
erythrocyte fluidity, smoking effect, 82–1343
viscosity, chronic smoking effect in men vs. women, 82–1358

BLOOD CHEMICAL ANALYSIS
cadmium levels in blood and placental tissues of smoking pregnant women, 82–1386

BLOOD CHOLESTEROL
acute myocardial infarction incidence in Oslo study, effect with smoking, 82–1356
high density lipoprotein cholesterol in younger vs. older men, smoking factors, 82–1346
intermittent claudication relationship with smoking and other risk factors, 82–1365
levels in progeny of parents who died of cancer or heart disease, smoking factor, 82–1282
myocardial infarction risk factor with smoking and blood pressure, 82–1377

BLOOD CIRCULATION
blood supply to muscles, nicotine and smoke effects in rats, 82–1240
chronic smoking effect, men vs. women, 82–1358
erythrocyte fluidity, smoking effect, 82–1343
finger temperature recovery, smoking and ambient temperature effects, 82–1350
microcirculation in bulbar conjunctiva vessels, smoking effect, 82–1369

BLOOD COAGULATION
platelet function, diet, alcohol, and smoking effect, 82–1364

BLOOD LIPIDS
high density lipoprotein cholesterol in younger vs. older men, smoking factors, 82–1346
levels in progeny of parents who died of cancer or heart disease, smoking factor, 82–1282
smoking cessation effect on plasma concentrations, 82–1372

BLOOD PLATELETS
aggregation and thromboxane$_2$ formation, exercise, propanolol and smoking effects, 82–1367
induced aggregation, diet, alcohol, and smoking effect, 82–1364

BLOOD PRESSURE
alcohol consumption and smoking effect, 82–1344
chlorpromazine influence, smoking effects, 82–1237
males aged 8 to 16, smoking effects, 82–1368
metoprolol influence on smoking-induced changes, 82–1353
myocardial infarction risk factor with smoking and cholesterol, 82–1377
nicotine effect, 82–1347
rapid smoking effect with high- and low-nicotine cigarettes in younger vs. older subjects, 82–1484
Singapore adults, smoking effect, 82–1359

BLOOD VESSELS
capillary measurement in rats, nicotine and smoke effects, 82–1240
subarachnoid hemorrhage from cerebral aneurysm, smoking risk, 82–1412

BODY TEMPERATURE
finger temperature recovery, smoking and ambient temperature effects, 82–1350
fingertip skin temperature, nicotine effect, 82–1347

BODY WEIGHT
attrition from weight loss program, smoking factor, 82–1454
inflammatory bowel disease patients, smoking effect, 82–1397
smoke effects in rats, 82–1349
smoking cessation effect, 82–1372
snuff users vs. nonusers, 82–1332

BRAIN
nicotine effect on catecholamine levels in rats, 82–1242
nicotine levels vs. antinociception in rats and mice, 82–1245

subarachnoid hemorrhage from cerebral aneurysm, smoking risk, 82-1412

BRONCHIAL EPITHELIUM
anti-inflammatory drugs, effect on smoke-exposed rats, 82-1321

BRONCHIAL MUCOSA
anti-inflammatory drugs, effect on smoke-exposed rats, 82-1321

BRONCHIAL NEOPLASMS
epidemiology in India, bidi, hookah, and cigarette smoking relationships, 82-1279
immunoglobulin E levels among smoking males, 82-1274

BRONCHIOLES
methacholine inhalation reactivity in smokers vs. nonsmokers, 82-1327
reactivity to inhaled methacholine in baboons, nicotine effect, 82-1341

BRONCHITIS
flax mill workers in Northern Ireland, dust and smoking relationship, 82-1313
glycerol in cigarettes as risk factor, 82-1492
infants and children, incidence and relationship to maternal smoking vs. breastfeeding, 82-1339
"medicinal" cigarette for treatment in China, 82-1503
primary prevention through antismoking education and intervention, 82-1342
salbutamol vs. ipratropium bromide in airways obstruction relief, smokers vs. nonsmokers, 82-1331
Vienna, Austria, incidence in smokers vs. nonsmokers, 82-1323

CADMIUM
accumulation in blood and placental tissues of smoking pregnant women, 82-1386
National Toxicology Program, carcinogen study, 82-1221

CAFFEINE
cimetidine and smoking effects on elimination, 82-1233
teratogenic effects with nicotine, barbiturates, and aspirin on chick embryo, 82-1382

CALIFORNIA
cancer of the upper respiratory tract, incidence in Los Angeles County, 82-1291

CANADA
alcohol, drug, and tobacco use, statistical analysis, 82-1417
infant morbidity among Indian vs. non-Indian groups, parental smoking factor, 82-1533
mortality and morbidity related to smoking and alcohol abuse, 82-1252
pharmacists' attitudes about smoking, 82-1456
Raynaud's phenomenon among forestry workers in Quebec, smoking effects, 82-1373
resource guide to public education materials on smoking and health, 82-1465
smoking survey statistics, 1965 to 1979, 82-1458
tobacco industry, economic outlook for 1981 and beyond, 82-1519

CARBAZOLES
National Toxicology Program, carcinogen study, 82-1221

CARBOHYDRATES
additives, tumorigenic effect after burning, 82-1492
polysaccharide protective colloid to improve tobacco casing systems stability, 82-1500

CARBON DIOXIDE
tobacco expansion application, 82-1502, 82-1510

CARBON MONOXIDE
atmospheric level comparisons, 82-1394
cardiovascular diseases, role in etiology and aggravation, 82-1376
cigarette tax based on tar—nicotine levels, health risk consideration, 82-1514
elevation in exhaled breath of low-yield cigarette smokers, 82-1229
expired levels in segregated nonsmokers, passive inhalation effect, 82-1404
exposure effect in firefighters, smokers vs. nonsmokers, 82-1244
ischemic heart disease, risk factor, 82-1362
measurement and correlation in an adolescent cardiovascular disease risk investigation, 82-1379
reduction in Chinese cigarettes containing bluish dogbane, 82-1503
reduction with variable-density smoking article, 82-1506
smoking prevention strategies with carbon monoxide feedback for sixth grade students, 82-1429

CARBON MONOXIDE CONTENT
indoor pollutants from cigarettes, review, 82-1413
passive smoking, recommended exposure limit, 82-1414
reduction by dilution, 82-1508

CARBOXYHEMOGLOBIN LEVELS
cigarette puffing behavior in men and women, relationship, 82-1418
firefighters, smokers vs. nonsmokers, 82-1244
index of smoke exposure, with plasma thiocyanate vs. self-report, 82-1247
ischemic heart disease, risk factor, 82-1362

- secondary polycythemia patients, smoking effect on treatment, 82–1308
- segregated nonsmokers' blood content, passive inhalation of smoke effect, 82–1404
- sheesha vs. cigarette smokers, 82–1250

CARCINOGENESIS
- enhancement by cigarette smoke condensate following irradiation in rats, 82–1284
- immunosuppressive effect of tobacco combustion process, 82–1391
- nitrosamine application and ethanol consumption in hamsters, 82–1217
- occupational vs. lifestyle factors, interactive effects, 82–1285
- smoking mechanisms, 1982 report of the Surgeon General, 82–1299

CARCINOGENS
- cell culture assay for detection of tumor promoters and inhibitors, 82–1260
- genotoxic vs. epigenic, mechanisms of action, 82–1301
- identification in cigarette smoke, 82–1293
- National Toxicology Program, Second Annual Report, 82–1221

CARCINOMA, BRONCHOGENIC
- asbestos exposure and smoking effect, 82–1262
- epidemiology in India, bidi, hookah, and cigarette smoking relationships, 82–1279

CARDIOVASCULAR DISEASES
- adolescent risk factor investigation, smoking, dietary fat, and dietary cholesterol relationships, 82–1379
- carbon monoxide in etiology, reevaluation, 82–1376
- indoor air pollutants, aggravating effect, 82–1413
- prevention, tobacco elimination as a practical approach, 82–1254
- smoking relationship, 82–1258, 82–1532

CARDIOVASCULAR SYSTEM
- carboxyhemoglobin level in firefighters, smokers vs. nonsmokers, 82–1244
- nicotine effects on heart rate, blood pressure, 82–1347

CATECHOLAMINES
- acetylcholine-induced release of antidiuretic hormone, effect in goats, 82–1246
- human pheochromocytoma tumor, nicotine effect of excretion of opioid peptides and catecholamines in vitro, 82–1249
- nicotine effect on levels in rat brain, 82–1242
- tyrosine treatment for prevention of withdrawal symptoms, 82–1234

CELLS
- human fibroblast culture, smoke extracts effects, 82–1315
- human pheochromocytoma tumor, nicotine effect on dissociated cells, 82–1249
- polymorphs, cytotoxicity of cigarette smoke condensate, 82–1319

CELLS, EPITHELIAL
- mast cell differences in respiratory epithelium of smoking vs. nonsmoking lung cancer patients, 82–1281

CENTRAL NERVOUS SYSTEM
- nicotine binding sites, localization by radiolabeling technique, 82–1232

CEREBROVASCULAR CIRCULATION
- erythrocyte deformability in smokers, effect, 82–1355

CESSATION OF SMOKING
- American Cancer Society's "Target 5" program, success rates, 82–1461
- antismoking campaign in Austria, effectiveness, 82–1427
- benefits and strategies, 1982 report of the Surgeon General, 82–1299
- critique of programs and recommended changes, 82–1451
- fear-arousing communications, 82–1455
- individualized multidimensional behavioral approach, 82–1443
- intervals of telephone followup, effect on success, 82–1480
- ischemic heart disease mortality relationship, 82–1374
- leukoplakia, effect on, 82–1406
- lung cancer, arteriosclerosis, and pulmonary disease risks, effect on, 82–1255
- lung cancer patients with long-term survival, quitting patterns, 82–1264
- perception of benefits by Swedish middle-aged men, 82–1433
- personal beliefs vs. social norms, efficacy in women, grade school girls, 82–1424
- physicians' advice, effect, 82–1452, 82–1462, 82–1473
- plasma lipoprotein concentrations, effects on, 82–1372
- post-myocardial infarction mortality, effect on, 82–1378
- psychological considerations for proper regimens, 82–1463
- reversible effect on carcinogenesis, evidence of a promotion effect, 82–1301
- tyrosine in treatment of withdrawal symptoms, 82–1234
- workplace programs, guidelines for implementation and evaluation, 82–1475

CESSATION OF SMOKING, METHODS
- acupuncture treatment for withdrawal, 82–1481, 82–1485

individualized behavioral intervention, based on self-reports of smoking variables, 82–1443
weekly target-setting, self- vs. therapist-paced, 82–1482

CHEWING TOBACCO
flavoring compositions, 82–1489

CHILDREN
asthma incidence relationship to parental smoking, 82–1317
Australia, brand preference and advertising recall, implications for antismoking programs, 82–1420
Australia, primary school smoking prevention program, evaluation, 82–1477
blood pressure in males aged 8 to 16, smoking effects, 82–1368
bronchitis and pneumonia, parental smoking effect, 82–1409
gastroenteritis in children, maternal smoking vs. breastfeeding relationship, 82–1339
indoor air pollutants, health effect, 82–1413
New Zealand, legislation vs. education in discouraging smoking, 82–1521
passive smoking risks, review, 82–1414
predicting adolescent substance abuse from first grade personality factors, 82–1437
smoking prevention strategies with carbon monoxide feedback, 82–1429

CHINA
"medicinal" cigarette for bronchitis treatment, 82–1503

CHROMIUM
National Toxicology Program, carcinogen study, 82–1221

CHROMOSOMES
lymphocytes, exchange-type aberrations in smokers vs. nonsmokers, 82–1236, 82–1248
sister-chromatid exchanges in lymphocytes of smokers vs. nonsmokers, 82–1228

CHRONIC BRONCHOPULMONARY DISEASE
hyperplasia, effect of anti-inflammatory drugs on smoke-exposed rat bronchus, 82–1321
ozone exposure effect on patients, smoking relationship, 82–1324
secondary polycythemia patients, smoking effect on treatment, 82–1308

CIGAR SMOKING
Canada, incidence in 1965 to 1979, 82–1458
myocardial infarct incidence in Puerto Rico, relationship, 82–1370

CIGAR WRAPPERS
perforation method for smoke dilution, 82–1504

CIGARETTE ASH
manganese content, source, 82–1214

CIGARETTE PAPER
manganese content, source, 82–1214
perforated, carbon monoxide reduction, 82–1508
perforating apparatus, 82–1498, 82–1504

CIGARETTE SMOKE
benzo(*a*)pyrene measurement technique, 82–1213
carcinogens, National Toxicology Program study, 82–1221
contribution to indoor air pollution, review of testing methods, 82–1220
dilution and analysis with a single particle aerodynamic relaxation time analyzer, 82–1218
manganese content, source, 82–1214
nitrogen oxides, effect in lungs, 82–1239
perforation of packaged cigarettes for toxicity reduction, 82–1509
toxic components reduction with various treatments and filters, 82–1511
tumor promotion of condensate, cell culture assay, 82–1260

CIGARETTES
changing consumption patterns, Canada 1965 to 1979, 82–1458
filtered vs. nonfiltered, lung cancer risk, 82–1294
self-extinguishing, 82–1507
tax changes, effect on prices, 82–1518
tax proposal based on tar and carbon monoxide yields, 82–1520

CIGARETTES, LOW-TAR
marketing strategies, 82–1516

CIGARETTES, LOW-YIELD
carbon monoxide levels in breath of consumers, 82–1229
China, bluish dogbane herb to reduce carbon monoxide, nicotine, 82–1503
disc for flavor improvement via smoke expansion, 82–1487
disease risk report, critical analysis, 82–1474
expansion process to reduce tobacco content, 82–1502
flavor additives as health risk factors, 82–1492
smoke components reduction with various treatments and filters, 82–1511
smoke toxicity reduction by dilution and filtration, 82–1508

COCARCINOGENS
epigenic chemical carcinogens, 82–1301
lung cancer, asbestos, uranium, air pollution, and smoking factors, 82–1293

SUBJECT INDEX

COFFEE CONSUMPTION
  guilt/shame association with use, smoking habit similarities, 82–1419

COGNITION
  adolescent smoking behavior relationship, 82–1440
  state-dependent learning, smoking effects, 82–1446

CORONARY DISEASE
  causative relationship of smoking questioned, 82–1374
  coronary artery disease before age 36, smoking risk factor, 82–1354
  coronary artery occlusion, alcohol and smoking effects, 82–1345
  Hawaiian men of Japanese ancestry, cultural and psychosocial risk factors, 82–1363
  intermittent claudication relationship with smoking and other risk factors, 82–1365
  ischemic heart disease, microcirculatory disorders as pathogenesis factor, smoking effect, 82–1369
  ischemic heart disease, smoking risk factors, 82–1362
  lipid vs. high density lipoprotein cholesterol vs. smoking in intervention trials, 82–1357
  major risk factors, and basis of prevention, 82–1360
  mortality in parents vs. serum cholesterol and serum lipid levels in progeny, smoking factor, 82–1282
  Paris men, incidence and relationship of leukocyte count and smoking, 82–1366
  primary prevention via lifestyle change, 82–1371
  problems in prevention, review, 82–1361
  Puerto Rican men, smoking relationship, 82–1370
  residential history and smoking and alcohol consumption relationship, 82–1441
  Soviet Union, trends in risk factors, 82–1352

COST BENEFITS ANALYSIS
  cigarette tax based on tar—nicotine levels, health benefit tradeoffs, 82–1514
  lung cancer screening, 82–1271
  post-myocardial infarction mortality reduction, smoking factor, 82–1378

COTININE
  amniotic fluid concentration, effect on fetus, 82–1390
  nicotine iminium ion intermediate, reactions with glutathione and cyanide, 82–1227

COUGH
  duration in patients with acute respiratory illness, smokers vs. nonsmokers, 82–1304
  oil aerosol and smoking relationship in machine shop workers, 82–1330
  tar content and degree of smoke inhalation relationship, 82–1334

COUNSELING
  workplace health promotion including smoking cessation, 82–1475

CUBA
  tobacco production and consumption, 82–1517

CYANIDES
  intoxication prevention with L-ascorbic acid and dehydroascorbic acid in rats, 82–1337
  possible association with amblyopia, 82–1393

DEMOGRAPHIC FACTORS
  asthma in urban vs. rural children, parental smoking factor, 82–1317
  computer simulation model of smoking for policy development, 82–1448
  endogenous psychosocial influences on cancer incidence, 82–1425
  lung cancer occurrence patterns, smoking relationship, 82–1259
  oral cancer incidence relationship, 82–1265
  residential history, smoking and alcohol consumption and chronic disease incidence relationship, 82–1441
  urban vs. rural Puerto Rican men, smoking and heart disease incidence, 82–1370

DENTAL DISEASES
  leukoplakia-like lesions in discoid lupus erythematosis patients, tobacco factor, 82–1408

DIABETES
  heart disease risk factor, with smoking, hypertension and lipid abnormalities, 82–1360
  intermittent claudication relationship, with cholesterol and smoking, 82–1365

DIBENZANTHRACENES
  National Toxicology Program, carcinogen study, 82–1221

DIET
  acute myocardial infarction incidence in Oslo study, effect with smoking, 82–1356
  blood platelet function, calcium, fat, alcohol, and smoking effects, 82–1364
  esophageal cancer risk factor with drinking and smoking, 82–1261
  maternal smoking effect on birth weight, comparison, 82–1388
  saturated fat and cholesterol intakes in progeny, relationship to mortality in parents, smoking factor, 82–1282
  smoking cessation effect on energy intake, body weight, and plasma lipoprotein concentrations, 82–1372
  sugar and fat consumption, smoking association in Finnish 19-year-old males, 82–1436
  vitamin A effect on gastric cancer incidence in Japan, smokers and nonsmokers, 82–1278
  vitamin C turnover in smokers vs. nonsmokers, 82–1396

DNA
binding with benzo(*a*)pyrene in the rat lung, 82–1223

DRUG ABUSE
adolescent problem behavior, theoretical model, 82–1434
alcohol, drug, and tobacco use in Canada, statistical analysis, 82–1417
analgesics and tobacco risk factors in renal pelvic cancer, 82–1283
avoidance of rejection scales, correlation, 82–1449
college student survey, smoking comparison, 82–1457
drug-paired stimuli in maintenance of drug self-administration, nicotine response in monkeys, 82–1426
first grade personality factors as predictors in adolescents, 82–1437
genesis and maintenance of abuse patterns, commonality, 82–1523
high school seniors, 1975-1981, 82–1435
morbidity and mortality association, tobacco and alcohol use interrelationship, 82–1252
opioid and tobacco dependence, functional similarities, 82–1431
schoolchildren, primary prevention program, 82–1471
teratogenic effects of nicotine, barbiturates, caffeine, and aspirin on chick embryo, 82–1382
women, psychotropic drug use and smoking relationship, 82–1447

DRUG METABOLISM
bronchial reactivity to inhaled methacholine in baboons, nicotine effect, 82–1341
chlorpromazine, smoking effects, 82–1237
cimetidine and smoking effects on caffeine elimination, 82–1233
citrinin antagonism of nicotine in mice, 82–1226
H_2-blocker treatment of peptic ulcer, healing rate in smokers vs. nonsmokers, 82–1392
metoprolol influence on smoking-induced blood pressure changes, 82–1353
nicotine-enhanced 5-hydroxytryptamine synthesis, reversal by naloxone, 82–1222
nicotine vs. narcotic vs. amphetamine in drug generalization in rats, 82–1231
propanolol, effect on platelet aggregation, smoking and exercise relationship, 82–1367
salbutamol vs. ipratropium bromide in airways obstruction relief, smokers vs. nonsmokers, 82–1331
sensitivity in the elderly, smoking effects, 82–1225
smoking effect, 82–1238
sucralfate vs. cimetidine for duodenal ulcer treatment, smoking effect, 82–1401
theophylline clearance in smokers with chronic obstructive pulmonary disease, age factors, 82–1306

DUST
coal dust and smoking effects on lung function, 82–1326
flax industry in Northern Ireland, byssinosis, chronic bronchitis, and smoking interrelationships, 82–1313
pulmonary function effect, smoking effect comparison, 82–1305

DYSPNEA
tar content and degree of smoke inhalation relationship in women, 82–1334

EARS
acupuncture site for withdrawal symptom control, 82–1485

ECONOMICS
Canadian tobacco industry, outlook for 1981 and beyond, 82–1519
cigarette tar—nicotine content tax, supply and demand effects, 82–1514
Cuba, tobacco production, 82–1517
Great Britain, cigarette price increase effect on consumption, 82–1513
health care costs related to alcohol, tobacco, and drug use, 82–1252
health vs. money, public policy issues in cigarette control, 82–1464
Pakistan, tobacco industry in 1981, prospects for 1982, 82–1512
smoking habit cost to business, 82–1398
specific vs. ad valorem taxes, effect on cigarette prices, 82–1518
Switzerland, cost of smoking habit, 82–1460
Third World countries, export of smoking, economic and health consequences, 82–1515
tobacco advertising revenue vs. smoking/health articles, magazine survey, 82–1528
United States, cigarette sales increases in 1981, 82–1516
United States, employee smoking cost to industry, 82–1526
worldwide smoking control legislation, analysis, 82–1524

EMBRYO
chick, teratogenic effects of nicotine, barbiturates, caffeine, and aspirin, 82–1382

EMPHYSEMA
glycerol in cigarettes as risk factor, 82–1492

ENZYME ACTIVITY
benzo(*a*)pyrene hydroxylase, lung induction in the rat, 82–1223
drug metabolism by enzyme induction, smoking effect, 82–1238

fibronectin role in phagocytosis by pulmonary alveolar macrophages in the lungs, smokers vs. nonsmokers, 82–1340
hepatic microsomal mixed function oxidases, tobacco and nicotine effects in rats, 82–1280
inhibition by nitric oxide and nitrogen dioxide, 82–1239

ENZYMES
benzo(*a*)pyrene hydroxylase, 82–1223
hepatic microsomal mixed-function oxidases, 82–1238
hepatic microsomal mixed function oxidases, aniline hydroxylase, phenacetin *O*-dealkylase, 82–1280

ERYTHROCYTES
deformability in smokers, effect on filtration rate, 82–1355
filterability in nondiabetic patients with chronic occlusive arterial disease, smokers vs. nonsmokers, 82–1348
filtration and aggregation, chronic smoking effect in men vs. women, 82–1358
fluidity, smoking effect, 82–1343
oxygen therapy for secondary polycythemia, smoking effect, 82–1308

ESOPHAGEAL NEOPLASMS
alcohol consumption as a promoter in smokers, 82–1301
epidemiology and worldwide incidence patterns, 82–1261

ESTERS
cyclohexene methanols and their esters as tobacco flavorants, 82–1494

ETHIOPIA
fetal lung maturation rate, comparisons with South Africa and the United States, 82–1389

ETHNIC GROUPS
black males, lung cancer incidence and smoking relationship, 82–1259
blacks, occupational factor in lung cancer incidence, 82–1298
blacks vs. whites, fetal lung maturation rate, 82–1389
blacks vs. whites, maternal hematologic levels and smoking relationship to pregnancy outcome, 82–1384
Chinese women, lung cancer incidence among nonsmokers, 82–1272
Hawaiian men of Japanese ancestry, coronary disease risk factors, 82–1363
Indians, infant morbidity and parental smoking in Canada, 82–1533

EUROPE
lung cancer prevalence, smoking and air pollution as risk factors, 82–1287

EX-SMOKERS
Canada, 1965 to 1979, 82–1458
myocardial infarct incidence in Puerto Rico, 82–1370
sex ratio in Scottish adults, 82–1415

EYE DISEASES
alcohol, drug, and tobacco use relationship, 82–1395
tobacco—alcohol amblyopia, biochemical basis for pathogenesis, 82–1393

EYES
microcirculation in bulbar conjunctiva vessels, smoking effect, 82–1369
tobacco amblyopia patients, smoking and drinking habits and effect of abstinence, 82–1399

FATTY ACIDS
mediation of dietary and smoking effects on blood platelet function, 82–1364

FEAR
health behavior change, effect of fear-arousing communications, 82–1455

FEDERAL REPUBLIC OF GERMANY
bronchitis prevention through antismoking education and intervention, 82–1342

FETAL GROWTH
maternal diet vs. smoking habit, effects, 82–1388
national patterns in Argentina, maternal smoking effect, 82–1385
retardation, maternal smoking relationship, 82–1381

FETUS
brain stem damage, nicotine effect in rats, 82–1383
cadmium levels in cord blood and placental tissues, maternal smoking effect, 82–1386
lung maturation rate, United States vs. South Africa vs. Ethiopia, 82–1389
maternal smoking effect, 82–1409
nicotine effects on blood variables in sheep, 82–1380
secondary and tertiary smoking, cotinine in amniotic fluid, 82–1390

FILTERS
acetate vs. charcoal, effect on smoke components, 82–1511
lower-yield smoke, production method, 82–1501
lung cancer risk, filtered vs. nonfiltered cigarettes, 82–1294
monitoring puff number by filter color comparison, 82–1483
perforation of wrappers, method and apparatus, 82–1504

retention efficiency, testing method, 82–1215
sonic, selectively modulated filtration, 82–1499
toxicity reduction of cigarette smoke, 82–1508

FINLAND
health practices among 19-year-old males, smoking correlations, 82–1436
intermittent claudication, prevalence, risk factors, and mortality, 82–1365

FIRES
prevention, self-extinguishing cigarette, 82–1507

FORMALDEHYDE
minute ventilation response in the rat, probable involvement, 82–1311

FRANCE
coronary disease in Paris men, leukocyte count and smoking relationship, 82–1366

GAS PHASE, CIGARETTE SMOKE
carcinogen identification, 82–1293
toxicity reduction by dilution, 82–1508

GASTROINTESTINAL DISEASES
inflammatory bowel disease patients, smoking effect on weight, 82–1397
prevention, tobacco elimination as a practical approach, 82–1254
ulcers, smoking effects on antacid vs. cimetidine treatment, 82–1410

GASTROINTESTINAL NEOPLASMS
relative risk in Japan, smoking and dietary influences, 82–1278

GEOGRAPHIC LOCATIONS
esophageal cancer, worldwide incidence patterns, 82–1261

GERMAN DEMOCRATIC REPUBLIC
cigarette consumption, 82–1254
smoking prevalence and antismoking measures, 82–1270

GLYCERIN
acrolein precursor and cigarette additive, 82–1492

HAWAII
coronary heart disease risk factors among men of Japanese ancestry, 82–1363

HEAD AND NECK NEOPLASMS
alcohol, smoking, and dietary factors, 82–1267
synergistic effect of chronic ethanol consumption on carcinogenicity of nitrosamines in hamsters, 82–1217
tobacco use as risk factor, 82–1265

HEALTH EDUCATION
adolescents at risk for cardiovascular disease development, smoking education recommendation, 82–1379
adult attitudes toward smoking and drinking education for youth in Scotland, 82–1416
age-related efficacy of school antismoking campaigns, 82–1466
Australian students, effect on smoking habit, 82–1469
behavior change strategies, 82–1472
bronchitis prevention through antismoking education, 82–1342
Canadian resource guide to smoking and health materials and distributors, 82–1465
cigarette advertising vs. antismoking campaigns, recommendations for Great Britain, 82–1529
Federal data bases and resources for health educators, 82–1459
"feeder" approach for primary prevention of drug abuse, 82–1471
health hazard/health risk appraisal, evaluation of efficacy, 82–1479
prevention of smoking strategy, 82–1478
primary school smoking prevention program in Australia, evaluation, 82–1477
recognition of smoking-related health hazards by Swedish middle-aged men, 82–1433
role in public policy on smoking control, 82–1464
seat belt use promotion, effect in smokers vs. nonsmokers, 82–1422
sixth grade, smoking prevention strategies with carbon monoxide feedback, 82–1429
smoking's harmful effects, 82–1258
special strategies recommended for targeted groups in Australia, 82–1432
worldwide legislative actions, analysis, 82–1524

HEALTH PRACTICES
adolescent problem behavior, theoretical model, 82–1434
adolescents, model for a health behavior change program, 82–1476
adults in Massachusetts, 82–1439
attrition from health behavior modification program, smoking factor, 82–1454
behavior change strategies for health education, 82–1472
factory workers' profile, implications for occupational nurses, 82–1442
habits among 19-year-old Finnish males, smoking correlations, 82–1436
health hazard/health risk appraisal, evaluation of efficacy, 82–1479
lifestyle and occupation, relationship to carcinogenesis, 82–1285
medical vs. law students, 82–1423
seat belt use in smokers vs. nonsmokers, 82–1422

smoking cessation and behavior change programs, critique and recommendations, 82-1451
women, psychotropic drug use and smoking relationship, 82-1447

HEART
nicotine receptors, in the rat, 82-1224
ventricular muscle weight, smoke effect in rats, 82-1349

HEART DISEASES
coronary artery occlusion, alcohol and smoking effects, 82-1345
ischemic, leukocyte count and smoking relationship to incidence in Paris men, 82-1366
ischemic, smoking cessation effect on mortality, 82-1374
major risk factors, and basis of prevention, 82-1360
primary prevention via lifestyle change, 82-1371
problems in prevention, review, 82-1361
ventricular fibrillation and cardiac arrest occurrence, smoking association, 82-1375

HEART FUNCTION
cardiac output with rapid smoking of high- and low-nicotine cigarettes by younger vs. older subjects, 82-1484
exercise test for myocardial infarction risk factors, smoking effect, 82-1377
mechanical performance of isolated rat ventricular muscle, smoke exposure effects, 82-1349

HEART RATE
nicotine effect, 82-1347
rapid smoking effect with high- and low-nicotine cigarettes in younger vs. older subjects, 82-1484

HEMATOCRIT
chronic smoking effect in men vs. women, 82-1358
dose-response relationship with smoking, 82-1442
pregnant women, blood variables and smoking effect on pregnancy outcome, 82-1384

HEMODYNAMICS
chronic smoking effect in men vs. women, 82-1358
erythrocyte filterability in smokers vs. nonsmokers, 82-1348
hemostasis after cigarette smoking, 82-1369
nicotine effects on maternal and fetal blood in sheep, 82-1380

HEMOGLOBINS
filterability of erythrocytes, oxygen saturation factor, 82-1355
pregnant women, blood levels and smoking effect on pregnancy outcome, 82-1384

HETEROCYCLIC COMPOUNDS
mutagenic capacity, bacterial tests, 82-1241

HORMONES
antidiuretic, nicotine receptors effect on acetylcholine mechanism of action in goats, 82-1246

HUMECTANTS
glycerol and glycols in cigarettes, health risks, 82-1492

HYPERTENSION
alcohol consumption and smoking relationship, 82-1344
blood platelet aggregation, propanolol, exercise and smoking effects, 82-1367
heart disease risk factor, with smoking, lipid abnormalities, diabetes, 82-1360

HYPOXIA
family correlation, COPD patients vs. sons, 82-1322
oxygen therapy in chronic obstructive pulmonary disease patients, smoking effect, 82-1308

IMMUNE SYSTEM
biological dust exposure and smoking effects, 82-1403
immunoglobulin E levels in smoking male bronchial cancer patients, 82-1274
salivary immunoglobulin A level in smokers, nonsmokers, and patients with minor aphthous ulceration, 82-1391

IMMUNOGLOBULINS
immunoglobulin E levels in smoking male bronchial cancer patients, 82-1274
saliva immunoglobulin A, levels in smokers, 82-1391

INDIA
blood pressure in males aged 8 to 16, smoking effects, 82-1368
bronchogenic cancer epidemiology, cigarette, bidi, and hookah smoking relationship, 82-1279

INFANT MORTALITY
maternal smoking and sudden infant death syndrome relationship, 82-1383

INFANTS
respiratory morbidity in Indians vs. non-Indians, parental smoking factor, 82-1533

INFORMATION SERVICES
Canadian resource guide to smoking and health materials and distributors, 82-1465
Federal data bases and resources for health educators, 82-1459

INTERMITTENT CLAUDICATION
prevalence in Finland, risk factors and mortality, 82-1365

IRELAND
lung cancer status, smoking factor, 82-1302

JAPAN
  gastric cancer vs. lung cancer incidence, smoking and dietary effects, 82–1278
  occupational cancer, smoking relationship, 82–1277

KENYA
  aldrin replaced by orthene for tobacco crop spraying, toxicity problems, 82–1235

KETONES
  alpha, beta unsaturated variety for tobacco flavor and aroma, 82–1497

KIDNEY NEOPLASMS
  analgesics and tobacco risk factors in renal pelvic cancer, 82–1283

LABELING
  dependency warning in addition to health warning on cigarette package, proposal, 82–1520
  worldwide legislative restrictions, analysis, 82–1524

LARYNGEAL NEOPLASMS
  alcohol, tobacco, and oral hygiene as risk factors, 82–1265
  occupational exposure risk, smoking factor, 82–1268

LEARNING
  state-dependent learning, smoking effects, 82–1446

LEGISLATION
  advertising and labeling regulations in Switzerland, 82–1460
  carcinogens, Federal regulations, 82–1221
  controls and restrictions on tobacco use, suggestions, 82–1523
  government regulation effect on teenage smoking, 82–1522
  prohibition on sale of cigarettes to minors in New Zealand, effect, 82–1521
  public policy on cigarette control, overview, 82–1464
  "safety tax" proposal for cigarettes, 82–1520
  smoking control measures recommended in Australia 82–1530
  tobacco sales ban for minors, low awareness in Canadian pharmacists, 82–1456
  workplace smoking regulations, Federal and State proposals, 82–1525
  worldwide smoking control measures, analysis, 82–1524

LEUKOCYTES
  coronary disease relationship with smoking in Paris men, 82–1366

LEUKOPLAKIA
  leukoplakia-like lesions in discoid lupus erythematosis patients, tobacco factor, 82–1408
  reduction or elimination with cessation of smoking, 82–1406

LIP NEOPLASMS
  sex ratio in Los Angeles County, tobacco relationship, 82–1291
  sunlight and tobacco as risk factors, 82–1265

LIPIDS
  coronary disease, high density lipoprotein cholesterol vs. lipids vs. smoking in intervention trials, 82–1357
  heart disease risk factor, with smoking, hypertension and diabetes, 82–1360

LIPOPROTEINS
  coronary disease, high density lipoprotein cholesterol as predictor, 82–1357
  high density cholesterol/smoking relationship, adolescent cardiovascular risk investigation, 82–1379
  high density lipoprotein cholesterol in younger vs. older men, smoking factors, 82–1346
  levels in progeny of parents who died of cancer or heart disease, smoking factor, 82–1282
  plasma concentrations, smoking cessation effect on, 82–1372

LONGEVITY
  lung cancer survival, factors, 82–1302

LOUISIANA
  lung cancer mortality in sugarcane workers, 82–1295

LUNG DISEASES
  smoking in etiology, preventive recommendations, 82–1286
  tobacco flavor additives as risk factors, 82–1492

LUNG FUNCTION
  airway response to smoking, tar level effects, 82–1333
  bagasse workers, smokers vs. nonsmokers, 82–1329
  biological dust exposure and smoking effects, 82–1403
  cessation of smoking effect, review, 82–1255
  clean vs. smoke-filled work environment, passive smoking effect, 82–1336
  coal dust exposure and smoking effects, 82–1326
  declining respiratory function in predicting obstructive airways disease, 82–1309
  electric welders, smokers vs. nonsmokers, 82–1335
  expiratory volume, age, height and weight, effects in smokers vs. nonsmokers, 82–1318
  flax mill workers, flax dust vs. smoking effects, 82–1313
  impairment effects on short-term mortality, 82–1307
  machine shop workers, occupational environment and smoking effect, 82–1330
  methacholine inhalation effects in smokers vs. nonsmokers, 82–1327
  monitoring device, noninvasive, 82–1312

occupational dust exposure vs. smoking effect, 82–1305
oil mist exposed metal workers, impairment in smokers vs. nonsmokers, 82–1320
ozone exposure responses, smoking effect, 82–1324
prediction equations and reference values, 82–1316
snuff effects, 82–1332

LUNG NEOPLASMS
air pollution, occupational and smoking risk factors, 82–1300
environmental carcinogens and smoking in etiology, 82–1293
filtered vs. nonfiltered cigarettes, risk, 82–1294
gastric cancer incidence in Japan comparison, smoking and dietary effect, 82–1278
incidence and smoking prevalence in East Germany, 82–1270
mast cell differences in respiratory epithelium of smoking vs. nonsmoking patients, 82–1281
miners, radon daughter exposure and smoking in etiology, 82–1292
mortality in sugarcane workers, smoking relationship, 82–1295
mortality in Sweden, occupational arsenic exposure and smoking interaction, 82–1288
nonsmoking Chinese women, incidence, 82–1272
occupational vs. smoking effect on incidence, 82–1298
onset rates, U.S. trends, 82–1289
passive smoking as risk factor, 82–1400
patients with long-term survival, smoking behavior, 82–1264
prevalence and prevention in the Soviet Union and Europe, 82–1287
prevalence patterns in United States, smoking relationship, 82–1259
prior tubercular infection in etiology in smoking vs. nonsmoking women, 82–1276
reduced risk with smoking cessation, 82–1255
routine screening and early diagnosis, effect on survival rates, 82–1271
smoking in etiology, preventive recommendations, 82–1286
survival factors, 82–1302
synergistic and/or cocarcinogenic factors in etiology, 82–1301
talc workers in upstate New York, smoking vs. occupational risks, 82–1257

LUNGS
benzo(*a*)pyrene binding and metabolism in rat lung, 82–1223
cytotoxicity of cigarette smoke condensate on polymorphs, 82–1319
Cytrel-containing cigarettes, effect in rats, 82–1310
maturation in fetuses of smoking vs. nonsmoking mothers in Ethiopia, South Africa, and the United States, 82–1389

LYMPHOCYTES
chromosomal aberrations in smokers vs. nonsmokers, 82–1236, 82–1248
sister-chromatid exchange rates, smoking effect, 82–1228
sister-chromatid induction by smoke condensate, 82–1243

MARIHUANA SMOKING
guilt/shame association with use, smoking habit similarities, 82–1419
high school seniors, 1975-1981, 82–1435
predicting adolescent substance abuse from first grade personality factors, 82–1437

MASS MEDIA
cigarette advertising in the United States, novel approach, 82–1516
magazine survey for smoking/health coverage, 82–1528
public response to media coverage of less hazardous cigarettes study, critical analysis, 82–1474

MASSACHUSETTS
prevalence of smoking among adults, 82–1439

MATERNAL SMOKING
bronchitis incidence in infants and children, effect with other risk factors, 82–1339
cadmium levels in blood and placental tissues, relationship, 82–1386
consequences to infant, review, 82–1532
diet effect on birth weight, comparison, 82–1388
fetal growth patterns in Argentina, effect, 82–1385
intrauterine growth retardation factor, 82–1381
lung maturation of fetus effect, in the United States vs. South Africa vs. Ethiopia, 82–1389
nicotine effects on blood variables in pregnant sheep, 82–1380
pregnancy outcome, effect with maternal hematologic level, 82–1384
premature rupture of fetal membranes, relationship, 82–1387
sudden infant death syndrome relationship, 82–1383

MATHEMATICAL MODELS
computer simulation model of smoking for policy development, 82–1448
lung function prediction equations and reference values, 82–1316
premature mortality from cigarette smoking vs. road crashes in Australia, 82–1251
single vs. multiple cancer risk exposures, formulas, 82–1266

MEMORY
state-dependent learning, smoking effects, 82-1446

METALS, METAL ISOTOPES
aluminum, bladder cancer risk factor, 82-1266
manganese, source in cigarette smoke and ash, 82-1214
protective colloid to improve stability of palladium in tobacco casing systems, 82-1500

MORBIDITY
acute respiratory tract illness, smoking factor, 82-1304
association with alcohol, tobacco, and drugs, 82-1252
cancer, occupational factors, 82-1290
cancer onset rates, U.S. trends, 82-1289
cancer, trends and estimates of cause, 82-1263
carcinogenesis, smoking as factor, 82-1269
elderly men in Sweden, smoking effects, 82-1256
occupational cancer in Japan, interactive effect of smoking, 82-1277
predicting obstructive airways disease in smokers vs. nonsmokers with lung function tests, 82-1309
respiratory illness among Australian waterside workers, risks of smoking vs. asbestos exposure, 82-1328
smoking cessation effect on lung cancer, arteriosclerosis, and pulmonary disease, 82-1255
smoking-related, review, 82-1532
vasectomized vs. nonvasectomized men, smoking relationship, 82-1405
welders vs. nonwelders, smoking effect, 82-1314

MORTALITY
all causes in Busselton, Australia, smoking and alcohol consumption relationship, 82-1253
all diseases, lung function impairment and smoking effects, 82-1307
association with alcohol, tobacco, and drugs, 82-1252
cancer and smoking relationship, 1982 report of the Surgeon General, 82-1299
cancer, endogenous psychosocial factors, 82-1425
cancer etiology, smoking as factor, 82-1269
cancer, occupational factors, 82-1290
cancer or heart disease of parents, relationship to risk factors in progeny, 82-1282
cancer rates, U.S. trends, 82-1273, 82-1289
concurrent decrease in rate with tobacco consumption decrease in New Zealand, 1976-1981, 82-1468
coronary disease in the Soviet Union, 82-1352
coronary disease rates, reduction in smoking correlation, 82-1371
coronary disease, review of problems in prevention, 82-1361
gastric vs. lung cancer in Japan, smoking and dietary relationship, 82-1278
intermittent claudication effect, with smoking and other risk factors, 82-1365
ischemic heart disease, smoking cessation relationship, 82-1374
lung cancer in miners, radon daughter exposure and smoking relationship, 82-1292
lung cancer in sugarcane workers, smoking relationship, 82-1295
lung cancer in Sweden, occupational arsenic exposure and smoking interaction, 82-1288
lung cancer patterns, smoking relationship, 82-1259
lung cancer screening, effect on survival rates, 82-1271
male talc workers in upstate New York, 82-1257
post-myocardial infarction, smoking cessation effect, 82-1378
road crashes vs. smoking-related in Australia, 82-1251
smoke-exposed mice, reduction with citrinin treatment, 82-1226
smoking-related, review, 82-1532

MOTIVATION
adolescent problem behavior, theoretical model, 82-1434
attrition from health behavior modification program, smoking factor, 82-1454
avoidance of rejection, correlation to drug use, 82-1449
health behavior change, effect of fear-arousing communications, 82-1455
nicotine self-administration and dependence in animal studies, 82-1428
physicians' advice, effect on smoking cessation, 82-1452, 82-1462, 82-1473

MOUTH NEOPLASMS
alcohol consumption as a promoter in smokers, 82-1301
alcohol, tobacco, and oral hygiene as risk factors, 82-1265
early diagnosis in smoking and drinking patients, 82-1275
Herpes simplex virus type 1 and smoking in etiology, 82-1297
immunosuppressive effect of tobacco combustion products, 82-1391
Los Angeles County, incidence and relationship of smoking, 82-1291
snuff dipping relationship, 82-1296

MUSCLES
blood supply, nicotine and smoke effects in rats, 82-1240

MUTAGENESIS
lymphocyte chromosomal aberrations in smokers vs. nonsmokers, 82-1236
sister-chromatid exchange analysis of cigarette smoke condensate, 82-1243
sister-chromatid exchange rates in lymphocytes, smoking effect, 82-1228

MUTAGENS
bacterial test of tobacco constituents, 82–1241
lymphocyte chromosomal aberrations in smokers vs. nonsmokers, 82–1248

MYOCARDIAL INFARCT
before age 36, smoking risk factor, 82–1354
cessation of smoking and diet, effect on incidence in Oslo study, 82–1356
middle-aged men, exercise test for risk factor identification, 82–1377
nicotine relationship, 82–1376
Paris men, incidence and relationship of leukocyte count and smoking, 82–1366
post-infarction mortality, smoking cessation effect, 82–1378
Puerto Rican men, smoking relationship, 82–1370
ventricular fibrillation and cardiac arrest occurrence, smoking association, 82–1375

NASOPHARYNGEAL NEOPLASMS
Los Angeles County, incidence and relationship of smoking, 82–1291
nitrosamine application and ethanol consumption, synergistic effects in hamsters, 82–1217

NEONATE
fetal growth patterns in Argentina, maternal smoking effect, 82–1385
hyaline membrane disease occurrence relative to maternal smoking in Ethiopia, South Africa, and the United States, 82–1389
poor muscle tone and diminished reflex activity, maternal smoking relationship, 82–1381

NEOPLASMS
asbestos-associated, review, 82–1262
causative factors, 82–1263, 82–1293
cell culture assay for detection of tumor promoters and inhibitors, 82–1260
cigarette additives relationship, 82–1492
endogenous psychosocial factors effect on incidence, 82–1425
etiological factors, smoking relationship, 82–1269
less hazardous cigarettes, critical analysis of disease risk report, 82–1474
mortality in parents vs. serum cholesterol and serum lipid levels in progeny, smoking factor, 82–1282
occupation-related in Japan, smoking factor, 82–1277
occupational factors relationship to incidence and deaths, 82–1290
onset rates, U.S. trends, 82–1289
opioid peptide and catecholamine secretion from human pheochromocytoma, nicotine effect in vitro, 82–1249
prevention, tobacco elimination as a practical approach, 82–1254
smoking-relationship, review, 82–1532
smoking relationship, 1982 report of the Surgeon General, 82–1299
U.S. mortality trends, 1950-1977, 82–1273

NEURONS
changes in potential in toxic optic neuropathy patients, smoking effect, 82–1399
nicotine receptors in the rat heart, 82–1224
nicotinic receptors in the cat, blocking by phenoxybenzamine or acetylcholine, 82–1230

NEW YORK STATE
lung cancer incidence among men of Erie County, risks, 82–1300
mortality in talc workers, 82–1257

NEW ZEALAND
adolescents, smoking attitudes, 82–1444
legislation vs. education in discouraging adolescent smoking, 82–1521
smoking control measures, 82–1468

NICOTINE
antinociception in rats and mice, 82–1245
blood variables in pregnant sheep, effect on, 82–1380
bronchial reactivity to inhaled methacholine in baboons, effect on, 82–1341
capillary measurement in rats, effect, 82–1240
cardiovascular diseases, role in etiology and aggravation, 82–1376
cardiovascular effects and metabolism, 82–1347
catecholamine levels in rat brain, effect, 82–1242
fetal brain stem damage in rats, relationship, 82–1383
hepatic microsomal mixed function oxidases, effect in rats, 82–1280
ischemic heart disease, risk factor, 82–1362
less hazardous cigarettes, critical analysis of disease risk report, 82–1474
opioid peptide and catecholamine secretion from human pheochromocytoma tumor, effect in vitro, 82–1249
physiological arousal, state-dependent learning relationship, 82–1446
plasma level, smoking characteristics relationship, 82–1418
receptors in the rat heart, ^{3}H-noradrenaline release effect, 82–1224
renal water and salt excretion in goats, effect with acetylcholine, 82–1246
self-administration in monkeys in drug-paired stimulus response experiments, 82–1426
teratogenic effects with barbiturates, caffeine, and aspirin on chick embryo, 82–1382
tyrosine in treatment of withdrawal symptoms, 82–1234
wrapper perforation method and apparatus for dilution, 82–1504

NICOTINE CONTENT
  wrapper perforating apparatus to influence smoke content, 82–1498

NICOTINE DERIVATIVES
  iminium ion intermediate, reactions with glutathione and cyanide, 82–1227

NICOTINE METABOLISM
  iminium ion intermediate, reactions with glutathione and cyanide, 82–1227

NICOTINE PHARMACOLOGY
  antinociception in rats and mice, 82–1245
  blocking of nicotinic receptor mechanism in the cat adrenal gland, 82–1230
  catecholamine levels in rat brain, mecamylamine and hexamethonium interactions, 82–1242
  central nervous system binding sites, localization by radiolabeling technique, 82–1232
  citrinin antagonism in mice, 82–1226
  dependence and self-administration in animal studies, 82–1428
  ^{3}H-noradrenaline overflow in rat heart, nicotine and p-aminophenethyltrimethylammonium effect, 82–1224
  human fibroblast culture, smoke extracts effects, 82–1315
  mutagenic capacity of tobacco constituents, bacterial tests, 82–1241
  naloxone reversal of nicotine-enhanced 5-hydroxytryptamine synthesis, 82–1222
  nicotine vs. narcotic vs. amphetamine in drug generalization in rats, 82–1231

NICOTINE REDUCTION
  bluish dogbane in Chinese cigarettes, effect, 82–1503
  compensation through puffing pattern changes, 82–1229
  intake monitoring by filter color comparison, 82–1483

NITROGEN CONTENT
  bright vs. Burley varieties, protein and nitrate content, 82–1216

NITROGEN OXIDES
  ambient nitrogen dioxide and smoking relationship to respiratory illness in Chattanooga, Tennessee, 82–1325
  enzyme-inhibitory effects, 82–1239

NITROSAMINES
  chronic ethanol consumption and its synergistic effect on etiology of head and neck cancer in hamsters, 82–1217
  nitrosonornicotine, principal tobacco carcinogen, 82–1301

NONSMOKERS
  bronchial reactivity to methacholine compared with smokers, 82–1327
  chronic bronchitis incidence in Vienne, Austria, smokers comparison, 82–1323
  discounted life insurance premiums in the United Kingdom, 82–1531
  health effects of passive smoking, 82–1336, 82–1394, 82–1409
  lung cancer incidence among Chinese women, 82–1272
  lung cancer incidence, occupational risk factors, 82–1298
  lung cancer risk in women, prior tubercular infection in etiology, 82–1276
  passive smoking, health risk controversy, 82–1400, 82–1407
  segregation from smokers, passive inhalation of smoke, 82–1404
  workplace exposure to cigarette smoke, health effects, 82–1398, 82–1526
  workplace, legal protection vs. rights of smokers, 82–1525

NORNICOTINE
  National Toxicology Program, carcinogen study, 82–1221

NORTH CAROLINA
  oral cancer mortality from snuff dipping among women, 82–1296
  schoolchildren, drug abuse primary prevention program, 82–1471

NORWAY
  acute myocardial infarction incidence in Oslo study, smoking cessation and diet effect, 82–1356

NOSE NEOPLASMS
  incidence in Los Angeles County, 82–1291

OBSTRUCTIVE AIRWAY DISEASES
  cessation of smoking, effect on risk, 82–1255
  cytotoxicity of cigarette smoke condensate on polymorphs of patients, 82–1319
  declining respiratory function and smoking as risk factors, 82–1309
  familial factors affecting blood gas values in patients, smoking relationship, 82–1322
  ozone exposure effect on patients, smoking relationship, 82–1324
  secondary polycythemia patients, smoking effect on treatment, 82–1308
  theophylline clearance in smokers, age factors, 82–1306

OCCUPATIONAL DISEASES
  byssinosis in flax mill workers, smoking effect, 82–1313

cancer incidence and deaths, occupational factors, 82-1263, 82-1290
cancer of all sites in Japan, interactive effect of smoking, 82-1277
laryngeal cancer, smoking factor, 82-1268
lung cancer, confounding and interactive factors in quantification, 82-1285
lung cancer in sugarcane workers, smoking relationship, 82-1295
lung cancer mortality in Sweden, arsenic exposure and smoking interaction, 82-1288
lung cancer, occupation vs. air pollution vs. smoking as risks, 82-1300
lung cancer, occupational exposure vs. smoking relationship, 82-1298
pesticide toxicity risks in tobacco crop spraying in Kenya, 82-1235
pneomoconiosis, dust exposure vs. smoking effect on lung function, 82-1305
pulmonary function impairment in welders, 82-1303
Raynaud's phenomenon among forestry workers in Quebec, smoking effects, 82-1373

OCCUPATIONS
agricultural workers, 82-1365
aluminum workers, 82-1266
asbestos workers, 82-1262
Australian waterside workers, 82-1328
bagasse workers, 82-1329
coal miners, 82-1326
copper smelter workers, 82-1288
electric welders, 82-1335
factory workers, 82-1442
farmers, 82-1364
firefighters, 82-1244
flax mill workers, 82-1313
forestry workers, 82-1373
machine shop workers, 82-1330
metal workers, 82-1277, 82-1320
miners, 82-1292
nurses, 82-1450
pharmacists, 82-1456
Pruteen plant workers, 82-1403
sugarcane farming, 82-1295
talc workers, 82-1257
tobacco crop farmers, 82-1235
welders, 82-1303, 82-1314

ORAL HYGIENE
mouth cancer risk factor, smoking and alcohol relationship, 82-1265

OVARIES
fertility, review of smoking effects, 82-1402

OXYGEN TRANSPORT
secondary polycythemia patients, smoking effect on treatment, 82-1308

PAKISTAN
tobacco industry in 1981, prospects for 1982, 82-1512

PARENTAL SMOKING
adolescent smoking factor, model for behavior change, 82-1476
ambient nitrogen dioxide and smoking relationship to respiratory illness in Chattanooga, Tennessee, 82-1325
asthma in children, relationship, 82-1317
behavioral influence on initiation, maintenance, and cessation of smoking, 82-1478
bronchitis and pneumonia incidence in children, relationship, 82-1409
perceived influence on children's smoking and drinking habits in Scotland, 82-1416

PASSIVE SMOKING
cotinine in amniotic fluid, effect on fetus, 82-1390
health consequences, 1982 report of the Surgeon General, 82-1299
health hazards, 82-1336, 82-1394, 82-1409, 82-1414
health risk controversy, 82-1400, 82-1407
indoor smoke concentrations, review, 82-1413
segregation of smokers, effectiveness, 82-1404
workplace exposure, health and economic consequences, 82-1398
workplace exposure, legal aspects, 82-1525

PATENTS
acyclic carboxamides having a menthol effect, 82-1493
alpha, beta unsaturated ketones for tobacco flavor and aroma, 82-1497
carbon monoxide and total particulate matter reduction with variable-density smoking article, 82-1506
caryophyllene alcohol mixtures as flavor enhancers, 82-1490
cellulosic smoking material, modification and method of preparation, 82-1505
chewing tobacco flavor compositions, 82-1489
cigarette modification with a disc to expand smoke for flavor, 82-1487
cyclohexene and cyclohexadiene methanols as flavorants, 82-1496
cyclohexene methanols as tobacco flavorants, 82-1494
6-hydroxy-2, 6-dimethylheptanal and intermediates, as tobacco flavorants, 82-1495
hydroxyethyl-substituted pyrazine and pyridine compounds as tobacco flavorant additives, 82-1488
lower-yield smoke, filter production method, 82-1501
1-N-butoxy-1-ethanol acetate as flavorant, 82-1486
packaged cigarettes perforating device, 82-1509
perforating apparatus for tobacco product wrappers, 82-1498
perforation of smoking material wrappers, method and apparatus, 82-1504

- protective colloid to improve stability of palladium in tobacco casing systems, 82–1500
- self-extinguishing cigarette, 82–1507
- sonic filter with modulated selectivity, 82–1499
- spirane derivatives for flavoring tobacco, 82–1491
- tobacco expansion process using carbon dioxide, 82–1502, 82–1510

PEER GROUPS
- adolescent problem behavior factor, theoretical model, 82–1434
- adolescent smoking factor, model for behavior change, 82–1476
- adolescents, perceived peer attitudes' influence on smoking habit, 82–1444
- adolescents, training to resist peer pressure to smoke, 82–1467
- behavioral influence on initiation, maintenance, and cessation of smoking, 82–1478

PENNSYLVANIA
- adult women, respiratory symptoms in smokers vs. ex-smokers vs. nonsmokers, 82–1334

PEPTIC ULCER
- antacid vs. cimetidine vs. placebo treatment, smoking effect on healing, 82–1410
- healing rates, smokers vs. nonsmokers, 82–1392
- sucralfate vs. cimetidine treatment, smoking effect on healing 82–1401
- vasectomized vs. nonvasectomized men, smoking relationship, 82–1405

PERCEPTION
- adolescents, perceived parental and peer pressure vs. benefits and hazards of smoking, 82–1444
- adolescents, self-images and smoking status/intentions relationship, 82–1421
- harmfulness of selected drugs including nicotine, by college students, 82–1457
- recognition of smoking-related health hazards by Swedish middle-aged men, 82–1433

PERSONALITY
- avoidance of rejection, correlation to drug use, 82–1449
- predicting adolescent substance abuse from first grade personality factors, 82–1437

PESTICIDES
- aldrin, replaced by orthene to reduce toxicity in agricultural tobacco workers in Kenya, 82–1235

PHAGOCYTOSIS
- fibronectin in pulmonary alveolar macrophages of smokers vs. nonsmokers, 82–1340

PHARYNGEAL NEOPLASMS
- alcohol, tobacco, and oral hygiene as risk factors, 82–1265
- early diagnosis in smoking and drinking patients, 82–1275
- Los Angeles County, incidence and relationship of smoking, 82–1291

PHYSICAL ACTIVITY
- blood platelet aggregation, effect with smoking and propanolol, 82–1367
- exercise test for myocardial infarction risk factors, smoking relationship, 82–1377
- South Australians, inverse relationship to smoking, 82–1430

PIPE SMOKING
- Canada, incidence in 1965 to 1979, 82–1458
- lip cancer relationship, 82–1265
- myocardial infarct incidence in Puerto Rico, relationship, 82–1370

PLACENTA
- cadmium levels, smoking effect, 82–1386

PNEUMOCONIOSIS
- bagassosis, symptoms in smoking vs. nonsmoking bagasse workers, 82–1329
- byssinosis in flax mill workers, smoking effect, 82–1313
- occupational dust exposure vs. smoking effect on pulmonary function, 82–1305

PRECANCEROUS CONDITIONS
- leukoplakia, cessation of smoking effect, 82–1406

PREGNANCY
- cadmium levels in blood and placental tissues, maternal smoking effect, 82–1386
- maternal diet vs. smoking effect on birth weight, 82–1388
- maternal hematologic levels and smoking, effect on fetus, 82–1384
- nicotine effects on blood variables in sheep, 82–1380
- premature rupture of fetal membranes, maternal smoking relationship, 82–1387
- smoking effects on fertility, review, 82–1402

PREMATURITY
- rupture of fetal membranes, maternal smoking relationship, 82–1387

PREVENTION OF SMOKING
- adolescents, health behavior change program model, 82–1476
- adolescents, program and health effects review, 82–1532
- adolescents, training to resist peer pressure, 82–1467

carbon monoxide feedback strategies in sixth grade students, 82-1429
"feeder" approach for primary prevention of drug abuse, 82-1471
primary school education program in Australia, evaluation, 82-1477
prohibition, regulation, and health education strategies, 82-1478
recommendations, 82-1258
total ban on tobacco ads recommended, 82-1529
worldwide antismoking legislation, analysis, 82-1524

PROGRAMS AND POLICIES
computer simulation model of smoking for policy development, 82-1448
national approaches to smoking control, effectiveness factors, 82-1470
New Zealand smoking control measures, 82-1468
workplace health and smoking cessation programs, guidelines, 82-1475
worldwide smoking control legislation, analysis, 82-1524

PROTEIN CONTENT
bright vs. Burley varieties, 82-1216, 82-1219

PSYCHIATRIC DISORDERS
tranquilizing effects of chlorpromazine, smoking influence, 82-1237

PUERTO RICO
coronary disease in middle-aged men, smoking relationship, 82-1370

PULMONARY ALVEOLAR MACROPHAGES
fibronectin content of bronchoalveolar fluid in smokers vs. nonsmokers, relationship, 82-1340

PULMONARY FIBROSIS
collagen synthesis and cell growth, tobacco smoke extracts effect, 82-1315

RADIATION
occupational exposure, cancer incidence and death relationship, 82-1290
radon daughter exposure and lung cancer in miners, smoking relationship, 82-1292
skin neoplasm induction in rats, with cigarette smoke condensate, 82-1284

RECIDIVISM
acupuncture treatment effect, 82-1481
drug-paired stimuli in maintenance of drug self-administration, nicotine response in monkeys, 82-1426
telephone followup intervals, effect on smoking cessation maintenance, 82-1480

REDUCTION OF SMOKING
fear-arousing communications, 82-1455
rapid smoking, physiological consequences, 82-1484
telephone followup intervals, effect, 82-1480
weekly target-setting program, self- vs. therapist-paced, 82-1482

RESPIRATORY FUNCTION TESTS
airway resistance measurements, tar level effects, 82-1333
bagasse workers, smokers vs. nonsmokers, 82-1329
electric welders, smokers vs. nonsmokers, 82-1335
forced expiratory volume impairment, effect on short-term mortality, 82-1307
forced vital capacity, coal dust and smoking effects, 82-1326
maximum expiratory flow, age, height, weight and smoking effects, 82-1318
metacholine inhalation effects in smokers vs. nonsmokers, 82-1327
minute ventilation changes in rat, smoke inhalation study, 82-1311
occupational dust exposure vs. smoking effect, 82-1305
oil mist exposed metal workers, smokers vs. nonsmokers, 82-1320
ozone exposure responses, smoking effect, 82-1324
prediction equations and reference values, 82-1316
respiratory inductive plethysmography vs. spirometry, 82-1312
snuff effects, 82-1332
vital capacity and forced expiratory volume, value in predicting obstructive airways disease, 82-1309
welders, smoking and welding fumes effect, 82-1303

RESPIRATORY SYMPTOMS
chronic cough and phlegm, oil aerosol and smoking relationship in machine shop workers, 82-1330
cough, phlegm, wheeze and dyspnea, tar content and smoke inhalation relationship, 82-1334
frequency in patients with acute respiratory tract illness, smokers vs. nonsmokers, 82-1304
oil mist exposed metal workers, smokers vs. nonsmokers, 82-1320

RESPIRATORY SYSTEM
Cytrel-containing cigarettes, effect in rats, 82-1310
mast cell differences in respiratory epithelium of smoking vs. nonsmoking lung cancer patients, 82-1281

RESPIRATORY TRACT DISEASES
acute respiratory illness, incidence in smokers vs. nonsmokers, 82-1304
ambient nitrogen dioxide and smoking, relationship in Chattanooga, Tennessee, 82-1325
cyanide as toxicant, 82-1337
familial factors in COPD and silicosis patients vs. sons, 82-1322

Indian infants in Canada, parental smoking as a factor, 82–1533
morbidity among Australian waterside workers, risks of smoking vs. asbestos exposure, 82–1328
prevention, tobacco elimination as a practical approach, 82–1254
smoking relationship, 82–1258
welding fumes and smoking effects in welders, 82–1314

SALIVA
immunoglobulin A levels in smokers, 82–1391

SAUDI ARABIA
carboxyhemoglobin levels in sheesha vs. cigarette smokers, 82–1250

SCOTLAND
adult attitudes toward underage drinking and smoking, 82–1416
adults, smoking survey, 82–1415
blood platelet function in farmers, diet, alcohol and smoking effects, 82–1364

SEX BEHAVIOR
puffing characteristics among male and female smokers, 82–1418

SEX RATIO
adult smoking habits in Scotland, 82–1415
chronic smoking effect on blood hemodynamics, 82–1358
cigarette consumption patterns, Great Britain 1972-1980, 82–1445
gastric cancer in Japan, smoking and diet factors, 82–1278
high-risk behavior among adults in Massachusetts, 82–1439
immunoglobulin E levels among bronchial cancer patients, 82–1274
prescription, over-the-counter, and social drug use among college students, 82–1457
smoking behavior and opinions in South Australia, 82–1430
smoking habits in Australia, 1980 survey, 82–1432
smoking habits in Canada, 1965 to 1979, 82–1458
smoking habits in East Germany, 82–1270
smoking habits in the Soviet Union, 82–1352
upper respiratory tract cancer incidence in Los Angeles County, tobacco relationship, 82–1291
ventricular fibrillation and cardiac arrest, sex, age and smoking as predictors, 82–1375

SINGAPORE
blood pressure in adults, smoking effect, 82–1359

SKIN DISEASES
leukoplakia-like lesions in discoid lupus erythematosis patients, tobacco factor, 82–1408

SKIN NEOPLASMS
radiation and cigarette smoke condensate induction in rats, 82–1284

SMOKE INHALATION, ANIMAL
bronchial mucous cell increase, effect of anti-inflammatory agents, 82–1321
capillary measurement in rats, smoke exposure effects, 82–1240
citrinin effects on exposure in mice, 82–1226
Cytrel-containing cigarettes, effect on rat lungs, 82–1310
mechanical performance of isolated rat ventricular muscle, effect on, 82–1349
minute ventilation changes at different concentrations, 82–1311

SMOKE STREAMS
constituents of mainstream and sidestream smoke, passive smoking effects, 82–1409
contribution to indoor air pollution, review of testing methods, 82–1220
filter retention efficiency, testing method and tobacco composition effects, 82–1215
hydroxyethyl-substituted pyrazine and pyridine compounds as flavorant additives, 82–1488
indoor pollutants from cigarettes, review, 82–1413
passive exposure at work, health and economic consequences, 82–1398
sidestream smoke elimination with self-extinguishing cigarettes, 82–1507

SMOKING
sheesha vs. cigarette, carboxyhemoglobin levels in Saudi Arabian males, 82–1250

SMOKING ARTICLES
carbon monoxide and total particulate matter reduction with variable-density smoking article, 82–1506
cigarette fire extinguisher, 82–1507
device for perforating packaged cigarettes, 82–1509

SMOKING CHARACTERISTICS
inhalation and respiratory symptoms relationship in women, 82–1334
inhalation patterns and measurement of exposure to smoke constituents, 82–1247
monitoring puff number by filter color comparison, 82–1483
puffing behavior among male and female smokers, 82–1418
puffing pattern changes in smokers of low tar/nicotine cigarettes, 82–1229

tax based on cigarette tar—nicotine levels, effect, 82–1514

SMOKING CONTROL PROGRAMS
- American Cancer Society's "Target 5" program, success rates, 82–1461
- Australia, critique of government role and recommendations, 82–1530
- Australia, primary school education program, evaluation, 82–1477
- critique of programs and recommended changes, 82–1451
- East Germany, 82–1270
- factors affecting efficacy, 82–1470
- legal restrictions suggested, 82–1523
- New Zealand, 82–1468
- psychological considerations for proper regimens, 82–1463
- public policy on cigarette control, overview, 82–1464
- schoolchildren, drug abuse primary prevention program, 82–1471
- Switzerland, 82–1460
- workplace programs, 82–1398, 82–1475, 82–1526
- worldwide legislation, analysis, 82–1524

SMOKING HABIT
- adolescents, cigarette-related cognition and social perceptions, 82–1440
- adolescents, beliefs and attitudes in New Zealand, Australia, and the United States, 82–1444
- adolescents, government regulation effect, 82–1522
- adolescents, self-images and smoking status/intentions relationship, 82–1421
- adult women, respiratory symptoms relationship, 82–1334
- adults and adolescents in Austria, 82–1427
- adults and adolescents in Canada, 82–1417, 82–1458
- adults and adolescents in East Germany, 82–1254
- adults in Australia, 82–1432
- adults in Canada, cigarette type preferences, 82–1519
- adults in East Germany, lung cancer incidence relationship, 82–1270
- adults in Massachusetts, 82–1439
- adults in Scotland, 82–1415
- adults in Singapore, effect on blood pressure, 82–1359
- adults in South Australia, 82–1430
- adults in the Soviet Union, 82–1352
- bronchogenic cancer patients in India, cigarette, bidi, and hookah use, 82–1279
- carcinogenesis, vitamin A deficiency, occupation, and lifestyle interactions, 82–1285
- college students, comparison with drug and alcohol use, 82–1457
- coronary disease relationship in Puerto Rican men, 82–1370
- dependency aspect, warning on labels proposal, 82–1520
- elderly men in Sweden, 82–1256
- guilt/shame association compared to alcohol, coffee, and marihuana use, 82–1419
- high school seniors, 1975-1981, 82–1435
- individual variables, implications for intervention strategies, 82–1443
- leukoplakia, relationship, 82–1406
- lung cancer patients with long-term survival, 82–1264
- male draftees in Switzerland, 82–1453
- medical and law students, 82–1423
- men in Sweden, practice and health beliefs, 82–1433
- men of Japanese ancestry in Hawaii, coronary disease risk relationship, 82–1363
- middle-aged men in Great Britain, employed vs. unemployed, 82–1351
- nurses, 82–1450
- opioid and tobacco dependence, functional similarities, 82–1431
- patients with subarachnoid hemorrhage, 82–1412
- personal beliefs vs. social norms as determinants, 82–1424
- personal space relationship, 82–1438
- pharmacists in Canada, 82–1456
- residential history relationship, 82–1441
- social development depicted in 19th century novels, 82–1527
- social influences and prevention strategies, 82–1478
- students in Australia, health education project effect, 82–1469
- talc workers in upstate New York, lung cancer incidence relationship, 82–1257
- tobacco amblyopia patients, 82–1399

SMOKING SURVEYS
- Australia, New Zealand, and the United States, adolescents, 82–1444
- Australia, smoking patterns in 1980 survey, 82–1432
- Australian schoolchildren, brand preference and advertising recall, 82–1420
- Austria, impact of antismoking campaign, 82–1427
- Canada, drug, alcohol, and tobacco use, 82–1417
- Canada, incidence and trends 1965 to 1979, 82–1458
- Canada, pharmacists, 82–1456
- college students, drug and alcohol use comparison, 82–1457
- East Germany, lung cancer and antismoking measures, 82–1270
- factory workers, 82–1442
- Great Britain, 1972-1980, 82–1445
- head and neck cancer patients, alcohol consumption and diet relationship, 82–1267
- high school seniors, 1975-1981, 82–1435
- nurses, 82–1450
- Scotland adults, 82–1415
- Singapore, prevalence and blood pressure effects, 82–1359

South Australians, smoking behavior and opinions, 82–1430
Soviet adults, 82–1352
Sweden, elderly men, 82–1256
Swiss male draftees, 82–1453
women psychotropic drug users, smoking correlation, 82–1447

SNUFF
pulmonary function effects, 82–1332

SNUFF-DIPPING
oral cancer mortality relationship, 82–1296

SOCIOECONOMIC STATUS
adult smoking habits in Scotland, relationship, 82–1415
blue collar vs. white collar factory workers, smoking rate relationship, 82–1442
bronchitis incidence in infants and children, effect with maternal smoking and breastfeeding, 82–1339
changing cigarette consumption patterns, Great Britain 1972-1980, 82–1445
South Australians, smoking prevalence higher among unemployed, 82–1430
Soviet adults, smoking rates vs. social class, 82–1352
19th century England, smoking as class behavior in novels, 82–1527

SOUTH AFRICA
discounted life insurance premiums for nonsmokers, proposal, 82–1531
fetal lung maturation rate, comparisons with Ethiopia and the United States, 82–1389

SPERMATOZOA
fertility, review of smoking effects, 82–1402

STATISTICAL ANALYSIS
alcohol, drug, tobacco use in Canada, 82–1417
cancer or heart disease mortality in parents, relationship to risk factors in progeny, 82–1282
computer simulation model of smoking for policy development, 82–1448
environmental chemicals vs. occupational exposure vs. smoking in estimating cancer cause, 82–1263
Federal data bases and resources for health educators, 82–1459
lung cancer incidence in sugarcane farming, smoking relationship, 82–1295
lung function of normal subjects and smokers with and without minimal respiratory disease, 82–1312
occupational cancer in Japan, smoking relationship, 82–1277
premature mortality from cigarette smoking vs. road crashes in Australia, 82–1251
smoke components in indoor air pollution, data and review of testing methods, 82–1220
smoking habits, Canada 1965 to 1979, 82–1458
U.S. cancer mortality trends, 1950-1977, 82–1273

STRESS
nurses' smoking behavior relationship, 82–1450

STUDENTS
age-related efficacy of school antismoking campaigns, 82–1466
all grades, health education effect on smoking habit in Australia, 82–1469
college, prescription, over-the-counter, and social drug use, 82–1457
"feeder" approach for primary prevention of drug abuse, 82–1471
first grade, personality factors as predictors of adolescent substance abuse, 82–1437
health behavior change program model, 82–1476
health education and behavior change strategies, 82–1472
high school, cigarette and drug use among seniors, 1975-1981, 82–1435
high school, perceptions related to smoking habit, 82–1440
high school, smoking beliefs and attitudes, 82–1444
medical and law, health promoting and smoking behavior, 82–1423
ninth and tenth graders, self-images and smoking status/intentions relationship, 82–1421
seventh and eighth graders in Australia, brand preference and advertising recall, 82–1420
seventh grade, pilot smoking prevention program, 82–1467
sixth grade, smoking prevention strategies with carbon monoxide feedback, 82–1429

SWEDEN
elderly men, smoking habits and health effects, 82–1256
lung cancer mortality, occupational arsenic exposure and smoking interaction, 82–1288
recognition of smoking-related health hazards by middle-aged men, 82–1433

SWITZERLAND
male draftees, smoking survey, 82–1453
smoking control program, 82–1460

TAR CONTENT
wrapper perforating apparatus to influence smoke condensate content, 82–1498

TAR REDUCTION
filter-diluted smoke, production method, 82–1501
intake monitoring by filter color comparison, 82–1483
modified cast sheeting treatments, 82–1511
tobacco expansion process to lower tar content, 82–1510

variable-density smoking article, 82–1506

TARS, CIGARETTE
airborne particulate matter extracts, lung metabolism in the rat, 82–1223
airways irritation, 82–1333
carcinogen content, 82–1293
cytotoxicity in human polymorphs, 82–1319
high vs. low content, respiratory symptoms relationship, 82–1334
less hazardous cigarettes, critical analysis of disease risk report, 82–1474
lymphocyte chromosomal aberrations, in vitro effect, 82–1248
mutagenic activity, sister-chromatid exchange, 82–1243
skin neoplasm induction in rats, after irradiation, 82–1284
tumor promotion, cell culture assay, 82–1260
wrapper perforation method and apparatus for dilution, 82–1504

TARS, TOBACCO
sizing with a single particle aerodynamic relaxation time analyzer, 82–1218

TAXATION
Federal excise tax on cigarettes, increase effect on teenage smoking, 82–1522
Great Britain, cigarette price increase effect on consumption, 82–1513
role in public policy on smoking control, 82–1464
social welfare costs of smoking, proposed funding, 82–1520
specific vs. ad valorem, effect on cigarette prices, 82–1518
tar—nicotine levels, based on health risk assessment, 82–1514
worldwide smoking control legislation, analysis, 82–1524

TENNESSEE
ambient nitrogen dioxide and smoking relationship to respiratory illness in Chattanooga, 82–1325

TERATOGENESIS
synergistic effect of nicotine with barbiturates, caffeine, and aspirin on chick embryo, 82–1382

TESTES
fertility, review of smoking effects, 82–1402
morbidity in vasectomized vs. nonvasectomized men, smoking relationship, 82–1405

THIOCYANATES
index of smoke exposure, with carboxyhemoglobin levels vs. self-report, 82–1247

TOBACCO ADDITIVES
acyclic carboxamides as menthol substitutes, 82–1493
alpha, beta unsaturated ketones for flavor and aroma, 82–1497
bluish dogbane in Chinese cigarettes, medicinal herb for bronchitis, 82–1503
caryophyllene alcohol mixtures as flavor enhancers, 82–1490
cyclohexene and cyclohexadiene methanols as flavorants, 82–1496
cyclohexene methanols as tobacco flavorants, 82–1494
flavoring compositions for chewing tobacco, 82–1489
6-hydroxy-2, 6-dimethylheptanal and intermediates, as tobacco flavorants, 82–1495
hydroxyethyl-substituted pyrazine and pyridine compounds as flavor/aroma enhancers, 82–1488
1-N-butoxy-1-ethanol acetate as flavorant, 82–1486
potential health hazards of low-yield cigarettes, 82–1492
spirane derivatives for flavoring, 82–1491

TOBACCO AROMA
alpha, beta unsaturated ketones, 82–1497
cyclohexene and cyclohexadiene methanols as flavorants, 82–1496
6-hydroxy-2, 6-dimethylheptanal and intermediates as additives, 82–1495
1-N-butoxy-1-ethanol acetate as flavorant, 82–1486

TOBACCO EXTRACTS
human fibroblast culture, smoke effects, 82–1315

TOBACCO FLAVOR
alpha, beta unsaturated ketones, 82–1497
caryophyllene alcohol mixtures as enhancers, 82–1490
compositions for chewing tobacco, 82–1489
cyclohexene and cyclohexadiene methanols as flavorants, 82–1496
cyclohexene methanols as additives, 82–1494
disc for smoke expansion and flavor improvement, 82–1487
6-hydroxy-2, 6-dimethylheptanal and intermediates as additives, 82–1495
hydroxyethyl-substituted pyrazine and pyridine compounds as enhancers, 82–1488
1-N-butoxy-1-ethanol acetate as flavorant, 82–1486
spirane derivatives, 82–1491

TOBACCO INDUSTRY
Australia, recommended control measures, 82–1530
Canada, economic outlook for 1981 and beyond, 82–1519
Cuba, production and exports, 82–1517
Great Britain, price rise and sales slump effects, 82–1513
legal liability for health effect of smoking, 82–1520

Pakistan, economic slowdown in 1981, prospects for 1982, 82–1512
social component influencing cigarette consumption, 82–1478
Third World seen as area of growth for tobacco consumption, 82–1515
United States, advertising via sponsorship of sport and artistic events, 82–1516

TOBACCO LEAF COMPONENTS
modified cast sheeting procedure, effects, 82–1511
protein content of bright and Burley varieties, 82–1216, 82–1219

TOBACCO MANUFACTURING
source of manganese in cigarette smoke and ash, 82–1214

TOBACCO PROCESSING
cast sheeting treatments, effect on smoke components, 82–1511
expansion process using carbon dioxide, 82–1502, 82–1510

TOBACCO PYROLYSIS PRODUCTS
filter retention efficiency, testing method and tobacco composition effects, 82–1215
levels in indoor air, review of testing methods, 82–1220

TOBACCO SMOKE
aeration mechanism, 82–1498
benzo(*a*)pyrene measurement technique, 82–1213
human fibroblast culture, extracts effects, 82–1315
sonic filter with modulated selectivity, 82–1499

TOBACCO SUBSTITUTES
cellulosic smoking material, 82–1505
Cytrel, effect on rat lungs, 82–1310
reduction of toxic smoke components, 82–1511

TOBACCO VARIETIES
bright vs. Burley, protein content, 82–1216
protein content of chlorophyll-deficient bright and Burley vs. normal, dark fire-cured, and Maryland, 82–1219

TRACHEAL NEOPLASMS
nitrosamine carcinogenesis in hamsters, 82–1217

TRIGLYCERIDES
levels in progeny of parents who died of cancer or heart disease, smoking factor, 82–1282

TUBERCULOSIS
lung cancer risk factor in smoking vs. nonsmoking women, 82–1276

UNION OF SOVIET SOCIALIST REPUBLICS
coronary disease, mortality and risk factor trends, 82–1352
ischemic heart disease, review of risk factors, 82–1362
lung cancer prevalence, smoking and air pollution as risk factors, 82–1287

UNITED KINGDOM
blood platelet function in farmers, diet, alcohol and smoking effects, 82–1364
byssinosis in the flax industry of Northern Ireland, smoking effect, 82–1313
cigarette price increase effect on consumption, 82–1513
coal miners, dust and smoking effects on lung function, 82–1326
cotinine in amniotic fluid of pregnant women in Southampton, 82–1390
discounted life insurance premiums for nonsmokers, 82–1531
health of unemployed vs. employed middle-aged men, 82–1351
pulmonary asbestos and dust content in an East Anglian population, 82–1338
pulmonary function impairment in welders in Sunderland, 82–1303
smoking survey, 82–1445
social development of smoking as depicted in 19th century novels, 82–1527
total ban on tobacco ads recommended, 82–1529

UNITED STATES
adolescents, smoking attitudes, 82–1444
cancer mortality trends, 1950-1977, 82–1273
cancer onset rates, trends, 82–1289
cigarette sales increases in 1981 and novel advertising approaches, 82–1516
fetal lung maturation rate, comparisons with South Africa and Ethiopia, 82–1389
lung cancer, occupational vs. smoking effect on incidence, 82–1298
lung cancer occurrence patterns, smoking relationship, 82–1259

URANIUM
lung cancer, synergistic effect with smoking, 82–1293

URINE
vitamin C excretion, smoking effect, 82–1411

UROGENITAL NEOPLASMS
analgesics and tobacco risk factors in renal pelvic cancer, 82–1283

UROGENITAL SYSTEM
fertility, review of smoking effects, 82–1402
morbidity in vasectomized vs. nonvasectomized men, smoking relationship, 82–1405

VASCULAR DISEASES
- Raynaud's phenomenon among forestry workers in Quebec, smoking effects, 82–1373
- subarachnoid hemorrhage from cerebral aneurysm, smoking risk, 82–1412

VIRUSES
- Herpes simplex virus type 1 and smoking in etiology of oral cancer, 82–1297

VITAMIN A
- carcinogenesis, interactive effect with occupation, 82–1285
- gastric cancer in Japan, relationship with smoking, 82–1278

VITAMIN B
- tobacco amblyopia, treatment, 82–1399

VITAMIN C
- excretion in urine, smoking effect, 82–1411
- L-ascorbic acid and dehydroascorbic acid as protectants against cyanide in rats, 82–1337
- turnover in smokers vs. nonsmokers, 82–1396

VITAMINS
- deficiencies, possible association with amblyopia, 82–1393

WITHDRAWAL SYMPTOMS
- acupuncture treatment, smoking cessation, 82–1481, 82–1485
- tyrosine treatment of catecholaminergic tone, 82–1234

WOMEN
- acute respiratory tract illnesses, occurrence in smokers vs. nonsmokers, 82–1304
- high psychotropic drug use, smoking relationship, 82–1447
- lung cancer in Chinese nonsmokers, incidence, 82–1272
- oral cancer mortality from snuff dipping, 82–1296
- predicting smoking behavior, theory of reasoned action, 82–1424
- respiratory symptoms and smoking habit relationship, 82–1334
- tubercular infection in lung cancer etiology in smokers vs. nonsmokers, 82–1276

SMOKING AND HEALTH LITERATURE SEARCH REQUEST

Individual requesting search	**Title or position**
Individual other than requester completing form	**Telephone number**
Organization	**Date of request**
Address including zip code	**Purpose of search** ☐ Research ☐ Paper presentation ☐ Writing article ☐ Lecture ☐ Other ______

SEARCH REQUIREMENTS Give below a detailed statement of information requirements together with any necessary background data to meet your needs. Be as specific as possible to help formulate search strategy.

If topic has been reviewed or cited in past issues of the *Smoking and Health Bulletin* or *Bibliography on Smoking and Health* give pertinent accession number(s) or citation of articles.

Give below other articles which may serve as a guide on subject area (list full bibliographic citation if known)

List relevant key words or index terms appropriate to search.

If a search has been performed on subject utilizing other data sources, please list sources, results of search and search strategy if known.

SEARCH RESTRICTIONS Check appropriate search out-put desired.

☐ Full range (no restrictions)

☐ Year(s) ______________ only

☐ Citation only (no abstract)

☐ English language only

☐ Other language ______________

AGENCY USE ONLY. Do Not Write Below This Line.

Search statement or criteria

Date search received

Date search released

Number of documents retrieved

☆U.S. GOVERNMENT PRINTING OFFICE: 1982-381-166:448

NOTICE

If you no longer need this publication, check here ____, return this sheet to the address below, and your name will be dropped from the mailing list.

If you have a change in name or address, please write the change on this sheet and return it to the address below.

____

____

____

WHEN REQUESTING A CHANGE PLEASE BE SURE YOUR FORMER LISTING ALSO APPEARS ON THIS SHEET.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health
Rockville, Maryland 20857

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS
BOOK

982/6

NEWARK PUBLIC LIBRARY

JAN 6

U. S. GOVT. PUBS.
DEPOSITORY 426

NOVEMBER-DECEMBER 1982
Abstracts 82-1534-82-1857

Smoking and Health Bulletin

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office on Smoking and Health

HE 20. 7211:

INTRODUCTION

All items appearing in the Smoking and Health Bulletin become part of the permanent document collection of the Technical Information Center (TIC) of the Office on Smoking and Health and can be referred to by their accession number.

In the past, at the end of each calender year, it has been customary to publish the Bibliography on Smoking and Health which contained a cumulative author and subject index based on the individual Bulletins issued that year. However, this year, due to fiscal and other constraints, it may not be possible to issue the index in the form of the Bibliography. All readers are therefore advised to keep the individual Bulletin issues for both 1981 and 1982 until further notice.

The TIC offers bibliographic search and retrieval services for information on smoking, tobacco, and tobacco use through its Automated Search and Retrieval System. Readers are encouraged to submit searches on the search request form found at the end of each Bulletin issue. Multiple copies of this form can be ordered from the TIC. Currently the automated file contains approximately 25,000 records, the majority of which were published and processed into the TIC collection since 1970. The TIC collection totals more than 35,000 items.

Bibliographic and reference services continue to be offered. Readers can take advantage of these services by writing or calling the TIC at the number listed below. Staff limitations currently exists for photocopying services, therefore your cooperation is sought when requesting such services. Until further notice no more than three items per Bulletin per request can be provided. Requests for services above this three document limit cannot be honored. In order that we may continue to provide photocopying services to the largest number of readers, we ask before requesting such items from the TIC collection, to first check your local resources regarding their availability. Visitors may use the TIC library during regular office hours, 8:30 a.m. to 5:00 p.m. weekdays. Advance arrrangements of any planned visits are suggested.

We welcome suggestions or comments from our readership regarding our publications and services.

Donald R. Shopland
Technical Information Officer

Technical Information Center
Office on Smoking and Health
Park Building, Room 116
5600 Fishers Lane
Rockville, Maryland 20857
Telephone: 301-443-1690

ISSN: 0081-0363
NOVEMBER-DECEMBER 1982
Abstracts 82-1534-82-1857

Smoking and Health Bulletin

CONTENTS

AGRICULTURE 352
CHEMISTRY 352
PHARMACOLOGY AND TOXICOLOGY 357
MORTALITY AND MORBIDITY 366
NEOPLASTIC DISEASES 367
NON-NEOPLASTIC RESPIRATORY DISEASES 379
CARDIOVASCULAR DISEASES 386
PREGNANCY AND INFANT HEALTH 395
OTHER DISEASES AND CONDITIONS 398
BEHAVIORAL AND PSYCHOLOGICAL ASPECTS 403
SMOKING PREVENTION AND INTERVENTION 410
SMOKING CESSATION METHODS 415
TOBACCO PRODUCT ADDITIVES 416
TOBACCO MANUFACTURING AND PROCESSING 419
TOBACCO ECONOMICS 421
LEGISLATION 424
GENERAL 425
AUTHOR AND ORGANIZATIONAL INDEX i
SUBJECT INDEX vii

AGRICULTURE

82–1534. Campbell, J. S.; Chaplin, J. F.; Boyette, D. M.; Campbell, C. R.; Crawford, C. B. **Effect of Plant Spacings, Topping Heights, Nitrogen Rates and Varieties of Tobacco on Nicotine Yield and Concentration.** *Tobacco International* 184(11): 72-75, May 28, 1982.

Experiments were conducted in 1974, 1975, and 1976 to determine the effect of cultural practices and varieties on nicotine concentration in tobacco leaves and the yield of nicotine per ha. The tests were modified each year after appraisal of results from the previous year. Varieties and classes tested were: Ky 16 (Burley), Ky 160 and Ky 170 (dark fired), and SC 58 and Speight G-23 (flue-cured). They were grown with varying nitrogen rates, topping heights, and within-row spacings. The nitrogen rates ranged from 112 to 224 kg/ha, topping heights from 12 to 18 leaves, and within-row spacings from 30.5 to 61.0 cm. Ky 160, Ky 170, and SC 58 produced higher nicotine levels in leaves and yield of nicotine per ha than other varieties tested. It would appear these varieties have similar genetic potentials for nicotine production. Nitrogen rates above 112 kg/ha did not significantly increase nicotine concentration of leaves or nicotine yield per ha. Plant spacings of 45.7 and 61.0 cm in the row resulted in higher nicotine levels than spacing plants 30.5 cm apart. Lower plant toppings, 12 and 15 leaves, resulted in a higher nicotine percentage, but topping the plants to 15 and 18 leaves resulted in higher nicotine production per ha. The increase in nicotine per ha was due to higher yields of dry matter in the higher topped plants. The overall results indicate that varieties had more effect on leaf concentration and production of nicotine than any of the other variables studied. However, nitrogen rates, topping heights, and within-row spacings were important. (Auth. Abs.)

82–1535. Link, L. A.; Terrill, T. R. **The Influence of Nitrogen and Potassium Fertilization on the Yield, Quality, and Chemical Composition of Burley Tobacco.** *Tobacco International* 184(14): 26-29, July 9, 1982.

Increased nitrogen (N) fertilization of Burley tobacco raised the nicotine levels of cured leaves during a series of field experiments. The experiments were conducted at four locations in 1973, 1974, and 1975 to study the influence of N and potassium (K) fertilization and the N-K balance on yield, quality, and chemical composition of Burley tobacco. Highly significant year, location, and year by location interactions were noted and were directly interpretable. The absence of treatment by year or treatment by location interactions permitted intensive examination of treatment effects. The reduced analysis, including N rates of 110, 170, and 225 kilograms per hectare (kg/ha) at the same level of K (280 kg/ha) revealed that significant differences in measured parameters were associated with changes in nitrogen fertilization. Highest yields were obtained for the 225 kg/ha rates of N, but they were not significantly different from those obtained with 170 kg N/ha. The high N rate generally caused problems with curing, especially under low humidity conditions. The rate of 110 kg/ha of N was too low for maximum yield and quality. There were no significant differences for yield or quality among the K rates applied. Similarly, no significant differences were associated with altered N-K ratio, although the K concentration of cured leaves was increased by higher rates of K. Higher rates of N increased the nicotine and nitrogen concentrations of cured leaves, but did not affect the other chemical constituents measured. The concentrations of phosphorus, calcium, and magnesium in cured leaves were not affected by the rate of N or K fertilization. (Auth. Abs. Mod.)

CHEMISTRY

82–1536. Andersen, R. A.; Kasperbauer, M. J.; Burton, H. R.; Hamilton, J. L.; Yoder, E. E. **Changes in Chemical Composition of Homogenized Leaf-Cured and Air-Cured Burley Tobacco Stored in Controlled Environments.** *Journal of Agricultural and Food Chemistry* 30(4):663-668, July-August 1982.

Effects of controlled-environment storage (aging) on chemical components in tobacco (*Nicotiana tabacum*) were determined, with emphasis on the effects on probable precursors of health-related components in leaf that may form undesirable or toxic components in smoke. Homogenized leaf-cured (HLC) Ky 14 Burley tobacco was prepared with and without removal of protein. Conventionally air-cured and HLC tobaccos were stored at 8, 12, and 16 percent moisture and at 20 and 30°C. Tobaccos kept in the six environments for 0, 20, 40, and 52 weeks were analyzed. After 52 weeks, concentrations of HLC and low-protein HLC tobacco components were greatly reduced (17-83 percent) in the case of total alkaloids, nitrate N, nitrite N, ammonia N, total volatile nitrogenous base N, amino acids, and petroleum ether extractables; only nitrate N decreased significantly (22 percent) in air-cured tobacco. In contrast, the carcinogenic tobacco-specific compound *N*-nitrosonornicotine (NNN) increased in all tobacco samples during storage. After 52 weeks, NNN concentrations were 200-300 times higher in HLC than in air-cured tobacco. Prior to the tobacco being aged, NNN was also much higher in HLC tobacco. Net changes in component concentrations were increased by storage at higher temperatures and moistures. (Auth. Abs. Mod.)

82–1537. Arrendale, R. F.; Severson, R. F.; Chortyk, O. T.; Snook, M. E. **Analyses of Mono- and Dihydroxybenzenes in Tobacco Smoke and Pyrolyzates by Glass Capillary Gas Chromatography.** *Journal of Chromatographic Science* 20: 136-143, March 1982.

Gas chromatography (GC) with wall-coated open tubular (WCOT) glass capillary columns has become the most widely used method of analysis for mono- and dihydroxybenzenes or phenols. Results are presented of investigations in which glass capillary column GC was used for the separation of mono- and dihydroxybenzenes as underivatized compounds and as trimethylsilyl derivatives of phenols occurring in cigarette smoke condensates, tobacco pyrolyzates, or their fractions. Methodology for the routine quantitative analysis of the major components from these samples is presented. Special emphasis was placed on the capillary inlet system and on liquid phase film thicknesses of capillary columns used in this work. The separations of cigarette smoke, pyrolyzate, and a purified phenolic fraction on SP-1000, SE-54, and SP-2250 WCOT columns are given. Considerations pertaining to routine analyses of a multitude of complex smoke samples are discussed. It is concluded that this is an excellent analytical technique for the quantitation of cigarette smoke condensates or tobacco pyrolyzates, but for complex mixtures, such as tobacco smoke and pyrolyzates, it was, at best, only a semiquantitative technique. (Auth. Abs. Mod.)

82–1538. Canada, Department of Health and Welfare. **Carbon Monoxide Yields of Cigarettes.** Canada, Department of Health and Welfare News Release 1982-11, January 26, 1982, 4 pp.

Results of a smoke analysis of 47 varieties of cigarettes sold in Canada in June 1981 are presented. The brands tested account for more than 85 percent of the cigarettes purchased in Canada. Average carbon monoxide yield in milligrams/cigarette and average tar yield/cigarette are listed for each brand. Average carbon monoxide yield is also presented for 41 brands analyzed in 1980. Of the 34 varieties examined in both years, 17 showed a decrease in the level of carbon monoxide, 8 showed an increase, and 9 remained the same. Canadian manufacturers have agreed to lower the carbon monoxide yield of their cigarettes over the next few years. It is noted that the numbers printed on the cigarette packages referring to tar and nicotine "content" reflect the tar and nicotine intake for the average smoker and will vary with smoking patterns.

82–1539. Chopra, N. M.; Verma, M. M.; Zuniga, T. H. **On the Fate of Maleic Hydrazide in Tobacco Smokes.** *Journal of Agricultural and Food Chemistry* 30(4): 672-676, July-August 1982.

Transfer rates of maleic hydrazide (MH) from tobacco to mainstream and sidestream smoke has fallen from above 57 percent in 1968 to 3.94-1.69 percent in 1980 cigarettes. MH is used as an effective substance for tobacco sucker control, but is also a suspected carcinogen. The German government has set a tolerance limit of 80 parts per million in cigarette tobacco. However, the crucial consideration to the smoker is how much of the MH and its degradation products are present in smoke. Three forms of MH were tested in 1R1, 1968, and 1980 cigarettes: acidic MH, MH diethanolamine salt (DEA-MH), and Royal MH-30 (K-MH). Transfer rates varied according to the form of MH used: the observed drop from the 1969 cigarettes is a result of a shift from DEA-MH to K-MH use during this period. Analysis revealed the following rates: (1) for acidic MH, 2.46 to 2.83 percent in the mainstream and 0.29 percent to undetected in the sidestream smoke; (2) for K-MH, 1.25-1.69 percent (mainstream) and 0.3-0.97 percent (sidestream); and (3) for DEA-MH, 5.54 percent (mainstream) and 3.33 percent (sidestream).

82–1540. Hwang, K. K.; Bhooshan, B.; Kouri, R. E.; Henry, C. J. **Synthesis of Tritium Labelled Catechol.** *Journal of Labelled Compounds and Radiopharmaceuticals* 19(1): 35-38, January 1982.

The preparation of tritium-labeled catechol by catalytic reduction of tetrabromocatechol is described. Recrystallization of catechol (U-^{3}H) after silica gel column purification gave 68 percent yield of the desired product with 98 percent purity as assayed by high performance liquid chromatography. The procedure may be important in health research, since catechol occurs in tobacco leaf and is also the most abundant phenol in cigarette smoke condensate. Catechol is known to have biological activity. (Auth. Abs. Mod.)

82–1541. Johnson, D. E.; Prevost, R. J.; Tillery, J. B.; Thomas, R. E. **The Distribution of Cadmium and Other Metals in Human Tissue.** Southwest Research Institute, San Antonio, Texas, Publication No. EPA-600/1-78-035, September 1977, 245 pp.

The relationship between cadmium, lead, zinc, and mercury burdens and age in five different tissues obtained from approximately 30 autopsy cases of sudden or accidental death victims from the same metropolitan area (Dallas, Texas) in each of five age groups were studied. Histories were collected on all cases to ascertain that a "nonexposed" population, with regards to cadmium, was sampled. Information on cigarette smoking, diseases, etc. was also collected and from the data obtained, separate means were determined across age groups for smokers and nonsmokers and a t-test was used to determine significant differences. A concurrent epidemiological study was conducted in the Dallas metropolitan area to investigate the cadmium concentrations in urine and feces of persons (six age groups) not exposed to cadmium. Cadmium questionnaires were also used and data concerning lifetime smoking incidence, current smoking status, amount of cigarettes smoked per day, and age at initiation were collected. Atomic absorption spectrophotometry (AAS) was used for all metal measurements. The analytical methodology used for cadmium in eight different types of biological samples was evaluated in a round robin study involving laboratories in Japan and Sweden. Tissue preparation was with "low temperature ashing" techniques and both flame and flameless AAS were used. Analytical methodologies for cadmium analysis were investigated. Statistical evaluation of the data was performed. Interrelationships between all autopsy tissues for each metal were examined. Total body burdens of cadmium were calculated for "standard man." (Auth. Abs. Mod.)

82-1542. Kallianos, A. G. **The Organic Chemistry of Tobacco and Tobacco Smoke.** *Tabak Journal International* 3:281-282, June 1982.

As a natural plant material, tobacco contains a complex mixture of chemical constituents. These include alkaloids, sugars, proteins and amino acids, cellulosic products, phenolics, hydrocarbons, carboxylic acids, sterols, pectic substances, isoprenoids, and several minerals. The specific composition of the leaf is influenced by genetics, agricultural practices, soil type and nutrients, climatic conditions, health of the plant, and stalk position, and varies during the growing, curing, and aging processes. Cigarette smoke represents a very dynamic system, comprised of thousands of compounds produced by pyrolysis and those transferring from tobacco unaltered. The chemistry of smoke reflects the chemistry of tobacco, which is further influenced by the cigarette manufacturing process. The major components that transfer unaltered include nicotine, saturated hydrocarbons, low molecular weight acids and esters, and carbonyl flavoring compounds. The smoking process itself adds pyridines and other nitrogenous bases, lactones, phenols, catechols, and polycyclic aromatic hydrocarbons to the smoke delivered by the burning cigarette. (Auth. Abs. Mod.)

82-1543. Kousaka, Y.; Okuyama, K.; Wang, C.-S. **Response of Cigarette Smoke Particles to Change in Humidity.** *Journal of Chemical Engineering of Japan* 15(1): 75-76, 1982.

Condensation of water vapor on cigarette smoke particles occurs only under supersaturation conditions. This was demonstrated by an experiment which measured size distributions of such particles generated with filters. The experimental setup consisted mainly of an ultramicroscopic sedimentation size analyzer, a smoking device, two humidifiers, and a psychrometer. Each humidifier consisted of a silica gel bed, a bubbler, and two control valves. Relative humidity of the airstream from each humidifier was controlled by adjusting the flow rates of the airstreams through the bubbler and the silica gel bed. Supersaturation conditions can be obtained by heating the water in the bubblers, and particles in cigarette smoke can grow by either Brownian coagulation or condensation. To minimize coagulation effect, a one hundredfold dilution by filtered, humid air was performed with the diluted smoke containing approximately 10^6 particles/cm^3. Smoke particles were measured at various relative humidities at 25°C and at one supersaturation condition. Residence time of particles in the system ranged from 10 to 30 secs. Although marked increase in particle size took place with supersaturation, no appreciable growth was seen at 100 percent or lower relative humidities. Size distribution of particles exhaled from a human subject were measured following smoke dilution of one hundredfold before inhalation. Size distribution differed only slightly from that of the prior experiment. It is concluded that the change in size of cigarette smoke particles in the respiratory system is caused mainly by Brownian coagulation and, possibly, by condensation of water vapor under supersaturated conditions, which may be induced by a drop in temperature of inhaled smoke.

82-1544. Masuo, Y.; Suga, Y.; Shibata, M.; Terahara, A.; Hatakeyama, M.; Takeda, K.; Yasumatsu, N.; Yamazaki, M.; Akaki, T.; Watanabe, R.; Tanaka, H.; Maeda, K. **Fuiruta Tabako no Hinshitsu ni Oyobosu Makigami to Chippu no Kaiko Koka (dai Jippo). Bento Tabako Kosei Yoin no Enseibun Oyobi Kokitsumi ni Oyobosu Eikyo. [Effect of the Degree of Perforation of Cigarette Paper and Tipping Paper on the Performance of Filter Cigarette (X). Effect of the Compositional Variables of Vent Cigarette on the Delivery of Smoke Components and Sensory Evaluation.]** *Scientific Papers of the Central Research Institute Japan Tobacco and Salt Public Corporation,* 123: 79-89, 1981, Japanese.

The influence of blend type, perforation method of filter, tow item, adhesion method of tipping paper, and air permeability of cigarette paper in decreasing mainstream smoke components and on sensory aspects was investigated. The delivery of smoke components in a cigarette was affected by blend type; however, the average yield of carbon oxides per puff and the reduction rate of smoke components vs. filter ventilation were not affected. With the same ventilation rate or the same delivery of nicotine, light American blend was the best in terms of ease in puffing and Virginia blend was the worst. Filters or cigarette paper allowing more ventilation per puff tended to weaken original tobacco taste and decrease ease in puffing. It was found that laser beam perforations allowed more ventilation than mechanical perforations. Also, filters having an open rather than closed lap of wrapping at the dry zone, and high instead of low permeability in cigarette paper, allowed greater ventilation. The 2.2Y/40000 filter, having a higher pressure drop and filtration efficiency for nicotine and dry condensate than the 4Y/43000 filter, resulted in less smoke and poorer taste. (Auth. Abs. Mod.)

82-1545. Masuo, Y.; Yasumatsu, N.; Shibata, M.; Tomita, H. **Fuiruta Tabako no Hinshitsu ni Oyobosu Makigami to Chippu no Kaiko Koka (dai Juippo). Nensho To Kusei to Enseibun no Kankei. [Effect of the Degree of Perforation of Cigarette Paper and Tipping Paper on the Performance of Filter Cigarettes (XI). On the Relationship Between Burning Characteristics and Delivery of Smoke Component.]** *Scientific Papers* *of the Central Research Institute Japan Tobacco and Salt Public Corporation* 123: 91-100, 1981, Japanese.

More reliable data on the amount of a cigarette consumed during a puff and between puffs became available using a newly designed apparatus. Burning characteristics, the volume ratio of influx air at the burning cone, and the delivery of smoke components were investigated in cigarettes varying in ventilation rate and blend type. The volume ratio of influx air at the burning cone was inversely proportional to the ventilation rate of cigarette paper and filter measured for an unlit cigarette. The burnt weight during a puff was dependent on the volume of influx air at the burning cone and was not affected by blend type. The amount of dry condensate generation per cigarette, calculated from the delivery in the mainstream and the retention on a filter, depended on the cumulative weight burnt during puffs. The amount of nicotine generated was further affected by the nicotine content in burnt shred. Carbon dioxide generation was dependent on the sum of the volume of influx air at the burning cone. A model for estimating the generation of dry condensate, nicotine, and carbon dioxide is presented utilizing regression equations obtained from combinations of factors such as cigarette weight, nicotine content in shred, burning characteristics, volume ratio of ventilation, and delivery of smoke components. The estimates agree fairly well with observed values. (Auth. Abs. Mod.)

82–1546. McRae, D. D. **The Refractive Index of Individual Cigarette Smoke Droplets.** *Journal of Colloid and Interface Science* 87(1): 117-123, May 1982.

The refractive indices of individual cigarette smoke droplets were determined from light-scattering measurements. Research-type cigarettes were smoked under standard conditions with the smoke being diluted 6400:1 at a relative humidity of 81 percent. The differential scattering patterns for single smoke droplets were measured using vertically polarized light with a wavelength of 514.5 nm. The scattering patterns were fitted to theoretical curves generated from Mie theory, and the size and refractive index for each droplet were determined. The average refractive indices for coagulated and uncoagulated smoke were 1.51 and 1.52, respectively. The individual refractive indices exhibited a wide range of values, from 1.43 to 1.59, with no apparent dependence on size. It is postulated that the relatively large differences in refractive index from droplet to droplet are the result of (1) compositional differences in the hydrophilic compounds present in each droplet which would lead to variations in the equilibrium amounts of water in the droplets, and (2) the loss of different types and amounts of volatile organics from the droplets. (Auth. Abs. Mod.)

82–1547. Mlynarczyk, W.; Sadowski, C.; Kowalczyk, H.; Laboda, K. **Wielopierscieniowe Weglowodory Aromatyczne (WWA) w Dymie Papierosowym. [Polycyclic Aromatic Hydrocarbons (PAH) in Cigarette Smoke.]** *Bromatologia i Chemia Toksikologiczna* 14(1): 57-61, 1981, Polish.

Ten polycyclic aromatic hydrocarbons (PAH) were identified and their concentration determined in the smoke condensate of five popular Polish cigarette brands. Smoke from Extra Moene cigarettes was found to contain the highest level of 3,4-benzopyrene (considered as an index of product contamination with polycyclic aromatic hydrocarbons), while in that from Giewont cigarettes the lowest one occurred. Moreover, the smoke from Extra Moene was shown to contain much more anthracene, 1,2-benzopyrene, 1,2,5,6-dibenzanthracene, and 1,12-benzoperylene than that from other kinds of products under test (i.e., Sport, Giewont, Klubowe, and Radomskie cigarettes). Extra Moene cigarettes are considered particularly dangerous to health. (Auth. Abs. Mod.)

82–1548. Muramatsu, M. **Kamimaki Tabako no Shizen Nensho ni Okeru Ido Gensho ni Kansuru Kenkyu. [Studies on the Transport Phenomena in Naturally Smoldering Cigarettes.]** *Scientific Papers of the Central Research Institute Japan Tobacco and Salt Public Corporation,* 123: 9-77, 1981, Japanese.

Two major reaction zones exist in the smoldering cigarette: (1) the evaporation-pyrolysis zone (100-450°C), and (2) the high-temperature burning zone (above 450°C). Many factors, such as reaction rate and mass and heat transfer, govern the processes occurring in the two smoldering zones. These factors and smoldering characteristics were experimentally evaluated using the following methods: (1) Effective thermal conductivity in cigarette columns and specific heat of tobacco were evaluated by a transient hot-wire method and by a DSC method, respectively. (2) Thermal analyses (TG, DTG, DSC) of tobacco and its carbonaceous residue were carried out in a helium atmosphere and in a nitrogen atmosphere containing a certain amount of oxygen, respectively. Applying nth order Arrhenius kinetics, apparent kinetic parameters for the pyrolysis reaction of tobacco and the oxidation reaction of the carbonaceous residue were determined. (3) The amounts of oxygen consumed and carbon oxides formed during the natural smolder of cigarette were measured in a closed chamber. The amount of heat evolved was estimated from the heat of formation of carbon oxides and water produced in the burning zone. (4) The behavior of the natural smolder was characterized by smoldering rate, and temperature and density profiles. On the basis of the experimental results and the postulated mechanism of natural smolder, theoretical models for both the evaporation-pyrolysis and the burning processes were developed, in which mass and heat transfer phenomena were expressed by a set of simultaneous differential equations. The smoldering rate, temperature, density, and oxygen profiles predicted from the numerical solutions of the equations approximated well to the experimental results, indicating the validity of the postulated mechanism of the smolder. Based on the model, the influence of many physical parameters on the smoldering rate and temperature were evaluated with respect to controlling the smoldering process of the cigarette. (Auth. Abs. Mod.)

82–1549. Novotny, M.; Merli, F.; Wiesler, D.; Fencl, M.; Saeed, T. **Fractionation and Capillary Gas Chromatographic—Mass Spectrometric Characterization of the Neutral Components in Marijuana and Tobacco Smoke Condensates.** *Journal of Chromatography* 238(1): 141-150, 1982.

Polar and nonpolar "neutral" constituents of the smoke condensates of Mexican marihuana and standard tobacco were resolved by capillary gas chromatography and structurally characterized through mass spectrometry. Comparisons of the two materials reveal substantial qualitative and quantitative differences. The cannabinoid-like substances partition into both the nonpolar and polar fraction. In total, over 130 "neutral" smoke components have been characterized. (Auth. Abs.)

82–1550. Samfield, M. **Tobacco—A Major Food Crop for the World?** *Tabak Journal International* 3: 188-189, 192-193, June 1982.

The advantages of using tobacco as a source of protein include the fact that (1) although the composition of proteins from tobacco is not essentially different from that of other leafy materials, the tobacco extraction process has been found to be much easier and less costly; (2) when grown primarily as a source of protein, tobacco gives a much higher yield per acre; and (3) protein-extracted tobacco tested in cigarettes was found to yield fewer nitrosamines and have less biological activity. The protein extraction processes used by Leaf Protein International, Inc. (LPI) and the U.S. Department of Agriculture (USDA) are described. LPI's process is for the development of products with commercial uses ranging from medicinal to additives for forage crops. The residue from the LPI process and that produced by the USDA's homogenized leaf curing (HDL) process have been found to provide a less hazardous smoking material. The product of the HDL process has a lower nicotine content, a higher starch content, and lower sugar content than normal tobacco.

82–1551. Sloan, C. H.; Curran, J. G. **Spectrophotometric Determination of Filtration Efficiency of Cigarette Filters.** *Tobacco International* 183(11): 26-29, May 29, 1981.

A spectrophotometric method developed for determining efficiency of cigarette filters correlates well with the gravimetric method and is not adversely affected by tobacco types or filter parameters. The method can also be used to determine indirectly the amount of dry cigarette total particulate matter (TPM) delivery. To determine filtration efficiency, one to four filter cigarettes are smoked. The TPM collected on the cigarette filters and Cambridge filters is extracted with methanol, and the absorbances of the solutions are measured at 310 nm. Filtration efficiency is determined by dividing the absorbance of the cigarette filter extract by the combined absorbance of the cigarette filter and Cambridge filter. The amount of dry TPM delivered by cigarettes is directly proportional to the absorbance of the methanol solutions. Thus the dry TPM delivery of a cigarette can be read directly from a previously prepared calibration curve of absorbance vs. milligrams of dry smoke TPM. The results obtained by the method agree well with those obtained by the standard gravimetric method. Relative standard deviation of the method is 1.8 percent. The advantages of the spectrophotometric method over the gravimetric method include: rapid analysis, possible automation, improved precision, and no determination of water in TPM. (Auth. Abs. Mod.)

82–1552. Yamamoto, T. **—Kyuen Nenshoryo to Shuryu Enchu—Sanka Tanso Oyobi Nisanka Tanso no ryo ni Oyobosu Tenju Mitsudo, Makimawari Oyobi Kizami no shurui no Eikyo. [The Effect of Packing Density, Circumference and the Kind of Shreds on the Weight Loss During a Puff and Carbon Oxides Delivery in the Mainstream Smoke.]** *Scientific Papers of the Central Research Institute Japan Tobacco and Salt Public Corporation* 123: 1-8, 1981, Japanese.

By measuring directly the weight loss of minicigarettes (35mm length) during a puff (W_p), the effects of packing density, circumference, and kinds of tobacco shreds on W_p and the delivery of carbon oxides were investigated. Results obtained are summarized as follows: (1) W_p decreased linearly with either an increase in the packing density or a decrease in the circumference of the cigarette. (2) CO delivery per puff was proportional to W_p. (3) In the cigarette made from same kind of tobacco leaf with a constant circumference, CO_2 delivery was substantially unchanged regardless of packing density; however, it changed proportionally to circumference when the packing density was constant. (4) Among four kinds of minicigarettes differing in blend type, with approximately the same packing density, no appreciable difference was found in W_p and carbon oxides delivery. (Auth. Abs. Mod.)

See also, **82–1768**

PHARMACOLOGY AND TOXICOLOGY

82-1553. Aeschbacher, H. U.; Chappuis, C. **Non-Mutagenicity of Urine From Coffee Drinkers Compared With That From Cigarette Smokers.** *Mutation Research* 89(2): 161-177, June 1981.

Urine fractions of heavy coffee drinkers were not mutagenic, whereas those of heavy cigarette smokers showed mutagenic activity, in a study of the urine of human coffee drinkers. The coffee drinkers ingested 12 g of instant coffee per day during 4 days in a first experiment, or 12 g within 2 hrs in a second experiment. The urine was fractionated by XAD-2 column chromatography. The nonpolar urine fractions so obtained were not mutagenic in the Ames Salmonella tester strains TA98 or TA100 in either experiment, either with or without β-glucuronidase treatment of the urine. The nonpolar urine fraction of smokers, who smoked 20-30 cigarettes per day during 4 days in the first experiment or 7-18 cigarettes during 7 hrs in the second experiment, was mutagenic when metabolically activated. A syngergistic effect with coffee was excluded. (Auth. Abs. Mod.)

82-1554. Alster, P.; Wennmalm, A. **Human Platelet Thromboxane Synthesis Is Unaffected by Nicotine.** *Clinical Physiology* 1(1): 115-122, 1981.

The effect of nicotine ($2x10^{-7}$ to $2x10^{-4}$ M) on arachidonate (AA)-induced platelet aggregation and on the bioformation of thromboxane (TxB_2) in human platelets was investigated. Human platelet-rich plasma (HPRP) and human platelet suspension (HPS) were prepared from venous blood from donors who had not taken aspirin-like drugs for at least 1 week. Using HPRP, nicotine was inefficient in blocking platelet aggregation from threshold AA concentrations—in contrast to indomethacin and aspirin—and also in prolonging the interval between the addition of AA and the initiation of aggregation. In other experiments, platelet microsomes prepared from HPS were incubated with ^{14}C-AA in the presence of nicotine, and the labelled metabolites were separated and quantified radiochromatographically. Addition of nicotine to the incubations did not affect the amount of TxB_2 formed compared to controls; indomethacin, on the other hand, efficiently inhibited TxB_2 formation as did pretreatment of the donors with aspirin. The results indicate that human platelet cyclo-oxygenase, unlike, for example, rabbit renal cyclo-oxygenase, is resistant to the inhibitory action of nicotine. The possible relevance of a tissue difference for tobacco smoking is pointed out. (Auth. Abs.)

82-1555. Autrup, H.; Grafstrom, R. C.; Brugh, M.; Lechner, J. F.; Haugen, A.; Trump, B. F.; Harris, C. C. **Comparison of Benzo(*a*)pyrene Metabolism in Bronchus, Esophagus, Colon, and Duodenum From the Same Individual.** *Cancer Research* 42: 934-938, March 1982.

The metabolism of benzo(*a*)pyrene has been investigated in cultured normal human bronchus, colon, duodenum, and esophagus obtained from the same patient. The highest total metabolism was found in bronchus and duodenum, while the highest mean binding level was observed in the bronchus followed, in order, by the esophagus, duodenum, and transverse colon. A 30-fold interindividual variation in the binding level was found in each of the four organs studied, and a positive correlation between the binding levels in bronchus, colon, and duodenum was found. In human bronchus, a positive correlation was found between level of binding of benzo(*a*)pyrene to DNA and the amount of both benzo(*a*)pyrene 7,8-diol and the combined group of 3-hydroxybenzo(*a*)pyrene, benzo(*a*)pyrene 9,10-diol, and water-soluble metabolites. A significantly higher relative amount of benzo(*a*)pyrene tetrols and benzo(*a*)pyrene 9,10-diol was formed by human bronchus compared to the gastrointestinal tissues, while a higher level of benzo(*a*)pyrene phenols was formed by the latter. The relative distribution of benzo(*a*)pyrene-DNA adducts was similar in all four organs, the major DNA adduct being formed by *trans*-addition of anti-7,8-dihydroxy-9,10-epoxide-7,8,9,10-tetrahydrobenzo(*a*)pyrene to the 2-amino group at guanine. These results indicate that the metabolism of benzo(*a*)pyrene by at least four different organs is qualitatively similar but that quantitative differences exist. (Auth. Abs.)

82-1556. Bardell, D. **Viability of Six Species of Normal Oropharyngeal Bacteria After Exposure to Cigarette Smoke in Vitro.** *Microbios* 32(127): 7-13, 1981.

Bacteria in culture medium at 37°C were subjected to eight puffs of smoke from one cigarette, and viable cell counts were done at intervals ranging from 0 min to 3 hr after exposure to smoke. The cigarette contained 23.0 mg of tar and 1.4 mg of nicotine. Each 25.0 ml puff of smoke from a mechanical smoking apparatus was passed over a 1.0 ml suspension of bacteria dispersed over a 25 cm^2 surface in a sterile flask. Filtered air was in contact with the suspension between puffs and during the intervals between smoke treatment and viable cell counts being done. There was a marked decrease in numbers of viable *Branhamella catarrhalis* at 15 min after exposure to smoke, and by 1 hr there were no viable cells in suspensions which originally contained 10^7 bacteria. No reduction in the number of viable *Branhamella catarrhalis* occurred in an untreated suspension of the microorganism. There was a marked decrease in viable *Neisseria perflava* at 30 min, and by 3 hr there were no viable cells. There was a 4 to 5 $\log_{10}$ reduction of viable *Neisseria sicca* compared with untreated bacteria at 3 hr after exposure to smoke. *Streptococcus mitis*, *Streptococcus salivarius* and *Streptococcus sanguis* were less susceptible to the harmful action of cigarette smoke, and by 3 hr there were 1 to 2 log$_{10}$ differences in viable cell counts between smoke-treated and untreated bacteria. Nonfilter cigarette smoke had a greater detrimental effect on all species of bacteria studied than smoke from filter-tipped cigarettes. (Auth. Abs.)

82–1557. Beatty, K.; Robertie, P.; Senior, R. M.; Travis, J. **Determination of Oxidized Alpha-1-Proteinase Inhibitor in Serum.** *Journal of Laboratory and Clinical Medicine* 100(2): 186-192, August 1982.

When the proportion of oxidized alpha-1-proteinase (α-1-PI) in the serum of young adult, healthy smokers and nonsmokers was measured, 23 percent of oxidized inhibitor was found in the smokers' sera, whereas no oxidized α-1-PI was detectable in sera of nonsmokers. A rapid procedure developed for determining the percentage of oxidized α-1-PI in plasma or serum based on the ratio of trypsin inhibitory capacity (TIC) to elastase inhibitory capacity (EIC) of α-1-PI in these tissues was used. The TIC/EIC ratio is not influenced by the other proteins in serum nor by the concentration of α-1-PI. When α-1-PI is oxidized, it loses its ability to inhibit porcine elastase, although it retains its TIC. Therefore, the ratio of TIC to EIC increases in proportion to the degree of oxidation of the inhibitor. Results indicate that smoking leads to oxidation damage to circulating α-1-PI. Reduction of the EIC of the α-1-PI in the serum of smokers was compensated by a 1.43-fold increase in their serum α-1-PI titers; however, these data do not exclude the possibility of local depletion of the absolute EIC in the extravascular space of the lungs. This assay for oxidized α-1-PI may be useful in studies of the relationship between oxidation of α-1-PI and the development of pulmonary emphysema, which appears to be a consequence of accelerated lung elastin degradation that results from either an elevated level of elastase activity or a decreased level of elastase inhibitor(s) within the lung, or a combination of both factors. (Auth. Abs. Mod.)

82–1558. Burney, R. E.; Wu, S.-C.; Nemiroff, M. J. **Mass Carbon Monoxide Poisoning: Clinical Effects and Results of Treatment in 184 Victims.** *Annals of Emergency Medicine* 11(8): 394-399, August 1982.

On the basis of a clinical and epidemiologic study of 184 persons exposed to potentially toxic levels of carbon monoxide (CO), it is concluded that smokers (n=77) should not be assessed by different criteria or treated differently from nonsmokers (n=107) for CO poisoning. Accidental exposure of students and teachers in a public high school to 500 ppm of CO for periods up to 150 minutes resulted in carboxyhemoglobin (COHb) levels as high as 30 percent and symptomatic illness in 87 percent of those exposed. Data obtained from questionnaires completed by all occupants of the school, and physicians and administrators at the hospitals where the victims were treated, were analyzed. The analysis showed that smokers did not differ from nonsmokers in their symptomatic response to CO poisoning, in COHb levels, or in rate of elimination of CO, but did feel back to normal sooner than did nonsmokers. Because the more rapid improvement of smokers cannot be attributed to more rapid elimination of CO, it is speculated that smokers do have more tolerance for low levels of CO than do nonsmokers.

82–1559. Chamson, A.; Frey, J.; Hivert, M. **Effects of Tobacco Smoke Extracts on Collagen Biosynthesis by Fibroblast Cell Cultures.** *Journal of Toxicology and Environmental Health* 9(5/6): 921-932, May-June 1982.

Tobacco smoke extract (35 nmol/ml nicotine) was found to increase collagen production but not protein secretion in fibroblast cell cultures from adult human skin explants. Aqueous tobacco smoke extracts were prepared and the nicotine content of the extract was used to characterize the concentration. Since the biosynthesis of collagen and noncollagen proteins is variable in the same subculture, tobacco smoke effects were determined by studying the biosyntheses at various times after subcultivation. Not only the rate of collagen synthesis but also the type of collagen produced were affected; in particular there was a decrease of type III collagen. It is concluded that these disturbances may contribute in vivo to the sclerosis phenomenon seen in arteriosclerosis and lung fibrosis consisting of a collagen deposit.

82–1560. Cook, C. E.; Brine, D. R.; Quin, G. D.; Perez-Reyes, M.; Di Guiseppi, S. R. **Phencyclidine and Phenylcyclohexene Disposition After Smoking Phencyclidine.** *Clinical Pharmacology and Therapeutics* 31(5): 635-641, May 1982.

Five men who smoked parsley cigarettes containing 100 μg of (^{3}H)-phencyclidine hydrochloride (PCP-HCl) inhaled 69 ± 5(SEM) percent of the total radioactivity in the cigarette. Both PCP and its pyrolysis product, I-phenylcyclohexene (PC), were found and measured in plasma. Calculations based on the assumption that the ratio of these two products was the same as in simulated smoking studies and based on either area under the curve or urinary excretion of PCP indicated that most of the PCP in smoke was absorbed. Mean half-life (t 1/2) of PCP (24 ± 7 hr, harmonic mean 18 hr) and ratios of metabolites in plasma and urine were close to those previously reported after intravenous and oral doses. A second peak in PCP plasma concentrations was observed, possibly due to slow efflux from the lungs. PC plasma concentrations (maximum 0.35 ± 0.06 pmol/ml) were lower than those of PCP (maximum 0.62 ± 0.09 pmol/ml) and its mean t 1/2 (14 ± 3 hr, harmonic mean 12 hr) was shorter than that of PCP. Only traces of PC were found in urine. Only small amounts of metabolites from PC were found nonconjugated in plasma (to about 0.1 pmol/ml) or urine (< 2 percent of radioactivity), but larger quantities were found as enzyme-hydrolyzable conjugates in urine (6 percent of radioactivity). Conjugates were also found in plasma (to about 0.12 pmol/ml). (Auth. Abs.)

82–1561. Davies, B. D.; Hoss, W.; Lin, J.-P.; Lionetti, F. **Evidence for a Noncholinergic Nicotine Receptor on Human**

Phagocytic Leukocytes. *Molecular and Cellular Biochemistry* 44(1): 23-31, April 16, 1982.

A noncholinergic nicotine receptor on human phagocytic leukocytes has been characterized using the binding of ^{3}H-(d,l)-nicotine. The average affinity ± standard deviation of (d,l)-nicotine for the receptor on neutrophils is 36 ± 18 nM ($n = 6$). The binding is saturable with an average of 8.7×10^4 sites per neutrophil. Monocytes and to a lesser extent lymphocytes but not erythrocytes also display specific binding. Bound nicotine is dissociable from the receptor and is not metabolized. Only close structural analogs of nicotine bind to the receptor, which is stereoselective for the (d)-isomer. The receptor can be occupied by (l)-nicotine at concentrations present in the blood of smokers. It is suggested that some of the adverse effects of smoking on leukocyte functions may be mediated by a specific nicotine receptor. (Auth. Abs.)

82-1562. De Marini, D. M. **Mutagenicity of Fractions of Cigarette Smoke Condensate in *Neurospora crassa* and *Salmonella typhimurium*.** *Mutation Research* 88(4): 363-374, April 1981.

The mutagenicities of selected fractions of cigarette smoke condensate (CSC) were studied in *Neurospora crassa* for the presence of direct-acting mutagens. CSCs from the University of Kentucky Reference Cigarette 1R1 were assayed in a forward-mutation test at the *adenine-3* region in resting conidia of a two-component heterokaryon. Direct-acting mutagenic activity was found in an enriched polycyclic aromatic hydrocarbons (EPAH) fraction and in a basic fraction (Swain 5). No direct-acting mutagenic activity was detected in an acidic fraction (Swain 8), although it was highly toxic to resting conidia. The EPAH fraction also was tested in the presence of S9 mix prepared from Aroclor-1254-induced rat liver. It was found to be mutagenic, but higher doses were required than in the absence of S9 mix. In addition, the mutagenicities of CSC and 10 fractions of CSC were investigated in *Salmonella typhimurium* TA1538 by the incorporation and preincubation methods. In general, preincubation did not enhance the mutagenicities of the fractions, and the two rankings of mutagenic potency of the condensates that were obtained by the two methods were not significantly different. (Auth. Abs.)

82-1563. Flicker, C.; Geyer, M. A. **Behavior During Hippocampal Microinfusions. II. Muscarinic Locomotor Activation.** *Brain Research Reviews* 4(1): 105-127, March 1982.

Male rats were examined for their behavioral responses to infusions of nicotine, carbamylcholine chloride (carbachol), atropine sulfate, and D-tubocurarine chloride during 40-minute experimental sessions. A computerized holeboard system measured the animals' locomotor activity, stimulus responsivity, and response to novelty, and permitted the reconstruction and analysis of their sequential patterns of movement. Infusions of the mixed muscarinic/nicotinic cholinergic agonist carbachol produced a dose-dependent increase in locomotor activity that was accompanied by increased holepoking and rearing. The response to novel objects and odors was depressed by carbachol. Carbachol-infused rats also exhibited dose-dependent changes in their patterns of locomotor activity. Hippocampal infusions of the muscarinic antagonist atropine produced nonsignificant decreases in locomotion and a perseverative pattern of locomotor activity and stimulus responding. Co-infusion of atropine blocked the effects of carbachol, whereas the effects of the nicotinic agonist nicotine and antagonist curare were negligible. Although nicotine is approximately 5 times more potent that carbachol as a nicotinic receptor agonist. the nicotine nonsignificantly increased locomotion, rearing, and holepoking. Curare's effects on motor activity were biphasic, interacting with the time-blocks factor. The nicotinic blocker caused a mild depression of locomotion and holepoking during the second 10 minutes of the session. These effects of nicotine and curare tend to suggest that there is a weak nicotinic contribution to the activating effects of carbachol. (Auth. Abs. Mod.)

82-1564. Galanakis, D. K.; Laurent, P.; Janoff, A.; Chung, S. I. **Cigarette Smoke Contains Anticoagulants Against Fibrin Aggregation and Factor XIIIa in Plasma.** *Science* 217(4560): 642-645, August 13, 1982.

Cigarette smoke contains two distinct coagulation inhibitors: one prolongs the clotting times of plasma by inducing delayed fibrin aggregation and requires the COOH-terminal region of fibrin α chains to exert its effect; the other inactivates factor XIIIa (plasma transglutaminase), preventing the cross-linking of fibrin polymers. The effects of smoke on human fibrinogen functions were studied in vitro. The loss of XIIIa activity was proportional to the concentration of the smoke extract, and it could not be reversed by removal of the smoke inhibitor by dialysis or by gel filtration. The second smoke inhibitor may affect other physiologically important proteins also known to be cross-linked to fibrin by XIIIa. These anticoagulant properties may play a role in the pathophysiology of smoking: diminished fibrin cross-linking results in impairment of normal tissue repair. (Auth. Abs. Mod.)

82-1565. Garg, S. K.; Ravi Kiran, T. N. **Effect of Smoking on Phenylbutazone Disposition.** *International Journal of Clinical Pharmacology, Therapy and Toxicology* 20(6): 289-290, June 1982.

A single oral dose of 6 mg phenylbutazone/kg body weight was administered to seven cigarette smokers and to seven healthy male volunteers serving as controls. Plasma phenylbutazone half-life was shortened significantly ($p < 0.05$) in the cigarette smokers as compared to the nonsmoking control group. Phenylbutazone was cleared from the blood significantly faster ($p < 0.06$) in the smoking group than in the nonsmoking group. No significant change was observed in the apparent volume of distribution of the two groups studied. This implies smokers need either larger doses or more frequent administration of drugs to obtain therapeutic responses similar to those in nonsmokers. (Auth. Abs. Mod.)

82–1566. Hitzenberger, G.; Fitscha, P.; Beveridge, T.; Nuesch, E.; Pacha, W. **Influence of Smoking and Age on Pharmacokinetics of β-Receptor Blockers.** *Gerontology* 28(Supp. 1): 93-100, April 1982.

In an examination of the influence of age and smoking on pharmacokinetics of pindolol and propranolol, no significant differences were found between smokers and nonsmokers. Thirty-two healthy male volunteers were divided into two groups of 16 each by age—young (age 20-30 years) and old (over 60 years). Each group was subdivided into eight smokers (>10 cigarettes/day) and eight nonsmokers (<10 cigarettes/day or had not smoked for 5 years or more). There was a statistically significant difference in certain pharmacokinetic parameters (maximum plasma concentration, area under the curve, elimination constant and elimination half-life) between elderly patients and young persons. In contrast with some reports in the literature, no significant differences were found as far as smoking and nonsmoking is concerned. Various reasons are suggested for this including the criteria chosen for defining smokers and nonsmokers. The higher plasma concentrations of both investigated drugs may be the result of a decreased metabolism of propranolol in older persons, in a decreased hepatic blood flow and a decreased renal elimination. Because of the lower sensitivity of β-receptors in old persons, the higher plasma concentrations of β-receptor blockers in this group of patients should not result in a diminished dose of the investigated β-receptor blockers in the elderly. (Auth. Abs. Mod.)

82–1567. Ho, C.-H.; Guerin, M. R.; Clark, B. R.; Rao, T. K.; Epler, J. L. **Isolation of Alkaline Mutagens From Complex Mixtures.** *Journal of Analytical Toxicology* 5(3): 143-147, May-June 1981.

A method for the preparative-scale enrichment of alkaline mutagens from isolates of cigarette smoke, crude petroleum, and petroleum substitutes is described. The ether soluble base fractions of two coal derived oils, a petroleum crude oil, and the two cigarette smoke condensates (CSC) were subjected to a subfractionation scheme with basic alumina and Sephadex LH-20 columns used sequentially. Up to 1 gram of the ether soluble base fraction of the pretreated sample was loaded onto the column and eluted with 500 mL benzene (benzene subfraction) followed by 700 mL ethanol. Ethanol was removed by rotary evaporation and the residue was further separated on the Sephardex LH-20 gel column. The column was eluted sequentially with 250 mL of isopropanol (isopropanol subfraction) and 600 mL of acetone (acetone subfraction). In all cases, the eluting solvent was removed by rotary evaporation leaving a residue for weighing and mutagenesis testing against *Salmonella typhimurium* strain TA98 after dissolution in dimethylsulfoxide (DMSO). It was found that the two-step approach effectively isolates alkaline mutagens from a variety of complex mixtures. Poor weight recoveries, as were observed for the CSC, are generally due to loss of samples during solvent reaction. CSC ether soluble base fractions largely contain nicotine which is easily lost by evaporation. The procedures for isolating alkaline mutagens appear generally applicable to differing complex mixtures of environmental importance. Conversely, their general applicability suggests that alkaline organics may be important environmental mutagens.

82–1568. Ioannou, Y. M.; Wilson, A.G.E.; Anderson, M. W. **Effect of Butylated Hydroxyanisole, α-Angelica Lactone, and β-Naphthoflavone on Benzo(*a*)pyrene:DNA Adduct Formation *in Vivo* in the Forestomach, Lung, and Liver of Mice.** *Cancer Research* 42(4): 1199-1204, April 1982.

The effects of α-angelica lactone (α-AL), butylated hydroxyanisole (BHA), and β-naphthoflavone (β-NF) on the amount of benzo(*a*)pyrene (BaP) metabolite:DNA adducts formed in the forestomach, lung, and liver of ICR/Ha mice were investigated 48 hr after oral administration of BaP. Analysis by high-pressure liquid chromatography showed that several BaP metabolite:DNA adducts were formed in each tissue examined. The major identified adduct in each tissue cochromatographed with the (±)-7β,8α-dihydroxy-9α, 10α-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene (BaPDEI):deoxyguanosine adduct. The (±)-7β,8α-dihydroxy-9β, 10β-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene (BaPDEII):deoxyguanosine adduct was detected in each of the tissues. As a percentage of total DNA-associated radioactivity, the BaPDEI:DNA and BaPDEII:DNA adducts accounted for 14 percent in the forestomach, 39 percent in the lung, and 3 percent in the liver. Another adduct, possibly derived from BaP:phenol(s), was detected in lung and liver. Early eluting unidentified DNA-associated radioactivity was also present in each of the tissues and accounted for the majority of the radioactivity (88 percent in forestomach, 57 percent in lung, and 97 percent in liver). Although total DNA-associated radioactivity in liver was approximately 15-fold higher than in lung and 5-fold higher than in forestomach, the specific activities of the BaPDEI:adducts and of the BaPDEI-I:adducts were approximately the same in these organs. Addition of α-AL or BHA to the diet inhibited BaPDEI:DNA adduct formation in the forestomach and liver but not in the lung. The effect of β-NF was not tissue-specific; this aryl hydrocarbon hydroxylase inducer decreased markedly (80 to 90 percent) BaPDEI:DNA adduct formation on all three tissues. The radioactivity associated with the early eluting peaks was also reduced when α-AL, BHA, or β-NF was fed to the mice. (Auth. Abs. Mod.)

82–1569. Ishihara, Y.; Sugiyama, Y.; Kitamura, S.; Kosaka, K.; Chijimatsu, Y.; Homma, H. **Kitsuen to Hai no Taisha—Kettchu Anjiotenshin I Henkan Koso Kasseii Atai, Serotonin, Hisutamin Atai ni Oyobosu Kitsuen no Koka. [Effects of Cigarette Smoking on Pulmonary Functions and on Plasma Levels of Serotonin, Histamine and Serum Angiotensin I Converting Enzyme Activity.]** *Japanese Journal of Thoracic Diseases* 19(10): 721-727, October 1981, Japanese.

Results from a study group of subjects suggest that cigarette smoking may affect pulmonary functions as well as selected plasma and serum factors. The lung is the first organ in the body to come into contact with nicotine inhaled in tobacco smoke and, thus, the possibility exists that a first-pass pulmonary metabolism of nicotine could be occurring which would be analogous to the first-pass liver metabolism of orally administered drugs such as aspirin and lidocaine in humans. Twenty-three healthy nonasthmatic volunteers consisting of 20 men and 3 women were studied. They ranged in age from 22 to 47 years, averaging 33.3 years. Five cigarettes for men and three cigarettes for women were smoked within 5 to 7 min. Peak flow, forced vital capacity (FVC), and forced expiratory volume in one second (FEV_1) were measured before, immediately after, and 20 min after smoking. Peripheral venous blood for an assay of histamine, serotonin, and angiotensin I converting enzyme (ACE) activity was obtained before, immediately after, and 20 min after smoking. Serum ACE activity displayed a significant increase immediately after smoking and returned to the control level 20 min after smoking. Plasma serotonin level showed a significant decrease immediately after smoking and stayed lower longer than 20 min. Plasma histamine level showed a marked decrease immediately after smoking and remained lower for more than 20 min. Before cigarette smoking, plasma histamine levels of smokers were significantly higher than those of nonsmokers. Peak flow values showed a significant decrease immediately after smoking but returned to the control values after 20 min. The FVC and FEV_1 showed a significant decrease immediately after smoking and were lower for over 20 min. The above results may indicate that cigarette smoking induces acute changes in pulmonary functions and in some humoral factors. (Auth. Abs. Mod.)

82-1570. Jochemsen, R.; Van Der Graaff, M.; Boeijinga, J. K.; Breimer, D. D. **Influence of Sex, Menstrual Cycle and Oral Contraception on the Disposition of Nitrazepam.** *British Journal of Clinical Pharmacology* 13(3): 319-324, March 1982.

Study results revealed that dosages of nitrazepam as a hypnotic will not need adjustment according to sex, oral contraceptive (OC) use, or menstrual cycle, but smoking status should be considered. Clearance of unbound plasma nitrazepam was studied in six healthy young males, in six healthy young females in the follicular and luteal phase of the menstrual cycle, and in six healthy young females using OC-steroids in two stages of the pill cycle. Higher values of nitrazepam intrinsic clearance, generally associated with heavy cigarette smoking, were observed, suggesting that smoking may accelerate nitrazepam clearance. No influence of the menstrual cycle on the pharmacokinetic parameters of nitrazepam was observed, nor was there a significant difference between these parameters in males and in females in either phase of the cycle. The elimination half-life was 27.3±1.3 h in males, 27.7±1.5 h in females in the follicular phase and 29.6±1.4 h in the luteal phase of the menstrual cycle. Total plasma clearance was 59.3±2.7 ml/min, 58.2±3.3 and 55.8±5.0 ml/min, respectively. Use of OC-steroids did not significantly alter the elimination half-life of nitrazepam; however, total clearance was significantly lower in females using OCs than in males. (Auth. Abs. Mod.)

82-1571. Kawano, S.; Sato, N.; Fukuda, M.; Shichiri, M.; Kamada, T.; Abe, H. **Inenmaku Ketsuryu ni Eikyo o Oyobosu Shoinshi no Kento—(Dai Ippo) Naishikyoka Hansha Supekutory Kaiseki ni Yoru Kitsuen no Eikyo no Kento. [Effect of Cigarette Smoking on the Gastric Hemodynamics. Analysis by Reflectance Spectrophotometry.]** *Japanese Journal of Gastroenterology* 79(2): 187-192, February 1982, Japanese.

In eight young healthy volunteers (5 habitual smokers, 20 cigarettes/day for 4 years, 3 unhabitual smokers, only a few cigarettes/year), the effect of cigarette smoking on the gastric mucosal hemodynamics was studied using reflectance spectrophotometry. In all cases, the gastric mucosal blood volume after one to three puffs of cigarette smoke decreased quickly. The decrease of the mucosal blood volume after cigarette smoking was 22 percent of the level before smoking in habitual smokers, while it was 63 percent of the level before cigarette smoking in unhabitual smokers. The recovery of the gastric mucosal blood volume after three puffs of cigarette smoke was faster in habitual smokers than in unhabitual smokers. The results suggest that the cigarette smoking weakens the defensive factors by lowering the gastric mucosal blood flow which would affect the occurrence and healing of the peptic ulcer. (Auth. Abs.)

82-1572. Lipsius, S. L. **Acetylcholine and Potassium-42 Movements in Right Atrial Muscle of the Guinea Pig.** *Journal of the Autonomic Nervous System* 5(3): 279-289, May 1982.

It is suggested that nicotinic receptors may mediate an acetylcholine (ACh)-induced decrease in potassium uptake in atrial muscle. The effect of ACh on potassium (K-42) movements was studied in right atrial tissue of the guinea pig. When quiescent fibers were stimulated at 108 beats/min, potassium uptake was increased to a small but significant extent. Administration of acetylcholine (2.7 X 10^{-6} M) induced an increase in potassium uptake that was greater in fibers that were quiescent than in the same fibers when stimulated. The ACh-induced increase in potassium uptake was abolished by atropine (5.2 X 10^{-7} M) and enhanced in the presence of nicotine (10^{-5} M). In addition, ACh induced an increase in potassium efflux that was greater in fibers that were quiescent than in the same fibers when stimulated. Atropine blocked the effect of ACh on potassium efflux. It is concluded that activity of atrial tissue increases potassium uptake and modifies the action of ACh on potassium uptake and efflux. In contrast to the nicotinic receptors, muscarinic receptors mediate the ACh-induced increase in potassium movements. (Auth. Abs. Mod.)

82-1573. Madle, S.; Korte, A.; Obe, G. **Cytogenetic Effects of Cigarette Smoke Condensates in Vitro and in Vivo.** *Human Genetics* 59(4): 349-352, 1981.

Various cigarette smoke condensates (CSC) were analyzed with respect to the induction of sister-chromatid exchanges (SCE) in human lymphocytes in vitro and in Chinese hamster bone marrow cells in vivo. The CSC from a reference cigarette, from three different tobaccos of the reference cigarette, and from a British cigarette induced similar, significantly increased SCE frequencies in the human lymphocytes. However, the CSC from the reference cigarette did not induce SCE in the Chinese hamster cells. (Auth. Abs. Mod.)

82–1574. Mannisto, J.; Hartiala, J.; Nienstedt, W.; Uotila, P. **The Metabolism of Arachidonic Acid in Isolated Rat Lungs Is Not Changed During Cigarette Smoke Ventilation.** *Prostaglandins Leukotrienes and Medicine* 8(1): 23-26, 1982.

Cigarette smoke ventilation of isolated perfused rat lungs partially inhibited the pulmonary vascular pressor response to arachidonic acid (AA). The amounts of metabolites of exogenous arachidonic acid in the perfusion effluent remained unchanged during smoke ventilation. The antiaggregatory effect of the effluent during pulmonary infusion of AA was not decreased by smoke ventilation. The cause of the previously reported increased platelet aggregation after smoking remains unclear. (Auth. Abs.)

82–1575. Mannisto, J.; Uotila, P. **Cigarette Smoke Ventilation Decreases Prostaglandin Inactivation in Rat and Hamster Lungs.** *Prostaglandins* 23(6): 833-839, June 1982.

Results of an investigation into the metabolism of exogenous prostaglandins E_2 (PGE_2) and $F_{2\alpha}$ ($PGF_{2\alpha}$) in isolated rat and hamster lungs indicate that the lungs do not adapt to the inhibitory effect of cigarette smoke, which is, therefore, repeated during every smoke exposure. When isolated lungs from animals were ventilated with cigarette smoke during pulmonary infusion of 100 nmol of PGF_2 or $PGF_{2\alpha}$, the amounts of the 15-keto-metabolites in the perfusion effluent were decreased. Preexposure of animals to cigarette smoke daily for 3 weeks did not change the metabolism of PGE_2 when the lungs were ventilated with air. Cigarette smoke ventilation of lungs from preexposed animals caused, however, a similar decrease in the metabolism of PGE_2 as in animals not previously exposed to smoke. After pulmonary injection of 10 nmol of ^{14}C-PGE_2 the radioactivity appeared more rapidly in the effluent during cigarette smoke ventilation suggesting inhibition of the PGE_2 uptake mechanism. In rat lungs pulmonary vascular pressor responses to PGE_2 and $PGF_{2\alpha}$ were inhibited by smoke ventilation. (Auth. Abs. Mod.)

82–1576. Mass, M. J.; Rodgers, N. T.; Kaufman, D. G. **Benzo(*a*)pyrene Metabolism in Organ Cultures of Human Endometrium.** *Chemico-Biological Interactions* 33(2/3): 195-205, January 1981.

Human endometrium maintained in organ culture is shown to have the capacity to metabolize the ubiquitous carcinogen benzo(*a*)pyrene (BaP) and this activity persists after prolonged culture. The proportion of BaP metabolites produced by unique endometrial specimens from 26 women was examined and interindividual variation was observed. Eight of the women smoked cigarettes but the variation encountered in these eight specimens did not differ from that in the 15 premenopausal nonsmokers. Specimens derived from postmenopausal women produced BaP metabolite profiles with greatly reduced BaP-sulfate conjugates which were unlike those of the statistical mean for the accumulated results with premenopausal endometrial specimens. BaP metabolism in the endometrium was studied because (1) accumulation of data regarding carcinogen metabolism for all human tissues may lead to a greater understanding of the role of metabolism as it relates to cancer-susceptible and nonsusceptible tissues; and (2) direct studies in human endometrial tissues in culture may allow the determination of the interrelationship between metabolism of steroid hormones and aromatic hydrocarbon carcinogens. (Auth. Abs. Mod.)

82–1577. Math, M. V. **High-Density Lipoprotein Concentrations Increase After Stopping Smoking. (Letter).** *British Medical Journal* 285(6334): 60-61, July 3, 1982.

A previous article (*British Medical Journal* 284(6328): 1511-1513, May 22, 1982) suggests that increases in high-density lipoprotein concentrations after smoking cessation are related to nutritional changes. In these patients, the fraction of esterified cholesterol increased at the expense of other lipid components of the lipoprotein. It is commented that nicotine causes the release of antidiuretic hormone, which may further decrease bile flow. As the liver selectively utilizes cholesterol for bile acids, the increase in bile flow after quitting may have increased the concentration of high-density esterified cholesterol, and decreased free cholesterol levels in the plasma. Alcohol intake may have contributed to this effect in the patients by suppressing the release of antidiuretic hormone. See also 82-1372.

82–1578. Matulionis, D. H. **Pulmonary Tissue and Cigarette Smoke. 1. Cellular Response to Hydrocortisone.** *Environmental Research* 27(2): 361-371, 1982.

Hydrocortisone acetate (HCA) administration significantly reduces the population of pulmonary macrophages and blood leukocytes in control, sham-treated, and smoke-exposed C57BL/6J mice. This treatment markedly impedes the influx of macrophages from bone marrow into the lungs. The small number of new phagocytes noted in lungs following HCA treatment appears to arise by proliferation of in situ pulmonary macrophages. Mortality rate of sham-treated and smoke-exposed mice was approximately twice that of control animals following HCA treatment. While severe pulmonary disorders were noted in lungs of HCA-treated, smoke-exposed animals, considerably milder abnormalities were seen in lungs of sham-treated mice. The data indicate that physical stress generated by manipulation during sham and smoke treatment, exposure to cigarette smoke, and reduction of pulmonary macrophages and leukocyte populations by HCA administration are factors which adversely affect pulmonary integrity and survival time of the animals. (Auth. Abs.)

82–1579. Mizuno, K.; Yaginuma, K.; Hashimoto, S.; Toki, T.; Nakamura, I.; Fukuchi, S. **Acute Effect of Cigarette Smoking on Serum Angiotensin-Converting Enzyme Activity in Normal Man.** *Tohoku Journal of Experimental Medicine* 137(1): 113-114, May 1982.

The acute effect of cigarette smoking on serum angiotensin-converting enzyme activity was evaluated in six healthy subjects consisting of four nonsmokers and two habitual smokers. Cigarette smoking resulted in rapid increases in serum-converting enzyme activity in five of six subjects within 5 min, and the converting enzyme activity remained above the control value at 30 min. The increase in the enzyme activity of nonsmokers was higher than that of habitual smokers at any time when the enzyme activity was determined. It is therefore suggested that cigarette smoke (or smoking) can cause the secretion of angiotensin-converting enzyme from the pulmonary endothelial cell, in which the enzyme may be produced, to the systemic circulation. It is also speculated that the increase in the enzyme activity may contribute to the initiation of cardiovascular changes associated with cigarette smoking. (Auth. Abs.)

82–1580. Muranaka, H.; Tamada, T.; Higashi, E.; Itani, S.; Morimoto, M. **Taiekichu Nikochin Oyobi Sono Taisha Sanbutsu no Biryo Teiryo: 3. Kitsuenji ni Okeru Kettchu Nikochin, Kochinin Nodo Oyobi 2,3 no Seitai Seibun no Hendo ni Tsuite. [Microdetermination of Nicotine and Its Metabolites in Biological Fluid:3. Effect of Smoking on Plasma Nicotine, Plasma Cotinine and Other Biochemical Variables in Human Being.]** *Rinsho Kagaku* 10(2): 127-135, 1981, Japanese.

Plasma concentrations of nicotine and its metabolite, cotinine, were measured by a gas-chromatographic method with a flame thermionic detector (FTD-GC) as reported previously. Plasma levels of carboxyhemoglobin (COHb), total cholesterol, HDL-cholesterol, and magnesium were also measured. Among 35 smokers, plasma nicotine and cotinine levels and COHb percentage were 12.6±1.24ng/ml, 198.9±22.27ng/ml, and 5.8±0.41 percent, respectively. Plasma nicotine in 22 nonsmokers was 2.0±0.48 ng/ml, but plasma cotinine was not detectable in most nonsmokers. COHb in nonsmokers was 1.0±0.06 percent. Total cholesterol, HDL-cholesterol, and plasma magnesium in smokers were lower than in nonsmokers. Atherogenic index, calculated from total cholesterol and HDL-cholesterol, did not show a significant difference between smokers and nonsmokers. After one cigarette, plasma nicotine level increased rapidly and reached a maximum immediately or within 5 minutes after smoking, returning to baseline values within 60 minutes. Plasma cotinine level elevated more slowly than plasma nicotine. COHb showed a change similar to plasma nicotine, but the increase was unrelated to nicotine yield of the cigarette. The decay curve of plasma nicotine after smoking had a slight fluctuation, probably due to redistribution and metabolic recycling of nicotine in vivo. Total cholesterol, HDL-cholesterol, atherogenic index, and plasma magnesium showed no significant change after smoking. Plasma nicotine and cotinine levels before or after smoking varied considerably with daily consumption, nicotine yield of the cigarette smoked, and individual smoking patterns. In this study, the plasma levels of nicotine, cotinine, and COHb correlated slightly with daily consumption and nicotine yield of cigarette. (Auth. Abs. Mod.)

82–1581. Norman, T. R.; Chamberlain, K. G.; French, M. A.; Burrows, G. D. **Platelet Monoamine Oxidase Activity and Cigarette Smoking.** *Journal of Affective Disorders* 4(1): 73-77, March 1982.

Platelet monoamine oxidase (MAO) activity and plasma thiocyanate concentrations were determined in 72 normal volunteers (22 nonsmokers and 50 smokers). Thiocyanate concentrations were used as an index of exposure to cigarette smoke. A significant negative correlation between MAO and thiocyanate concentration was observed for the group. When examined by sex, the correlation was significant for females but not for males. Mean MAO activity was significantly lower for female smokers than for female nonsmokers. For males, no significant differences in MAO activity were observed between smokers and nonsmokers. It is possible that smoking causes inhibition of MAO by a direct effect or by indirect effects such as altering hormone levels. Alternatively women with a low MAO activity have a greater innate tendency to smoke. (Auth. Abs.)

82–1582. Peto, R. **Blood Carboxyhaemoglobin, Plasma Thiocyanate, and Cigarette Consumption: Implications for Epidemiological Studies in Smokers. (Letter).** *British Medical Journal* 284(6333): 1951, June 26, 1982.

It is claimed that inappropriate statistical methods were used in a previous paper (*British Medical Journal* 284(6328): 1516-1518, May 22, 1982), which assumed that only one blood sample used to assess carbon monoxide content was an appropriate measure to test the correlation between reported and actual cigarette smoke exposure. However, a single blood carboxyhemoglobin measurement is not necessarily indicative of an individual's daily, year long mean carbon monoxide concentration. Epidemiologists are advised that reported consumption may therefore be more reliable than a single blood level measurement. More data are needed to confirm the claims made in the paper. See also 82-1247.

82–1583. Rotenberg, K. S. **The Pharmacokinetics of Nicotine.** *Pharmacy International* 3(3): 91-93, March 1982.

The pharmacokinetics of nicotine, which accounts for about 95 percent of the total alkaloid content of tobacco, and is the most potent alkaloid in tobacco smoke, is discussed in relation to its absorption, distribution, metabolism, and excretion. Although knowledge of the disposition of nicotine in humans and animals is still incomplete, it is known that the absorption of nicotine from the pulmonary tract is affected by the length of contact in the mixed alveolar air, the depth of inhalation, and the characteristic frequency of inhalation. Research involving animals indicates that nicotine achieves a rapid and wide distribution in the body, and the formation of cotinine from nicotine appears to be the main in vivo route of metabolism for most species. Nicotine's ultimate metabolic product seems to be 3-pyridylacetic acid which has been isolated in the urine of animals and humans given nicotine or cotinine. Nicotine and its metabolites are excreted primarily in urine, particularly when urine pH is acidic.

82-1584. Schwab, L. S.; Kritzer, M. F. **The Effect of Cholinergic Antagonists on a Central Response to Nicotine.** *Experientia* 38(1): 119-120, January 15, 1982.

Cannulations were carried out on male Sprague-Dawley rats and a series of cholinergic antagonists were administered 1 min before nicotine challenge (100 nmoles). Response was graded from 0 (no response) to 4 (completely prostrate and immobile) in half-point increments. An alternate-day injection regimen was utilized to delay tolerance development (e.g., day 1, nicotine only; day 3, antagonist + nicotine; day 5, nicotine; etc.) Percent antagonism was defined as 100 percent-100 percent—((response to antagonist + nicotine)/ (average of the response to nicotine alone before and after test for antagonism)). Each animal thus served as his own control; at each dose, the individual percent antagonism values (≥ 6) were ranked to give the median and 95 percent confidence limits. Mecamylamine was the only antagonist tested which afforded complete blockade of the nicotine response (at twice the molar dose of nicotine). It was also effective over a wide dose range (10 moles, i.e., one-tenth the molar dose of nicotine, and greater), but not over as broad a span as was hexamethonium. Decamethonium and d-tubocurarine were less effective, and physostigmine did not elicit the response. The results argue in favor of cholinergic involvement in the prostration-immobilization syndrome; nicotine's cholinergic action is necessary but not sufficient to explain the prostration syndrome. (Auth. Abs. Mod.)

82-1585. Simpson, W. W.; Curtis, M. B.; Priola, D. V.; Mittleman, J. **Responsiveness of Intracardiac Neurons to Cholinergic Stimulation in Normal and Thyroidectomized Dogs.** *Research Communications in Chemical Pathology and Pharmacology* 36(3): 367-382, June 1982.

The responsiveness of the intrinsic cardiac nerves (ICN) to nicotine is examined to determine if it is also affected by thyroidectomy (THX). Mongrel dogs were anesthetized and an electrode catheter was advanced into the right atrium for recording His bundle activity (HB). A second cannula was placed into the carotid artery with its tip near the coronary ostia, so that the responses to the drugs injected would be confined to the heart. Changes in the A-H interval (the time between right arterial and His bundle depolarization in msecs) of the HB were recorded in response to both acetylcholine (ACH) 2-50 ug, and nicotine HCl (NIC) 5-100 ug, before and after beta blockade with metoprolol (1-4 mg). There were no important differences between the two groups in the responses of A-V nodal conduction to ACH over the dose range used. However, the THX animals showed an apparent increase in responsiveness to NIC; i.e., ca. 5 percent decrease in A-H at 10 ug (control) compared to ca. 30 percent increase after THX. The apparent catecholamine-releasing effect of NIC was also enhanced in the THX animals. In addition, the responses to ACH and NIC were compared in animals both "on" and "off" cardiac bypass. The data suggest that hypothyroidism increases the effectiveness of nicotinic stimulation of intracardiac neurons. (Auth. Abs.)

82-1586. Stepney, R. **Are Smokers' Self-Reports of Inhalation a Useful Measure of Smoke Exposure?** *Journal of Epidemiology and Community Health* 36(2): 109-112, June 1982.

An inhaling smoker's self-report of inhalation depth was found to be of little use in predicting actual exposure to toxic smoke components. The relation between self-assessed and objective measures of inhalation was studied in 75 smokers who assigned themselves to one of four inhalation categories, and who also estimated inhalation using a rating scale. The analysis of presmoking carbon monoxide concentration in expired air, and of the rise in carbon monoxide concentration over smoking, provided an objective measure of inhalation. There was a weak but significant correlation between self-rated inhalation and rise in carbon monoxide, but no correlation with the longer term exposure measured by presmoking levels of carbon monoxide. Differences in exposure to carbon monoxide according to self-assessed inhalation category were nonsignificant. It is also suggested that any attempts to estimate inhalation by using a rating scale would not prove any more useful in assessing long-term exposure to smoke than the method of asking smokers to assign themselves to an inhalation category. To achieve more useful results, not only must the depth of inhalation be considered but perhaps the duration of inhalation and the pattern of puffing as well. (Auth. Abs. Mod.)

82-1587. Stich, H. F.; Stich, W. **Chromosome-Damaging Activity of Saliva of Betel Nut and Tobacco Chewers.** *Cancer Letters* 15(3): 193-202, 1982.

Saliva of volunteers chewing betel quid, cured betel nut (*Areca catechu*), betel leaves (*Piper betle*), a mixture of quid ingredients (dried betel nut flakes, catechu, cardamon, lime, copra, and menthol), and Indian tobacco was collected and examined for its genotoxic activity. Chromosome aberrations (chromatid breaks and chromatid exchanges) in Chinese hamster ovary (CHO) cells were used to estimate the genotoxic effect. After 5 min of chewing, the betel quid, betel nut, betel leaves, quid ingredients, and Indian tobacco all showed relatively potent clastogenic activities. The addition of transition metals Mn^{2+} and Cu^{2+} to the saliva samples of betel nut and Indian tobacco chewers enhanced their clastogenic activities, whereas Fe^{3+} increased the clastogenicity of the betel nut saliva but decreased the genotoxic effect of the saliva of Indian tobacco chewers. After removal of the betel quid or its components from the mouth, the clastogenic activity disappeared within 5 min. The western-type chewing tobacco did not produce a genotoxic activity in the saliva of chewers. No detectable levels of clastogenic activity were observed in the saliva of nonchewing individuals. (Auth. Abs. Mod.)

82–1588. Stich, H. F.; Stich, W.; Lam, P.P.S. **Potentiation of Genotoxicity by Concurrent Application of Compounds Found in Betel Quid: Arecoline, Eugenol, Quercetin, Chlorogenic Acid and Mn^{2+}.** *Mutation Research* 90(4): 355-363, December 1981.

Five components of betel quid were examined for their clastogenic activities individually and in various combinations. The clastogenic activity was estimated as the frequency of metaphase plates with at least one chromatid break or chromatid exchange, or the average number of chromatid breaks and exchanges per Chinese hamster ovary (CHO) cell. The betel quid components included the alkaloid, arecoline from the betel nut; eugenol from the betel vine; chlorogenic acid from tobacco leaves; quercetin from fennel seeds; and the ubiquitous transition metal Mn^{2+}. The clastogenic effects of the concurrent applications of arecoline plus eugenol, arecoline plus quercetin, and arecoline plus chlorogenic acid were greater than the sum of the action of each individual component. Similarly, the combinations of arecoline, chlorogenic acid, and Mn^{2+} induced frequencies of chromosome aberrations which exceeded the sum of the clastogenic activities of individually applied compounds or the sum of the clastogenic activities of two jointly applied compounds (arecoline plus Mn^{2+}, or chlorogenic acid plus Mn^{2+}). A potentiating (enhancing) action was also evident when two clastogens were used at doses which would not lead to a detectable increase in the frequency of chromosome aberrations when applied individually. (Auth. Abs. Mod.)

82–1589. Stockley, R. A.; Afford, S. C.; Burnett, D. **The Electrophoretic Mobility of α_1-Proteinase Inhibitor: Effects of Proteolysis and Cigarette Smoke.** *Zeitschrift fur Physiologische Chemie* 363(4): 387-393, April 1982.

An aqueous solution of cigarette smoke increased the electrophoretic mobility of pure α_1-proteinase inhibitor. Factors which affect the inhibitor may act as determinants in the development of emphysema. The electrophoretic mobility of purified α_1-proteinase inhibitor was compared with that of carbamoylated transferrin. The results ranged from 64.0 to 68.9 percent of the distance moved by the transferrin and were increased by cigarette smoke solution (range 70.4 percent to 75.0 percent of carbamoylated transferrin). Studies on sputum showed a wide range of mobility from 68.0 to 45.0 percent with no difference in mobility between samples from smokers and nonsmokers. (Auth. Abs. Mod.)

82–1590. Stoner, G. D.; Daniel, F. B.; Schenck, K. M.; Schut, H.A.J.; Goldblatt, P. J.; Sandwisch, D. W. **Metabolism and DNA Binding of Benzo(*a*)pyrene in Cultured Human Bladder and Bronchus.** *Carcinogenesis* 3(2): 195-201, 1982.

Explants of human bladder and bronchus were fully capable of metabolizing the carcinogen, benzo(*a*)pyrene (BaP) into forms that were bound covalently to cellular DNA. Three-day cultures were exposed to radiolabeled BaP for 24 hours, and metabolism was determined by analysis of the level of binding of reactive metabolites to DNA, and by the release of metabolites into the medium. For a given individual, the DNA binding level and extent of metabolism was usually higher in the bladder than in the bronchus. In specimens obtained from 16 individuals, the average DNA-binding levels were 6.4 ± 5.0 µmol BaP/mol deoxyribonucleotide for the bladder and 3.1 ± 1.9 µmol BaP/mol deoxyribonucleotide for the bronchus. In tissues obtained from the same individual, the binding levels of BaP metabolites to bladder cell DNA was not strongly correlated to that of bronchial cell DNA ($r=0.54$). The media of both tissues contained small amounts of free, unconjugated metabolites of BaP (<3 percent of the total) and large amounts (30 to 85 percent of the total) of unidentified, highly polar material. Human bladder appears to be the most active human explant tissue yet studied with respect to its ability to activate BaP to DNA binding forms. The relevance of this observation to human bladder cancer is, as yet, unknown. (Auth. Abs. Mod.)

82–1591. Sulochana, G.; Arunagiri, R.; Panchanathan, T. B. **Effect of Smoking on Serum Cholesterol, Phospholipid and Free Fatty Acid.** *Clinician* 45(8): 322-325, August 1981.

Smoking five cigarettes within 30 minutes significantly decreased phospholipid and very significantly increased free fatty acid concentrations. The acute effects of smoking were tested in 25 healthy male subjects, 19 to 30 years old, in a fasting state and after a 10-hour lapse from their last cigarette. No significant differences in cholesterol levels were observed.

82–1592. Toda, N.; Hayashi, S. **Responses of Canine Coronary Arteries to Transmural Electrical Stimulation and Nicotine.** *European Journal of Pharmacology* 80(1): 73-81, May 7, 1982.

In helical strips of dog coronary arteries contracted with prostaglandin $F_{2\alpha}$, transmural electrical stimulation and nicotine elicited a transient relaxation. The response to electrical stimulation was abolished by tetrodotoxin, bretylium, or pretreatment of the dogs with reserpine, and was potentiated by cocaine. Atropine was ineffective. The relaxations were abolished or converted to contractions by sotalol; the contraction was suppressed by phentolamine. The nicotine-induced relaxation was suppressed by hexamethonium, cocaine, bretylium, and sotalol. When nicotine was present in the media, further addition of nicotine failed to produce relaxation. However, nicotine dose—response curves were obtained with single-dose washings. Atropine and tetrodotoxin did not affect the response. Following pretreatment with reserpine, relaxant responses to nicotine were partly reduced; the remaining relaxations were suppressed by atropine, slightly inhibited by sotalol, and potentiated by physostigmine. It may be concluded that electrical stimulation liberates norepinephrine from adrenergic nerves, which preferentially activates β-adrenoceptors. The reuptake mechanism appears to function to inactivate liberated norepinephrine. Nicotine-induced relaxations are mediated mainly by liberated norepinephrine; however, as shown by the pretreatment with reserpine, the release of an acetylcholine-like substance may also be involved. (Auth. Abs. Mod.)

82–1593. Topping, D. C.; Martin, D. H.; Nettesheim, P. **Determination of Cocarcinogenic Activity of Benzo(*e*)pyrene for Respiratory Tract Mucosa.** *Cancer Letters* 11(4): 315-321, February 1981.

The importance of the target tissue as a determining factor in cocarcinogenic activity was demonstrated in the tracheal mucosa of rats exposed for 6 months to both benzo(*a*)pyrene (BaP) and benzo(*e*)pyrene (BeP). At BaP/BeP ratios of 1:1 and 0.5:1, BeP had no cocarcinogenic effects on tracheal epithelium, and at the higher BaP/BeP ratio, BeP appeared to reduce the carcinoma incidence from 65 percent (BaP alone) to 40 percent (BaP plus BeP). In sharp contrast to the carcinoma incidence, the tracheal and peritracheal sarcoma incidence was enhanced twofold to threefold by BeP. Thus, while not cocarcinogenic for tracheal epithelium, BeP was cocarcinogenic for connective tissue. Thus the target tissue type is a decisive factor in determining the activity of BeP as a cocarcinogen. (Auth. Abs. Mod.)

82–1594. Watanabe, M.; Abe, T. **Aryl Hydrocarbon Hydroxylase and Its Components in Human Lung Microsomes.** *Gann: Japanese Journal of Cancer Research* 72(5): 806-810, October 1981.

See also, **82–1678**

No correlation between smoking and the activity of the aryl hydrocarbon hydroxylase (AHH) system or the levels of its components was found in lung tissue samples acquired from 16 patients suffering from pulmonary tumors (14 diagnosed carcinomas) and undergoing pneumonectomy. Lung microsomal fractions were measured for the controls of cytochrome P-450 (P-450) and of cytochrome b_5 (b_5), as well as for the activities of NADPH-cytochrome P-450 reductase (f_{PT}) and NADH-cytochrome b_5 reductase (f_{PD}). Results indicated that AHH activities found in lung microsomes from those patients with bronchogenic carcinoma were very low regardless of smoking habit. The P-450 was undetectable. No correlation was discovered between smoking and the levels of f_{PT}, f_{PD}, and b_5.

82–1595. Wietholtz, H.; Voegelin, M.; Arnaud, M. J.; Bircher, J.; Preisig, R. **Assessment of the Cytochrome P-448 Dependent Liver Enzyme System by a Caffeine Breath Test.** *European Journal of Clinical Pharmacology* 21(1): 53-59, October 1981.

Based on preliminary rat studies, (7-methyl-^{14}C) and (3-methyl-^{14}C) caffeine were chosen for assessing the feasibility of a caffeine breath test in man, using five normal volunteers and two patients with compensated liver cirrhosis. Since the variability of the levels of the specific activity in subjects with normal livers suggested the influence of extraneous factors, a second group of normal volunteers, smokers and nonsmokers, was investigated. The methyl labels clearly discriminated between smokers and nonsmokers; the exhalation rate of $^{14}CO_2$ peaked faster in smokers, and on average was at least double the rate in nonsmokers. $^{14}CO_2$ exhalation curves in cirrhotics were clearly different from those in normal volunteers, characterized by a slower rise and a lower overall exhalation rate. The more rapid achievement of a peak, particularly in smokers, and the increased level of specific activity following administration of (3-methyl-^{14}C) caffeine support the contention that demethylation in position 3 represents the dominant metabolic pathway in man. It is concluded that specifically labeled caffeine is a suitable and promising substrate for studying demethylation by breath analysis. Presumably, caffeine represents a safe and sensitive indicator of the activity of the cytochrome P-448 system. (Auth. Abs. Mod.)

MORTALITY AND MORBIDITY

82–1596. Bottiger, L. E.; Carlson, L. A. **Risk Factors for Death for Males and Females.** *Acta Medica Scandinavica* 211(6): 437-442, 1982.

In the Stockholm Prospective Study involving 6,224 persons, 531 male and 163 female deaths had occurred during 14.5 years of observation. Deaths were divided into three categories: "ischemic," "neoplastic," and "other" and their relationship to entry characteristics were analyzed. For ischemic vascular deaths age, smoking, systolic blood pressure, and triglycerides were independent risk factors for both sexes, while cholesterol and weight/height index were not. For neoplastic deaths age, smoking, systolic blood pressure, and erythrocyte sedimentation rate (ESR) were positively and cholesterol and weight/height index negatively associated. This negative association was found especially for colon carcinoma and not for, e.g., prostatic and bronchial carcinoma in men. For other deaths age, blood pressure, and ESR were independent risk factors for men, and age, smoking, and ESR for women. (Auth. Abs.)

82–1597. McMichael, A. J.; Hartshorne, J. M. **Mortality Risks in Australian Men by Occupational Groups, 1968-1978. Variations Associated With Differences in Drinking and Smoking Habits.** *Medical Journal of Australia* 1(6): 253-256, March 20, 1982.

Male mortality rates among 9 occupational groupings in Australia were analyzed. The risk of mortality from liver cirrhosis and lung cancer, the sentinel diseases for drinking and smoking behavior, was consistently high in the mining, transportation, trades, and service groups (over 40 percent were smokers). With few exceptions, so was the mortality risk from alcohol-related and cigarette-related diseases, including cerebrovascular disease. For service, sport, and recreation workers, cigarette-related risk of mortality was not elevated, although alcohol-related mortality risk was. Among the professional, technical, administrative, clerical, sales workers, and farmers, the risks of mortality from diseases related to drinking and smoking were generally below average, particularly for professional and technical workers. In contrast, the risks of mortality from cancer of the colon, thought to be influenced by dietary "affluence," are consistently higher for professional and white-collar groups, though stomach cancer mortality risks are higher for service and blue-collar groups. Clerical workers had a slightly elevated risk of mortality from cardiovascular disease. The rural workers had a distinctly high risk of mortality from alcoholism and alcoholic psychosis, although their risk of mortality from liver cirrhosis was low. (Auth. Abs. Mod.)

82–1598. Rosenfield, A. **The Pill: An Evaluation of Recent Studies.** *Johns Hopkins Medical Journal* 150(5): 177-180, May 1982.

The net yearly death rate associated with use of oral contraceptives (OC) per 100,000 women is 6.5 in smokers compared with 1.8 in nonsmokers. These excess deaths are mainly due to cardiovascular complications: smoking women who did not use OCs were found to have a 7-fold risk for myocardial infarction, while nonsmoking OC users were at a 4-fold risk, and women who both smoked and used OCs were at a 39-fold risk. In summarizing the known risks of OC use, it is noted that if only nonsmokers used the pill, oral contraceptive-associated mortality would decrease by at least half. No increased risk of cancer, infertility, or poor pregnancy has been found, and some benefits have been observed. For instance, OC users are less likely to have benign breast disease, ovarian cysts, or iron deficiency anemia, and only half as likely to suffer from arthritis or pelvic inflammatory disease. Thus, for nonsmokers under age 30, mortality risk is lower than for those using no birth control, although for smokers the risk is 2.5 times higher.

See also, **82–1714**

NEOPLASTIC DISEASES

82–1599. Andreasson, L.; Bjorlin, G.; Korsgaard, R.; Mattiasson, I.; Trell, E. **Munhalecancer i Relation Till Rokning, AHH-Inducibilitet och Oral Halsa. [Carcinoma of the Oral Cavity in Relation to Smoking, AHH-Inducibility and Oral Health.]** *Lakartidningen* 78(35): 2953-2957, 1981, Swedish.

Etiological factors, some clinical details, and methods and results of treatment of carcinoma of the oral cavity have been investigated and evaluated in 20 consecutive cases (16 males, 4 females, average age 64.7 years) seen at the departments of otorhinolaryngology and oral surgery, Malmo General Hospital, between the years 1976-1979. All of the men and two of the women were smokers. In the male patients, there was an overrepresentation of heavy alcohol consumers. The tumor localization involved the tongue in 10 of the cases. Thirteen of the tumors were advanced but resectable, while two were nonresectable. The aryl hydrocarbon hydroxylase (AHH) inducibility was investigated in the data and there was a statistically significant ($p<0.001$) overrepresentation of cases with high AHH inducibility and an underrepresentation of cases with low AHH inducibility. It is concluded that smoking is an important exogenous factor associated with the development and course of oral cancer, and that high AHH inducibility may be a genetically determined, constitutional background to increased susceptibility for smoke-dependent carcinogenesis in the oral cavity. (Auth. Abs.)

82–1600. Armstrong, B. **An Epidemiological Basis for Cancer Control.** *Cancer Forum* 5(3): 136-143, September 1981.

The role of smoking in the rapidly increasing incidence of cancer in Australia, which now accounts for six times as many deaths as road accidents, is discussed along with other environmental and lifestyle factors considered vital to primary (prevention) and secondary (early treatment) control. Prevention of all cancers may be impossible, but health education and lifestyle changes, particularly in terms of diet and smoking cessation, would greatly reduce cancer morbidity and mortality. Priority for prevention action is suggested for cancers of the lung, breast, and colon, which together account for over 40 percent of new cases and premature mortality. Smoking is considered the prime risk factor for lung cancer, which has the largest number of new cases and the highest early mortality rate. Cessation of smoking, which can be an essential part of either primary or secondary control of lung cancer, could also reduce incidence of other cancers where smoking is one of the risk factors. Diet and overexposure to sunlight were determined to be the other high risk factors for the cancers studied, but use of oral contraceptives, alcohol, and coffee, as well as occupational exposures, were found to be risk factors for the development of specific types of cancer.

82–1601. Auerbach, O. **Pathogenesis of Lung Cancer.** *Comprehensive Therapy* 7(12): 11-21, December 1981.

Morphologic studies confirm the epidemiological evidence that lung cancer is caused by smoking. Microscopic examination of lung sections of both smokers and nonsmokers revealed a gradient of cellular changes with continued smoke exposure. The first response to inhalation of smoke occurs among the basal cells of the tracheobronchial epithelium. The basal cells pile up additional layers, with cells varying in shape and many having atypical nuclei, resulting in basal cell hyperplasia. The degree of atypism parallels the amount of inhaled carcinogens. The second stage is loss of ciliated and mucous cells, though normal cells are still present. One of three lesions results from loss of ciliated cells; squamous metaplasia, stratification of the luminal surface, or carcinoma in situ (area of preinvasive cancer). Continued exposure to smoke stimulates proliferation of anaplastic cells until the underlying tissues are penetrated. Studies have shown that a reversal of these histologic changes occurs with cessation of smoking if the underlying tissues have not been invaded. Smoke inhalation experiments with dogs showed lower levels of cellular alterations using lower yield cigarettes. A classification of lung neoplasms according to sites of origin and according to histologic type is also given.

82–1602. Barthel, E. **Increased Risk of Lung Cancer in Pesticide-Exposed Male Agricultural Workers.** *Journal of Toxicology and Environmental Health* 8(5/6): 1027-1040, November-December 1981.

A causative relation between tobacco consumption and increased lung cancer mortality in a group of pesticide-exposed agricultural workers was rejected. The cancer morbidity in 1,658 male German agricultural workers exposed to pesticides was investigated through a retrospective cohort study. A total of 169 malignant tumors were diagnosed in the men who began to work with pesticides between 1948 and 1972, and who continued this type of activity for at least 5 years. The standardized mortality ratio (SMR) of 2.0 for lung cancer morbidity (mortality) in these pesticide-exposed subjects was significantly higher than that for the general male population of East Germany. The smoking habits of the exposed workers were studied by analyzing the responses of a random sample of 163 subjects to a standardized questionnaire. The control group consisted of an identical number of unexposed persons of the same age and sex selected at random during a routine chest X-ray examination of the general public. No significant differences could be demonstrated between the smoking habits or the tobacco consumption of the two groups. The increase in the slope of the SMR with increasing duration of exposure also provides evidence against such a causative connection with tobacco smoking. Finally, the types of carcinomas found, with a predominance of small-cell and undifferentiated tumors, also represent an argument against the assumption that the cancers were caused by smoking, because smokers' carcinomas are known to be predominantly of the squamous epithelium type. A positive correlation between the duration of employment and the mortality due to lung cancer suggested a dose—effect relation. (Auth. Abs. Mod.)

82–1603. Blot, W. J.; Davies, J. E.; Brown, L. M.; Nordwall, C. W.; Buiatti, E.; Ng, A.; Fraumeni, J. F., Jr. **Occupation and the High Risk of Lung Cancer in Northeast Florida.** *Cancer* 50(2): 364-371, July 15, 1982.

In Duval County (Jacksonville), Florida, the age-adjusted rate for lung cancer, 1970-1975, among white males was the highest of all urban counties in the United States. Interviews with 321 male lung cancer patients and 434 controls, or their next of kin, were undertaken to identify reasons for the high mortality rate. Increased risks of 40-50 percent were associated with the shipbuilding, construction, and lumber/wood industries. Among shipyard workers and workers in the lumber/wood industry, an excess risk was shown among cigarette smokers, raising the possibility of a synergistic or cocarcinogenic effect between asbestos or wood dust and smoking. Excess risks were also linked to fishing and forestry though the number of cases was small. Although it is difficult to disentangle the effects of smoking, chronic obstructive lung disease has been reported to predispose to lung cancer, and emphysema was more often reported for cases and controls associated with the shipbuilding and construction industries. Though occupational factors did not appear to fully account for the area-wide excess of lung cancer, and no evidence was found to implicate smoking habits, migratory patterns, or diagnostic and reporting practices, it is speculated that smoking-related chronic pulmonary disease may have contributed to the area's excess lung cancer rates.

82–1604. Brugere, J.; Lavet, C.; Asselain, B.; Thanh, P. **Cancers des Voies Aero-Digestives Superieures et Facteurs Professionnels. Resultats d'une Enquete Prospective Portant sur 1934 Cas. [Cancers of the Superior Respiratory-Digestive Tract and Occupational Factors. Results of a Prospective Survey of 1934 Cases.]** *Archives des Maladies Professionnelles* 42: 307-310, 1981, French.

Etiologic and epidemiologic factors for epidermoid carcinoma of the buccal cavity, oropharynx, larynx, and hypopharynx were analyzed in 1,738 men and 196 women. Most (98.5 percent) of the men but only 58 percent of the women were regular smokers. Only 10 percent of men versus 23 percent of women smoked less than 10 cigarettes per day. On the average, a male patient consumed 27 g of tobacco per day. The daily tobacco consumption was significantly higher in the patients who developed cancer before the age of 50. Of the patients sampled, 18 percent started smoking before the age of 15: 30 percent of these had the carcinoma before the age of 50. Among the 34 percent of the patients who started smoking after the age of 20, only 20 percent developed carcinoma before the age of 50. Ninety-five percent of the male patients have been smoking for over 20 years. Only 1 out of 28 nonsmoking male patients and only 3 out of 83 nonsmoking female patients had been drinking over 80 g of alcohol/day. All epidermoid carcinoma cases studied except glottic cancer are characterized as men aged 45 to 65, alcoholics, and smokers. Only a reduction in tobacco and alcohol consumption may lead to a decrease in incidence of the studied cancers.

82–1605. Burch, P.R.J. **Smoking, Lung Cancer and Hypothesis Testing.** *Medical Hypotheses* 7(12): 1461-1470, December 1981.

It has been shown previously that the precipitator hypothesis of causation is able to account for certain key features of the epidemiological evidence that conflict with initiator and promoter hypotheses. The precipitator hypothesis states that the carcinogenic risk depends linearly on the average rate of smoking at an interval of τ years before death and that the "doubling dose-rate" of D cigarettes per year is constant with respect to age from 35 years and above. In an earlier paper this hypothesis was tested by analyzing secular trends in sex- and age-specific mortality from lung cancer recorded in England and Wales in relation to secular trends in cigarette consumption. The periods considered extended from 1950 up to 1975. The great majority of data failed to support the precipitator hypothesis but the well-known errors in death certification were undoubtedly responsible for many anomalies. There were some indications of improved diagnostic accuracy toward the end of the period and hence it was considered important to update the analysis. This paper considers the trends over the recent period, 1974-75 to 1977-78. Unfortunately, these newer data also appear to contain anomalies and they fail to support the precipitator hypothesis. In view of the difficulties confronting initiator and promoter hypotheses, there is little alternative but to adopt Oldham's agnostic attitude " ...we still do not know how cigarettes cause lung cancer, nor even, if we are particularly rigorous in our use of scientific logic, whether they do." (Auth. Abs.)

82–1606. Cohen, W. L. **Herpes Simplex Virus and Oral Cancer.** *Dental Hygiene* 55(11): 31-39, November 1981.

Tobacco use is identified as a significant factor in the development of oral cancer in a study of the association between herpes simplex virus and oral cancer. The carcinogenicity of herpes viruses is discussed for oral and other sites of cancer growth, and characteristics of the growths are described. Early detection is important for oral cancer treatment; and survival rates, which depend on cell type, site, local extension, degree of metastasis, and adequacy of treatment, are also affected by health habits, such as alcohol consumption and tobacco use. The association between tobacco use and oral cancer has been demonstrated—twice as many smokers have been found among oral cancer patients as compared to controls. Cessation of smoking has been shown to decrease the risk. A relationship has also been shown for the use of pipes and lip carcinoma, and for tobacco chewing and carcinomas arising at sites where the tobacco was held. Additional research on the role of herpes simplex virus should prove its association with oral cancer, but other factors, such as tobacco use and alcohol consumption, must also be taken into consideration.

82–1607. Cooper, M. A.; Hallgrimsson, J. **Tumours in Iceland. 4. Tumours of the Upper Respiratory Tract and Ear. A Histological Classification and Some Etiological and Epidemiological Considerations.** *Acta Pathologica et Microbiologica Scandinavica; Section A: Pathology* 89(5): 377-387, 1981.

All primary tumors from the upper respiratory tract and ear submitted for a histological diagnosis in Iceland during the 20 years, 1955-1974, were reviewed and typed according to the World Health Organization Classification published in 1978. The larynx was the most common site for malignant tumors of the upper respiratory tract in males and the third in order in females. Smoking and alcohol consumption, well-known risk factors for laryngeal cancer, have been growing continuously in Iceland. The sale of cigarettes/year/person in Iceland rose from 200 cigarettes in 1930 to 450 in 1940, to 900 in 1950, to almost 1,600 in 1974. Allowing for a cancer latency period of around 20 years, the increased incidence of laryngeal cancer in 1965-1974 may be related to the sharp rise in smoking and drinking in 1940-1950. In addition, the increase in smoking among Icelandic children and adolescents, particularly females, indicates a continuing rise in the incidence of laryngeal cancer.

82–1608. Cuckle, H. S.; Kinlen, L. J. **Coffee and Cancer of the Pancreas.** *British Journal of Cancer* 44(5): 760-761, November 1981.

As smoking is an established risk for pancreatic neoplasms, it was accounted for in analyzing the relationship between coffee consumption and incidence of cancer of the pancreas. Coffee consumption data were collected for the 16 member countries of the International Coffee Organization for which the World Health Organization holds sufficient pancreatic cancer mortality statistics. Changes in lung cancer mortality were used as an index of the changes in smoking-related pancreatic cancer. The mortality correlation coefficient was .43 for males and .41 for females; after adjustment for smoking, the coefficient was .47 for males and .61 for females.

82-1609. De Caro, L.; Benfield, J. R. **Lung Cancer in Young Persons.** *Journal of Thoracic and Cardiovascular Surgery* 83(3): 372-376, March 1982.

A comparison of the records of 35 lung cancer patients younger than 40 (study group) with the records of 683 lung cancer patients over 40 (control group) reveals a higher percentage of women and inordinately heavy smokers among the younger patients. The study group included 21 men and 14 women (1.5:1) with all but one, a nonsmoker, smoking more than two packs of cigarettes per day. The control group consisted of 570 men and 113 women; smoking histories were not sufficiently detailed to permit precise analysis. The comparison also showed a predominance of adenocarcinomas and a high incidence of small (oat) cell undifferentiated cancers in young patients, and a survival rate for nonoperatively treated young patients which was strikingly worse than that of older patients. However, the survival rate of young persons whose tumors were resected was equal to and perhaps exceeded that of the control group.

82-1610. Fain, P. R.; Lynch, H. T.; Albano, W. A.; Ruma, T. A. **Sex Differences in Lung Cancer Incidence: A Genetic Model.** *Medical Hypotheses* 7(8): 1109-1112, 1981.

Family histories pertaining to cancer of all anatomic sites were ascertained on 88 Caucasian patients (61 males, 27 females) with histologically verified lung cancer. Lifetable analysis revealed that relatives of female probands had a significantly higher risk for cancer (all sites) at younger ages ($p < .04$) compared to relatives of male probands. This trend was peculiar to nonsmoking-associated cancer sites, and was not apparent for smoking-associated cancer sites. Certain components of genetic liability to lung cancer appear to be common to nonsmoking-associated malignant neoplasms, and females who develop lung cancer appear to be more extreme with respect to genotype than are males who develop lung cancer. This hypothesis accounts for the lower incidence of lung cancer among females, as well as the apparent increased susceptibility to cancer among their relatives. It is concluded that effective programs for cancer prevention should be aimed at genetically predisposed segments of the population. (Auth. Abs. Mod.)

82-1611. Gardner, J. W.; Lyon, J. L. **Cancer in Utah Mormon Men by Lay Priesthood Level.** *American Journal of Epidemiology* 116(2): 243-257, August 1982.

Mormons have been shown to have low cancer rates at several common sites, particularly those associated with tobacco and alcohol use. This likely reflects adherence to their church doctrines advocating abstention from the use of these substances. Not all Mormons, however, adhere to the health practices of their church, and this study classifies Utah Mormon men by their lay priesthood offices, which reflect degree of adherence to church doctrines. Followup cancer rates for 1966-1970 indicate that the most devout group (Seventies and High Priests) have lung cancer rates 80 percent lower than those of the least devout group. The same was seen for all smoking- and alcohol-associated cancer sites combined. Cancer of the stomach and the leukemias and lymphomas also had lower rates in the most devout group. Cancers of the colon-rectum, prostate, and pancreas showed little difference in rates when classified by lay priesthood office. These data provide a demonstration of the effects of a healthy lifestyle on cancer occurrence in men. (Auth. Abs.)

82-1612. Gardner, J. W.; Lyon, J. L. **Cancer in Utah Mormon Women by Church Activity Level.** *American Journal of Epidemiology* 116(2): 258-265, August 1982.

Female Mormon cancer patients in Utah were classified according to measures of adherence to church doctrines. The distribution by church activity level is compared for each site to a group of other cancer sites felt to represent the overall activity level distribution of Utah Mormon women. An inverse association of tobacco- and alcohol-related cancers with church activity level was demonstrated among Mormon women. However, this association was not so strong as that seen in Mormon men, possibly due to the smaller likelihood for nonactive women to smoke, or the inadequacy of the church-involvement scoring system. When smoking-related neoplasms were excluded, cancer of the uterine cervix also showed lower rates in the more active groups, but this finding was not statistically significant. Cancers of the breast and ovary did not show consistent associations with church activity level, nor did most of the gastrointestinal cancers. (Auth. Abs. Mod.)

82-1613. Garner, R. C.; Mould, A. J.; Lindsay-Smith, V.; Cartwright, R. A.; Richards, B. **Mutagenic Urine From Bladder Cancer Patients. (Letter).** *Lancet* 2(8294): 389, August 14, 1982.

A pilot study found mutagens to be present in the urine of both smoking and nonsmoking bladder cancer patients. It was reasoned that although the cancer may have been diagnosed many years after initiation of the disease, the bladder mucosa would still be exposed to the original mutagens. No clear pattern was seen in the mutagenic response when analyzed with regard to the assay, suggesting that the mutagens were chemically diverse from sample to sample. As the influence of smoking tends to mask the presence of mutagens from noncigarette sources, future studies of this type should concentrate on data from nonsmokers.

82-1614. Ginns, L. C.; Goldenheim, P. D.; Miller, L. G.; Burton, R. C.; Gillick, L.; Colvin, R. B.; Goldstein, G.; Kung, P. C.; Hurwitz, C.; Kazemi, H. **T-Lymphocyte Subsets in Smoking and Lung Cancer. Analysis by Monoclonal Antibodies and Flow Cytometry.** *American Review of Respiratory Disease* 126(2): 265-269, August 1982.

A number of abnormalities in circulating immunoregulatory T-cells was found both in smokers and in lung cancer patients. Peripheral T-lymphocytes in 26 patients with untreated lung cancer were characterized. The results in patients with primary squamous cancer (SC) ($n = 10$), primary adenocarcinoma (AC) ($n = 7$), and secondary lung metastases (M) ($n = 9$) were compared with each other and to subjects without cancer ($n = 48$), including nonsmokers ($n = 29$) and smokers ($n = 19$). OKT3^{+} (mature, peripheral) T-lymphocytes, including both OKT4^{+} (inducer/helper) and OKT8^{+} (cytotoxic/suppressor) lymphocytes, were increased in light-to-moderate smokers, but OKT4^{+} cells were decreased in heavy smokers. The ratio of OKT4^{+} to OKT8^{+} (4/8) lymphocytes, reflecting the balance of immunoregulatory cells, was normal in light-to-moderate smokers, but was decreased in heavy smokers. The profile of circulating T-cells in patients with SC was similar to the smokers. In contrast, in patients with AC, a decreased percentage of OKT8^{+} cells was found. The 4/8 ratio was elevated in patients with AC. In patients with M, there was a decreased percentage of OKT3^{+} cells reflected in both OKT4^{+} and OKT8^{+} subsets. The 4/8 ratio in patients with M was low. The results suggest that immunoregulatory abnormalities contribute to the pathogenesis of lung cancer. (Auth. Abs. Mod.)

82–1615. Heilbrun, L. K.; Nomura, A.; Stemmermann, G. N. **The Effects of Nonresponse in a Prospective Study of Cancer.** *American Journal of Epidemiology* 116(2): 353-363, August 1982.

Data obtained by the Honolulu Heart Program from 9,877 Japanese men on the island of Oahu, Hawaii, in 1965, and after 10 years of followup, hint at but do not statistically support an association of cigarette smoking level and lung cancer risk. Of 11,136 subjects contacted, 8,006 responded to a mailed questionnaire and were examined; 1,871 only responded to the questionnaire. Of the examinees, 56.6 percent were current cigarette smokers as opposed to 61.6 percent of the unexamined men. After followup, the examined men had a significantly lower risk of death from all causes, death from cancer, and incidence of stomach cancer than the unexamined men. Minimal differences were also noted between the two groups in the time interval between the beginning of followup and the diagnosis of cancer of the lung and large bowel, and these favored the examined men. The examined men had a higher risk of prostate cancer incidence, although not significantly so. When the 8,006 examined and 1,871 unexamined men who responded to the mailed questionnaire were evaluated with respect to the association of cigarette smoking with lung cancer incidence, the age-adjusted odds ratio estimate for smokers was 19 for the examined men and 7 for the unexamined men, a threefold difference. However, since these odds ratios are not significantly different, there would be little bias in odds ratio estimates of cigarette smoking for lung cancer if the observation was limited to examined men. With regard to an association of low education with stomach cancer, the odds ratio estimates were nearly identical at 1.2 for the examined group and 1.1 for the unexamined group. (Auth. Abs. Mod.)

82–1616. Herity, B.; Moriarty, M.; Bourke, G. J.; Daly, L. **A Case—Control Study of Head and Neck Cancer in the Republic of Ireland.** *British Journal of Cancer* 43(2): 177-182, February 1981.

A retrospective case—control study in Dublin, Ireland, of 200 patients with head and neck cancer, and 200 controls matched for age and sex, found a male—female ratio of 3:1 for these malignancies and confirmed the strong association between smoking and alcohol consumption in the etiology of these diseases. Among the male cases, whose mean age was 63 years, 63.1 percent were heavy smokers compared with 31.6 percent of controls, and only 5.3 percent were nonsmokers compared with 15.8 percent of controls. Only 8 of a total of 152 tumors occurred in nonsmokers, 3 of which were parotid tumors and 2 paranasal sinus tumors, which are not strongly associated with smoking. Tobacco consumption was most strongly associated with the development of cancer of the larynx in this series of patients, but all the head and neck tumors with the exception of tumors of the nasopharynx and paranasal sinuses were significantly increased in male smokers. Alcohol consumption was also much higher among male cases than controls. Apart from the female cases of laryngeal cancer, who were all heavy cigarette smokers, the women in this study, whose mean age was 64 years, were not in the main heavy consumers of tobacco or alcohol. However, female mortality from neoplasms of the tongue and larynx is increasing in this country, despite improved results from treatment, consistent with a cohort effect of the greatly increased consumption of both cigarettes and alcohol by women since the 1940s.

82–1617. Hiatt, R. A.; Friedman, G. D.; Bawol, R. D.; Ury, H. K. **Breast Cancer and Serum Cholesterol.** *Journal of the National Cancer Institute* 68(6): 885-889, June 1982.

Recent studies have suggested a role for dietary fat in the etiology of breast cancer. The relation of serum cholesterol and other serum lipid measures to breast cancer incidence was investigated in a cohort of 95,179 women who had multiphasic health checkups from 1964 through 1972. Possible confounding factors, such as smoking, reproductive history, exogenous estrogen use, demographic variables, alcohol consumption, previous medical history, height, and weight, were also analyzed. Age-adjusted incidence rates were 1.45, 1.37, 1.31, and 1.40/1,000 person-years from the lowest to the highest quartile of serum cholesterol level, respectively. Similarly, no statistically significant relation was detected between β-lipoprotein or total lipids and breast cancer. Incidence rates were not significantly higher in smokers than in nonsmokers, but were significantly higher in ex-smokers than in current smokers. The expected relation of breast cancer to established risk factors was confirmed by univariate analysis, and serum cholesterol and breast cancer were not associated after simultaneous consideration of nine other risk factors by multivariate analysis. These data suggest that the postulated causal relationship between dietary fat and breast cancer does not act via an effect on circulating lipid levels. (Auth. Abs. Mod.)

82-1618. Higenbottam, T.; Shipley, M. J.; Rose, G. **Cigarettes, Lung Cancer, and Coronary Heart Disease: The Effects of Inhalation and Tar Yield.** *Journal of Epidemiology and Community Health* 36(2): 113-117, June 1982.

Ten-year mortality rates for lung cancer and coronary heart disease have been related to cigarette smoking habits in 17,475 male civil servants (5,726 cigarette smokers) aged 40-64 and in a sample of 8,089 male British residents (3,707 cigarette smokers) aged 35-69. Both diseases were more frequent in smokers. Lung cancer rates were higher overall for "non-inhalers" smoking 25 or more cigarettes daily. Tar yield correlated with the risk of lung cancer in non-inhalers but less so in inhalers. Conversely, coronary deaths were more common among inhalers, and the effect of tar/nicotine yield (such as it was) was confined to inhalers. It appears that there are subtle interactions between the amount smoked, the tar/nicotine yield of the cigarette, and the style of smoking. Thus the effects of a change in cigarette characteristics are hard to predict, and they may be different for respiratory and cardiovascular disease. Greater caution may be necessary in advocating a switch to different tar and nicotine yields, with more research into its long-term effect on the absorption of smoke products. (Auth. Abs. Mod.)

82-1619. Higgins, I.; Welch, K.; Oh, M.; Bond, G.; Hurwitz, P. **Influence of Arsenic Exposure and Smoking on Lung Cancer Among Smelter Workers: A Pilot Study.** *American Journal of Industrial Medicine* 2: 33-41, 1981.

A pilot study of 300 smelter workers from the cohort reported by Lee and Fraumeni (1969) has been reviewed, using data from 150 men in their heavy exposure group and 150 men randomly selected from the rest of the cohort. Usable smoking habits were obtained from 86 percent of the sample. Estimates of the total lifetime work exposure were made from available measurements. A very high risk of lung cancer was observed among the most heavily exposed workers. Smokers had a slightly higher mortality than nonsmokers in the heavy exposure group, and there was also a higher proportion of smokers (85.3 percent vs. 80.0 percent) in the randomly selected cohort. Excess risks, though still present, were lower in those with lower exposures. This indicates that there may be some interaction between smoking and arsenic exposure. Moreover, comparison of smoking habits reported by smelter workers suggested that, as a group, they smoked more heavily than average for the U.S. population. A small study of proxy smoking histories confirmed the questionnaire data to be valid and unbiased. The work continues on an enlarged sample. (Auth. Abs. Mod.)

82-1620. Hilt, B.; Rosenberg, J.; Langard, S. **Occurrence of Cancer in a Small Cohort of Asbestos-Exposed Workers.** *Scandinavian Journal of Work, Environment and Health* 7(3): 185-189, September 1981.

Fatal cancers were observed only among smokers in a cohort of 20 men who had been occupationally exposed to asbestos dust 50 years earlier. The men, born between 1910 and 1914, were heavily exposed to asbestos dust for 1 year (1928-1929) while working on the construction of a saltpeter plant. During the observation period, January 1, 1943, through June 1980, three deaths occurred from lung cancer and two from mesothelioma. The smoking members (n=16) of the cohort had a cancer (all sites) mortality of 43 percent before the age of 65, compared with no cancer deaths among the nonsmokers (n=4), who included two ex-smokers. No differences in the prevalence of asbestos-related radiographic changes were found between smokers and nonsmokers.

82-1621. Husum, B.; Wulf, H. C.; Niebuhr, E. **Sister-Chromatid Exchanges in Lymphocytes in Women With Cancer of the Breast.** *Mutation Research* 85(5): 357-362, October 1981.

A statistically significant positive correlation was found between cigarette smoking and sister-chromatid exchanges (SCE) in lymphocytes of 131 women, aged 17 to 90 years, with tumors in the breast. A venous blood sample was drawn from each woman prior to a peroperative histological examination performed to determine the character of the tumor. Fifty-two patients with peroperatively demonstrated carcinoma of the breast had 9.39±0.17 SCE/cell and the remaining 79 women with nonmalignant fibroadenomatosis had 9.88±0.18 SCE/cell. Multiple regression analysis indicated that the character of the tumor, the patient's age, hormone treatment, and preoperative examination by mammography all were without significant influence on the SCE rate. However, the fact that the 45 cigarette-smoking patients had 10.49±0.23 SCE/cell compared with 9.26±0.13 SCE/cell in the 86 nonsmokers demonstrates a positive correlation between SCE and cigarette smoking. It is concluded that spontaneous SCE in lymphocytes is not an indicator of carcinoma of the breast. (Auth. Abs. Mod.)

82-1622. Kato, H.; Iizuka, T.; Watanabe, H.; Hirata, K.; Saito, T.; Hirashima, T.; Itabashi, M.; Hirota, T. **Esophageal Cancer Associated With Gastric Cancer.** *Japanese Journal of Clinical Oncology* 11(2): 315-320, August 1981.

Smoking was a common habit among cancer patients in Japan who had esophageal cancer associated with gastric cancer. Of 1,137 patients with esophageal cancer admitted to the National Cancer Center Hospital in Tokyo, there were 44 cases (42 men, 2 women) of esophageal cancer associated with gastric cancer, an incidence of 3.9 percent. The majority of the patients were between 60 and 70 years old. Eleven of these 44 patients had a third cancer. Six had multiple cancers in the esophagus and/or stomach. Eighteen patients had early gastric cancer. Thirty-two of the cancers were synchronous and 12 were metachronous. Of these 44 patients, 21 had family histories of cancer, 37 were smokers (30 of whom had smoked over 10 cigarettes daily for over 20 years), and 36 were drinkers. Thirty-two patients (72.7 percent) had both smoking and drinking habits. (Auth. Abs. Mod.)

82–1623. Kilburn, K. H. **Lung Cancer—Time for a New Strategy.** *Urban Health* 10: 15, 46-47, July-August 1981.

More effective strategies are needed for the prevention and treatment of lung cancer which has an incidence in the United States of over 100,000 cases/year and a survival rate unchanged in 20 years. Cigarette smoking is accepted as the principal causal factor with occupational factors, especially those involving asbestos and uranium, producing a multiplicative effect. A program based on 5,000 high-risk individuals selected on the basis of risk factors from an overall population of 25,000 to 50,000 is proposed. These risk factors should be (1) 15 years or more since initial asbestos exposure, (2) continuing cigarette smoking or its recent cessation, and (3) age 45 years or greater. If this strategy detects more small cancers and if they respond to treatment so that survival is lengthened, then the strategy could be applied to other industrially exposed smokers or ex-smokers who have quit within 5 to 8 years. The problems and assets associated with various therapies, chemoprevention techniques (such as beta carotene, vitamin A, vitamin C, or selenium), and detection techniques (such as sputum studies and chest X-rays) are explored. Additionally, computed cost benefits as well as benefits to be accrued from the opportunity to evaluate various treatments are discussed.

82–1624. Kjuus, H.; Lislerud, A.; Lyngdal, P. T.; Omland, H.; Stave, O.; Langard, S. **Cancer and Polluted Work Places: A Case-Control Study.** *International Archives of Occupational and Environmental Health* 49: 281-292, 1982.

The possible interaction of smoking and occupational exposure, particularly in relation to lung cancer, is touched upon in an examination of the possible association between selected cancers and polluted workplaces in a hospital-based, case—control study. By dividing all jobs in the participants' working career into "polluted" and "clean," a crude measure for the total industrial exposure a worker experiences throughout his life was established. Among 103 age-matched, case—control pairs the overall estimated relative risk (RR) for exposed subjects (≥ 10 years in a polluted workplace) of developing cancer compared to nonexposed (< 10 years in a polluted workplace) was 1.1. The only subgroup where a significant difference was found between the cases and the controls was the lung cancer subgroup (RR = 4.0). When the 30 lung cancer cases were compared to an alternative control group consisting of 60 subjects matched for age and smoking habits, an estimated RR of 4.5 was found. A moderate, but not significant, association between lung cancer and definite asbestos exposure was also found (RR = 2.3). Of the lung cancer cases, 25 were considered "heavy smokers" and 5 "light smokers." A high proportion of male lung cancers appearing in the industrial area might be related to the combined influence of smoking and industrial agents. As most workers are exposed to a variety of industrial agents throughout their working careers, further development of methods for characterizing combined exposures are needed, both for retrospective and prospective purposes. (Auth. Abs. Mod.)

82–1625. Konno, T.; Sekiguchi, T.; Mashimo, N. **I Shudan Kenshin ni Okeru Ishikkan Kokikendogun no Kento. [A Study on the High Risk Group for Gastric Cancer in Mass Survey.]** *Kitakanto Medical Journal* 31(1): 41-53, 1981, Japanese.

Smoking habits, sex, age, past medical history, irregular eating habits, and general stomach complaints appeared to be useful factors in screening for stomach cancer. Age, sex, cancer and gastric ulcer heredity in the family, food habits, kinds of meals, condition of teeth, smoking habits, drinking of liquor, and complaints of the subjects were analyzed using cards submitted in a gastric mass survey. The subjects consisted of 700 normal persons, 690 patients with gastritis, 441 with gastric ulcer, and 268 with gastric cancer diagnosed by endoscopic examination. High risks were associated with female sex, smoking, poor dental condition, and irregular eating habits. Age was also a high relative risk factor. Epigastralgia, heartburn, fullness, and discomfort were the complaints associated with gastric ulcer, and epigastralgia was associated with stomach cancer. (Auth. Abs. Mod.)

82–1626. Kulcar, Z.; Strnad, M.; Stoiljkovic, C.; Hrabak-Zerjavic, V. **"Pusacki Rak" Zena u SR Hrvatskoj. [Female "Smoker's Cancer" in SR Croatia.]** *Lijecnicki Vjesnik* 103(8): 321-322, August 1981, Serbo-Croatian.

The 1969 to 1975 Croatian statistics show that the incidence of "smoker's cancer" in women younger than 59 increased during that period by 10.7 percent, while the incidence of all cancers during the same period decreased by 2.8 percent. At the same time, the incidence of gynecologic cancer in the same category of women decreased by 6.1 percent, while that of breast cancer increased by 7.0 percent. The male population of Croatia is still at a greater risk of the "smoker's cancer" than the female population, since the incidence of this cancer in men younger than 59 increased by 31.6 percent over the same period. Consideration of new measures for "smoker's cancer" prevention, particularly in women, is suggested.

82–1627. Lynch, H. T.; Fain, P. R.; Albano, W. A.; Ruma, T. A.; Black, L.; Lynch, J.; Shonka, M. **Genetic/Epidemiological Findings in a Study of Smoking-Associated Tumors.** *Cancer Genetics and Cytogenetics* 6(2): 163-169, June 1982.

Smoking histories were obtained for relatives of an overlapping series of patients with smoking-associated tumor sites. Particular attention was given to carcinoma of the lung, oral cavity, esophagus, urinary bladder, and pancreas. Findings revealed that, although a significant cohort effect was observed with respect to smoking habits for both relatives of lung cancer probands and for relatives of probands with other smoking-associated tumors, a corresponding trend for lung cancer frequency was observed only for relatives of lung cancer probands. This result suggests the importance of host factors in combination with environmental exposures in determining lung risk. A cohort trend for lung cancer was also apparent among relatives of breast cancer probands, but not for relatives of colon cancer probands, suggesting the possibility of an intrinsic association between carcinomas of the breast and lung. It is suggested that further elucidation of host factor susceptibility in lung cancer may have important etiological and preventive implications. (Auth. Abs. Mod.)

82–1628. Martell, E. A.; Cohen, J. I.; Cohen, B. S.; Harley, N. H.; Hill, C. R.; Wagner, W. L.; Ravenholt, R. T.; Hoffmann, D.; Wynder, E. L.; Di Franza, J. R.; Winters, T. H. **Radioactivity in Cigarette Smoke. (Letters).** *New England Journal of Medicine* 307(5): 309-313, July 29, 1982.

Eight responses concerning the carcinogenicity of polonium-210 (^{210}Po) in cigarette smoke were prompted by a letter in a previous issue (*New England Journal of Medicine* 306(6): 364-365, February 11, 1982). Comments are as follows. ^{210}Po is soluble and rapidly cleared, and the presence of lead-210 (a precursor) at lung bifurcations indicates the source of polonium is the persistence of insoluble, lead-enriched particles deposited at the bifurcations. Lower yield cigarettes and filters have no protective effect against ^{210}Po exposure; thus those who smoke more low-yield cigarettes may be at greater risk than smokers of fewer higher yield cigarettes. ^{210}Po accounts for 99 percent of the alpha activity of cigarette smoke. Nuclear-track-etch film technique reveals alpha activity equaling annual doses of 2.0 to 20,000 mrem in various areas of smokers' bronchial epithelium. However, it is noted that the concept of deposition of particles in "hot spots" may be unfounded: a prior study found no evidence of surface concentrations of alpha activity of more than a mean dose of 10 mrem per year. Conversely, dose—response studies revealed a reduction in dose—response ratios with decreasing doses. Thus ^{210}Po's carcinogenic nature must be due to high density ionizations along the track of alpha radiation. ^{210}Po is not limited to bronchial tissue: it is carried by systemic circulation throughout the body, causing cellular deviations and mutations, accelerated aging, and other diseases associated with radiation. Finally, it is pointed out that ^{210}Po is only one of a wide spectrum of carcinogens present in cigarette smoke, and may be cocarcinogenic or synergistic with compounds such as benzo(*a*)pyrene. See also 82-0691.

82–1629. Mason, M. K.; Jordan, J. W. **Outcome of Carcinoma in Situ and Early Invasive Carcinoma of the Bronchus.** *Thorax* 37(6): 453-456, June 1982.

Seventeen patients in whom a squamous carcinoma in situ of the bronchus had been resected were followed for up to 16 years. Except for five subjects whose smoking habits were not known, all patients were either cigarette smokers or pipe smokers. Cigarette consumption among the patients ranged from 20 to 50 cigarettes daily. Consumption for the pipe smokers ranged from 3 to 4 oz per week. The crude mortality rate for these patients is high: seven (41 percent) dead after 5 years and 14 (82 percent) after 10 years. Half the patients had died as a direct result of malignant disease by 10 years—because of recurrence, metastatic disease, or the development of a second tumor. A guarded long-term prognosis must be given in these patients owing to the many deaths (mainly from other respiratory diseases) besides those from recurrent cancer. The degree of any accompanying lung disease and the smoking habits of the patient must be considered when the doctor is trying to arrive at a prognosis, since these appear to be the most important factors in determining the outlook in patients who have a slowly growing tumor. (Auth. Abs. Mod.)

82–1630. Matthay, R. (Editor). **Symposium on Recent Advances in Lung Cancer.** *Clinics in Chest Medicine* 3(2): 217-454, May 1982.

Carcinoma of the lung is among the most prevalent and most aggressive types of cancer in humans. In the United States alone, it is responsible for more cancer-related deaths than any other tumor in men and soon will surpass breast carcinoma as the most fatal form of cancer in women. Since cigarette smoking—a major etiologic factor—continues without evidence of rapid decline, lung cancer is likely to remain prevalent and perhaps will continue to increase in incidence. The 16 articles of this symposium provide an up-to-date review of lung cancer, emphasizing etiology, epidemiology, pathology, diagnosis, staging, and treatment. Topics include: (1) environmental and occupational factors which contribute to lung cancer incidence; (2) the natural history, detection, and rate of tumor growth and prognostic factors in resectable and nonresectable disease; (3) a review of the Philadelphia Pulmonary Neoplasm Research Project; (4) pathologic features and etiological theories of the major neoplasms (squamous cell, small cell, large cell, and adenocarcinoma) and also giant cell carcinoma, combined lung carcinomas, second primary lung carcinomas, and mesothelioma; (5) a critical evaluation of lung cancer staging; (6) a discussion of proteins currently being evaluated as potential "markers" of tumor presence; (7) a review of ongoing studies of screening techniques for early diagnosis, including flexible fiberoptic bronchoscopy and percutaneous needle biopsy; (8) potential use of computerized tomography; and (9) a role for mediastinoscopy and mediastinotomy in diagnosing and staging lung tumors. The final seven articles update therapy, and discuss diagnostic probabilities and management of solitary and multiple lung nodules; the preoperative evaluation and surgical procedures for lung cancers, including resection of localized small cell carcinoma; chemotherapy for small cell carcinoma; radiotherapy for nonsmall cell carcinoma; chemotherapy for inoperable nonsmall cell cancer; immunoincompetence and enhancement of antitumor response by immunotherapy; and diagnosis and appropriate management of malignant pleural effusions.

82–1631. Mausner, J. S. **Epidemiology of Cancer and Implications for Management.** *Urban Health* 10(8): 26-28, 47-48, October 1981.

Through epidemiologic investigations, substantial gains have been made in identifying factors associated with increased incidence of various cancers. Basically, there are two complexes of factors—host susceptibility, and environmental exposures such as cigarette smoking, which are potentially changeable. Though the contributions of different factors to overall cancer occurrence can only be estimated, it is still useful to attempt quantification based on information about occurrence patterns (male/female ratios, relative risk for occupationally exposed and unexposed persons, differences in rates between smokers and nonsmokers, etc.). According to these estimates, tobacco use accounts for about one-third of male cancers with multiple environmental contributors such as combined use of alcohol and tobacco and occupational cofactors complicating the issue. In the case of excess risk, distribution is site-specific rather than throughout all cancer sites for a particular carcinogen; cigarette smoke enhances the risk of multiple as well as single tumors of the mouth, tongue, pharynx, larynx, esophagus, and lung. Processes which determine disease occurrence may span a period of years during which the host appears healthy. Though this latent period may constitute a serious problem in the conduct of epidemiologic studies, it can serve a useful function by providing an interval for preventive activities. In conclusion, physicians need to be alert to the need for early detection and intervention, thus playing a major role in cancer reduction.

82–1632. McGlashan, N. D.; Bradshaw, E.; Harington, J. S. **Cancer of the Oesophagus and the Use of Tobacco and Alcoholic Beverages in Transkei, 1975-6.** *International Journal of Cancer* 29: 249-256, 1982.

Results of a questionnaire-based 2-year survey among young and old, males and females in three regions of Transkei, South Africa, show that smoking and alcohol drinking are indulged in by 58 percent of all Transkei men and 10 percent of the women. The regions were selected on the basis of having proved contrasts of incidence experience from esophageal cancer. Significant gradients of social fabric and of customary usage of tobacco and alcohol were found across the three incidence regions. In particular, the concept of a synergistic relationship between tobacco and alcohol is supported when both are used by one individual, with the major role being played by tobacco, especially when smoked in pipes. Pipe smoking raises the risk of esophageal cancer more than 20-fold. Among women the smaller numbers who smoke make the picture less clear, although cigarette smoking evidently increases relative risk. (Auth. Abs. Mod.)

82–1633. Morrison, A. S.; Verhoek, W. G.; Leck, I.; Aoki, K.; Ohno, Y.; Obata, K. **Artificial Sweeteners and Bladder Cancer in Manchester, U.K., and Nagoya, Japan.** *British Journal of Cancer* 45(3): 332-336, March 1982.

In an evaluation of the relationship between cancer of the lower urinary tract (bladder cancer) and the use of artificial sweeteners in Manchester, England, and Nagoya, Japan, cigarette smoking was found to be associated with the risk of bladder cancer. Case—control studies were conducted in both cities where extensive use of artificial sweeteners, primarily saccharin, occurred 30-40 years ago. Patients with cancer of the lower urinary tract (555 in Manchester, 293 in Nagoya) and controls chosen from the general population (735 in Manchester, 589 in Nagoya) were interviewed. Analysis of data on the use of sugar substitutes according to cigarette smoking history (current smoker, ex-smoker, nonsmoker) shows that in Manchester the relative risk associated with using sugar substitutes was highest in nonsmokers of both sexes, but the increases were small. In Nagoya the relative risks were <1 in all smoking categories. Results of the study suggest that use of artificial sweeteners confers little or no risk of bladder cancer.

82–1634. Newhouse, M. **Epidemiology of Asbestos-Related Tumors.** *Seminars in Oncology* 8(3): 250-257, September 1981.

The investigation of asbestos-related tumors presents problems in that there is a considerable delay between exposure and onset of disease. The presence of smokers in study populations and misclassification of neoplasms have further confounded epidemiological studies. Among 79 asbestos workers who did not smoke cigarettes (39 were pipe or cigar smokers), no deaths from lung cancer were observed over a 10-year period: 0.05 to 0.13 were expected. Among 283 cigarette smokers, however, 24 deaths were observed where only 3.1 were expected. This strongly suggests a synergistic rather than an additive effect of smoking and asbestos exposure, and was particularly evident in females. A study of U.S. insulators and steam fitters revealed a 2:1 excess of deaths from cancer of the larynx. However, this was not found in Canadian crysotile miners. As smoking and ethanol are also strong etiologic factors, a multitude of synergistic elements may interact to produce laryngeal neoplasms. It remains to be proven whether asbestos is a carcinogen, cocarcinogen, or a potentiator of other carcinogens such as cigarette smoke.

82–1635. Pezzuoli, G.; Roviaro, G.; Varoli, F.; Maruotti, R. A.; Zannini, P.; Sartori, F.; Calabro, F. **Clinical and Immunological Study of 150 Cases of Adenocarcinoma of the Lung.** *International Surgery* 66(3): 221-226, July-September 1981.

A smaller percentage of smokers (84 percent) was found in patients with lung adenocarcinoma than in those with other types of lung cancer, in a study of 1,287 patients (smokers and nonsmokers), 919 with histologically diagnosed cancers. Other histological types examined were squamous cell (epidermoid)—91.0 percent smokers; undifferentiated small oat-cell—89.6 percent smokers; and undifferentiated large cell—93.1 percent smokers. The peak incidence of lung cancer was in those patients who had smoked more than 20 cigarettes/day for more than 25 years. The differences between adenocarcinoma and other types of lung cancers as to location, association with other pulmonary diseases, operability, and survival rates are also discussed. Although cigarette dose—response curves have been made for lung cancer in general, this may not be so with adenocarcinoma: the high incidence of nonsmokers as compared to other histotypes suggests the presence of other carcinogens responsible for adenocarcinoma development.

82-1636. Rang, E. H.; Acheson, E. D. **Cancer in Furniture Workers.** *International Journal of Epidemiology* 10(3): 253-261, September 1981.

A followup study of 5,371 men who had worked in one or more of nine furniture factories in Buckinghamshire for an average of 19 years up to 1968 is reported. The incidence of nasal adenocarcinoma in furniture workers taken as a whole was found to be about 100 times that expected in the local population, and a significant relationship was found between increasing incidence of the tumor and increasing dustiness of work within the cohort. Similar comparisons with the local population produced no evidence for an increased risk of cancer of any other site in the furniture workers, including bronchial cancer and malignant disease of the reticulo endothelial system. However, when comparisons were made between men exposed to different amounts of dust within the industry, the incidence and mortality of bronchial cancer increased with increasing dustiness of work, the latter trend but not the former being statistically significant. No significant differences between the smoking habits of the men in jobs of various degrees of dustiness were found. There were slightly fewer current smokers and more ex-smokers among the furniture workers as a whole than among men in southeastern England. The slightly lower current cigarette smoking incidence among furniture workers than among other men may make a small contribution to their more favorable general mortality experience and their lower mortality from bronchial cancer; however, differences in smoking habits cannot account for the trend of increasing incidence and mortality from bronchial cancer seen with increasing dustiness of work. (Auth. Abs. Mod.)

82-1637. Rios-Dalenz, J.; Correa, P.; Haenszel, W. **Morbidity From Cancer in La Paz, Bolivia.** *International Journal of Cancer* 28(3): 307-314, September 15, 1981.

Data from a population-based Cancer Registry in La Paz, Bolivia, indicate a low cancer frequency, especially among males. La Paz is an epidemiologically unique population because of its high altitude and because its Aymara Indian background provides genetic and cultural characteristics different from most populations for which cancer data are available. Incidence rates in females are high for cancer of the cervix, the gallbladder, and the thyroid gland. Males display unusually high rates of testicular cancer. Stomach cancer rates are lower than among other Andean populations. Cancer of the nasal cavity and paranasal sinuses is unusually high in frequency. All smoking-related sites have low frequency in La Paz, probably indicating less exposure to smoke than most populations. Chewing of coca leaves is a frequent custom which may to some extent replace the smoking habit and improve oral hygiene by mechanical friction and by increased salivation. (Auth. Abs. Mod.)

82-1638. Selikoff, I. J. **Carcinogenic Risk Management in the United States.** *Annals of the New York Academy of Sciences* 363: 283-293, 1981.

In a review of the problems involved in management of carcinogenic substances, multiple risks such as industrial exposure combined with cigarette smoking are discussed. Cancer trends such as multiple factor interactions and dosage relationships add urgency to the matter of policy development. An illustration is given in the asbestos industry: among 87 men who had never smoked cigarettes, fewer than one death from lung cancer was expected, and none occurred. Of the 283 cigarette smokers in the group, 3 deaths were expected and 24 occurred. Other studies, both human and animal, have confirmed the existence of a much greater increase in cancer incidence where two or more agents are interacting. A study which followed one million Americans from 1960 confirmed not only the carcinogenicity of cigarette smoke, but also the presence of a dose—response relationship, and the absence of a threshold. The management of substances carcinogenic to humans is further discussed with respect to their end product use, latency of associated cancers, the absence of threshold dosages, the dose—induction period, possible reversal of risk, mutagenicity vs. carcinogenicity, their reproductive effects, the occurrence of noncarcinogenic disease, problems of control vs. banning, extrapolation of animal data to man, and examples from the past.

82-1639. Severson, R. K.; Davis, S.; Polissar, L. **Smoking, Coffee, and Cancer of the Pancreas. (Letter).** *British Medical Journal* 285(6336): 214, July 17, 1982.

That the association between coffee consumption and pancreatic cancer is confounded by factors such as cigarette smoking and occupational exposures was the conclusion of a recent review (*British Medical Journal* 284(6313): 400-402, February 6, 1982). In a response, it is further noted that in an analysis of 22 cases of pancreatic cancer and 485 population controls, a significant dose—response relationship for smoking was evident when the data were adjusted for age, sex, and coffee drinking. Furthermore, no excess risk from coffee consumption was found after controlling for smoking habits. Although based on a small number of cases, the estimate of risk from smoking is higher than previously reported, and additional studies are suggested. See also 82-0662.

82-1640. Shively, J. E.; Spayth, V.; Chang, F.-F.; Metter, G. E.; Klein, L.; Presant, C. A.; Todd, C. W. **Serum Levels of Carcinoembryonic Antigen and a Tumor-Extracted Carcinoembryonic Antigen-Related Antigen in Cancer Patients.** *Cancer Research* 42(6): 2506-2513, June 1982.

Levels of carcinoembryonic antigen (CEA) and a tumor-extracted CEA-related antigen (TEX) were determined in 331 patients with cancers of the breast, colon, lung, head and neck, and a number of miscellaneous categories, and from serum samples of 100 healthy controls (59 nonsmokers, 41 smokers).

Based on the cutoff values established for control nonsmokers, 5 percent of the control smokers showed elevation in the level of CEA and 29 percent in TEX, suggesting that smoking history does not necessarily increase the frequency of CEA elevation but does increase that for TEX. In analyzing data for breast and colon cancer patients, the rise in the mean TEX values in smokers was not comparable to that seen in the controls. For head and neck cancer and lung cancer, virtually all patients were smokers; thus, a statistically significant conclusion could not be reached.

82–1641. Silber, W. **Advances in Carcinoma of the Oesophagus.** *South African Cancer Bulletin* 25(1/2): 24-30, January-June 1981.

Various national and international surveys, undertaken to establish common factors in esophageal cancer epidemiology, are discussed as are genetic and environmental influences in the etiology of the disease. Risk factors universally associated with esophageal cancer are the excessive use of alcohol and tobacco; however, the association does not yet account for various epidemiological features encountered. The remarkable mutagenicity of substances obtained from Transkei home-grown tobacco pyrolosate which are ingested, and the five-fold gradient of incidence of the disease within the Transkei, seem to correlate with a higher smoking incidence in the high-risk group and a poor soil occurrence in the region with crops low in nitrogen, potassium, and manganese; and a high magnesium and low potassium-manganese ratio as well as a molybdenum deficiency. The possibility of fungi and mycotoxin contamination of the maize is discussed. A randomized trial to investigate a β-carotene intake deficiency is currently underway in this region to confirm an etiological linkage. Diagnostic and therapeutic techniques are also described.

82–1642. te Velde, E. R.; Persijn, J. P.; Ballieux, R. E.; Faber, J. **Carcinoembryonic Antigen Serum Levels in Patients With Squamous Cell Carcinoma of the Uterine Cervix: Clinical Significance.** *Cancer* 49(9): 1866-1873, May 1, 1982.

In 114 patients with invasive cervical cancer of the squamous cell type, pretreatment carcinoembryonic antigen (CEA) levels were determined. Since cigarette smoking and age may influence CEA levels, these factors were also investigated by determining the age-dependent upper limit of normal for CEA serum levels in heavy smokers ($n=23$), moderate smokers ($n=33$), and nonsmokers ($n=58$). The data obtained showed that considerable differences existed with regard to both factors. After adjusting for these confounding factors, an individual upper limit of the normal range was derived. Pretreatment CEA levels exceeding the upper limit of normal concurred with a very poor prognosis, regardless of the stage of the tumor. The clinical value and the possible therapeutic consequences of the findings presented are discussed in relation to the available knowledge of tumor growth in cervical cancer. (Auth. Abs. Mod.)

82–1643. Thomas, C. B.; Duszynski, K. R.; Shaffer, J. W. **Cholesterol Levels in Young Adulthood and Subsequent Cancer: A Preliminary Note.** *Johns Hopkins Medical Journal* 150(3): 89-94, March 1982.

Mean serum cholesterol levels of young male doctors who subsequently developed some form of major cancer were not significantly different from those of men who did not develop the disease, regardless of whether or not these levels were age-adjusted. When the cancer and noncancer groups of this cohort of 1,337 subjects were compared on smoking habits in medical school, the mean amounts were between 5 and 10 cigarettes a day for both groups and were not significantly different. Because of current interest in the relationship of lifetime smoking habits to cancer epidemiology, the amount smoked in the years preceding cancer diagnosis for 29 of the patients were surveyed. Overall, 12 (41.4 percent) of these men had never smoked; 8 (27.5 percent) had smoked lightly, moderately, or variably; and 9 (31.0 percent) had smoked heavily (20 cigarettes or more a day) for an average of 19.9 years (range, 14-29 years). Of the five men with lung cancer, three had smoked heavily, one was a variable smoker, and one had never smoked. Of the remaining 24 men in the other cancer site groups, 6 (25.0 percent) had smoked heavily; 7 (29.2 percent) had smoked lightly, moderately, or variably; and 11 (37.9 percent) had never smoked. The numbers are small, but they suggest that for most of these men smoking had not played an important role in the development of cancer by early middle life. Implications of a two-population hypothesis are also discussed wherein both populations have overlapping cholesterols but the characteristics of the populations are different. The findings of heterogenous regressions of cholesterol levels on age for the cancer and noncancer groups tend to support this view, and raise the possibility of differences in susceptibility to cancer, to coronary heart disease, and to premature death. (Auth. Abs. Mod.)

82–1644. Walker, A. M.; Dreyer, N. A.; Friedlander, E.; Loughlin, J.; Rothman, K. J.; Kohn, H. I. **An Independent Analysis of the National Cancer Institute Study on Non-Nutritive Sweeteners and Bladder Cancer.** *American Journal of Public Health* 72(4): 376-381, April 1982.

In a reanalysis of data from the 1978 National Bladder Cancer Study conducted by the National Cancer Institute (NCI), it is concluded that cigarette consumption does not appear to interact with nonnutritive sweeteners (NNS) use, either by masking a small effect or by potentiating an effect which is otherwise minimal. The original NCI study, which involved obtaining information on 3,010 patients with urinary bladder cancer and over 5,000 controls, found no evidence of an association between NNS and bladder cancer. In reevaluating the original data, the relationship between relative risk for bladder cancer in various NNS exposure categories and cigarette smoking was examined by investigating the relative risk estimates, adjusted for design variables, for each level of NNS exposure at each of five levels of total cigarette consumption. Although cigarette consumption itself is a strong predictor of bladder cancer risk, there were no trends of relative risk over categories of cigarette use for any of the measures of NNS use. The general conclusions of the original study are reaffirmed; however, findings relative to two subgroups, nonsmoking white females and heavily smoking white males, did not agree. The possibility that these two subgroups might be at increased risk for bladder cancer if they used moderately large amounts of NNS was not confirmed. (Auth. Abs. Mod.)

82-1645. Weiss, W.; Boucot, K. R.; Seidman, H. **The Philadelphia Pulmonary Neoplasm Research Project.** *Clinics in Chest Medicine* 3(2): 243-256, May 1982.

Results of the Philadelphia Pulmonary Neoplasm Research Project (PNRP) conducted between 1951 and 1965 are summarized. Over a period of 4 years, 1951 to 1955, 6,027 men aged 45 or over were enrolled in PNRP. Each man was screened every 6 months for 10 years with chest photoflurograms, and questioned about respiratory symptoms, smoking habit, and weight change. Sixty-nine percent of the men were current smokers and 17 percent were ex-smokers. During the 10-year period, 121 men (2 percent) developed lung cancer, all of whom were or had previously been regular smokers. Smokers of pipes or cigars had an incidence rate of only 0.4 per 1,000 man-years, whereas cigarette smokers had a rate of 3.3. In the latter group the rate was 1.3 for ex-smokers and 3.8 for current smokers as defined at the start of observation. Among current smokers of cigarettes there was an increasing incidence with increasing daily consumption, the rate reaching 10.0 per 1,000 man-years (seven cases) among 103 men who had smoked two or more packs per day. Duration of smoking was also an important risk factor. All three major histologic types of lung cancer (squamous cell, small cell, and adenocarcinoma) showed a dose—response relationship to cigarette smoking. Other risk factors such as race, occupation, and degree of compliance with protocol, along with early manifestations of lung cancer and survival factors, are also discussed. (Auth. Abs. Mod.)

82-1646. WHO Advisory Committee on Medical Research. **Report on the First Session.** Geneva, World Health Organization, Advisory Committee on Medical Research, Subcommittee on Cancer, ACMR/CAN. 1/81 Report, 1981, 13 pp.

The global cancer problem is discussed and several recommendations are made, including suggested actions for preventing cancers by discouraging tobacco consumption. Issues addressed include primary prevention, early detection, therapeutic investigations, and relieving pain. Primary prevention chiefly involves cancers of the lung, mouth, cervix, and liver, which account for a large proportion of cancer cases on a global scale, and have reliably known preventable factors. Approximately 80-90 percent of lung cancer cases in developed nations are due to tobacco, mainly from smoking manufactured cigarettes. It is expected that lung cancer will be a worsening problem in the developing nations if present increases in cigarette use continue. Cancers of the mouth, pharynx, larynx, esophagus, and bladder are also related to smoking, as are bronchitis, emphysema, ischemic heart disease, and other vascular diseases. Three recommendations given concerning tobacco smoking are: preventing advertising of cigarette brands that have tar levels of 10 mg or higher, taxation on cigarettes that greatly raises their real cost, and government responsibility for informing the public of the quantitative dangers of smoking. Oral cancer is a great problem in India, Sri Lanka, and adjacent nations, and is associated with quid chewing. Primary prevention could include attempts to improve oral hygiene and to inform people of the dangers of chewing. Such attempts to discourage smoking and chewing may be difficult, but should be tried.

82-1647. Winkler, D. G.; Baky, A. A.; Hunter, N. R.; Greenberg, S. D.; Rogers, T. D.; Spjut, H. J.; Estrada, R. **Image Analysis of Atypical Bronchial Squamous Epithelial Cells. A Quantitative and Qualitative Examination of Squamous Cell Carcinogenesis.** *Analytical and Quantitative Cytology* 3(4): 295-298, December 1981.

An objective cell image analysis method for examining and monitoring atypical bronchial squamous epithelial cells was used among a study group of smokers. Sputa specimens were acquired from cigarette smokers, with and without diagnosed lung cancer. Their ages ranged between 20 and 60. A new system known as the atypia status index (ASI) was used. This dual-purpose system specifically classifies cells into five distinct classes of progressive atypia as well as quantitates the degree of atypicality of each individual cell. The ASI range has well-defined intervals, representing the five different atypia classes used in the study (squamous metaplasia, mild atypia, moderate atypia, severe atypia, and carcinoma). The distribution of cellular ASI values was arranged from lowest to highest and plotted for each subject as a profile. Subjects at different stages of carcinogenesis were found to have significantly different profiles. Thus objective methods for evaluating cell changes are crucial in detecting potentially severe diseases at an early stage. (Auth. Abs. Mod.)

82-1648. Wynder, E. L. **The Etiology, Epidemiology, and Prevention of Lung Cancer.** *Seminars in Respiratory Medicine* 3(3): 135-139, January 1982.

Prevention of smoking in children, cessation of smoking in the adult population, and the further development of less harmful cigarettes should be the strategy in the war against lung cancer. Though there is no longer any doubt as to the relationship between cigarette smoking and lung cancer, particularly of the Kreyberg I and Kreyberg II variety, there still remains some uncertainty concerning lung cancer and passive smoking. Although two recent studies have suggested an association, the fact that heavy cigar and pipe smokers who breathe in their own smoke have a low risk, as do bartenders and waiters who work in smoke-filled rooms, casts some doubt on a relationship. An example of smoking prevention is the American Health Foundation's "Know Your Body" child health education program. The intent of the program is to create a greater awareness and respect of young people for their bodies and to increase self-esteem through increased health knowledge. In the development and improvement of the "less harmful" cigarette, vigilance with regard to additives placed in cigarettes to make up for taste reduction is recommended to ensure that the tumorigenic burden of tobacco smoke is not increased.

See also, **82–1594, 82–1649, 82–1671, 82–1792**

NON—NEOPLASTIC RESPIRATORY DISEASES

82–1649. Artvinli, M.; Baris, Y. I. **Environmental Fiber-Induced Pleuro-Pulmonary Diseases in an Anatolian Village: An Epidemiologic Study.** *Archives of Environmental Health* 37(3): 177-181, May-June 1982.

Pleuro-pulmonary radiological findings from 312 residents of the Turkish village of Tuzkoy revealed that all abnormalities, with the exception of diffuse interstitial pulmonary fibrosis, were more common in smokers than in nonsmokers. The prevalence of pleural mesothelioma and other malignancies in the group was compared with 95 residents of another village, and X-rays revealed no malignancies in the control group, while the Tuzkoy group had various abnormalities. The number of cigarettes consumed and the duration of smoking were similar in the two groups. The presence of zeolite, an asbestiform mineral, was determined to be responsible for the pleuro-pulmonary diseases identified in Tuzkoy.

82–1650. Bauer, L. A.; Bauer, S. P.; Blouin, R. A. **The Effect of Acute and Chronic Renal Failure on Theophylline Clearance.** *Journal of Clinical Pharmacology* 22(1): 65-68, January 1982.

Theophylline total body clearance was measured during a continuous intravenous infusion in 29 anuric, chronic obstructive pulmonary disease patients with acute or chronic renal failure. They were divided into two groups depending on the absence (group 1, $n=16$) or presence (group 2, $n=13$) of congestive heart failure and compared to normal renal function control patients with similar disease states. All study and control patients smoked cigarettes (18 to 25 cigarettes/day in group 1 and 15 to 20 cigarettes/day in group 2). The theophylline mean total body clearance values (± S.D.) for group 1 were 68.0 ± 14.6, 64.5 ± 12.9, and 62.6 ± 17.3 ml/kg/hr for acute renal failure patients, uremic chronic renal failure patients, and control patients, respectively. For group 2, the corresponding values were 25.6 ± 6.1, 28.6 ± 8.7, and 27.4 ± 12.9 ml/kg/hr. Neither acute renal failure nor uremia significantly changed theophylline clearance in cigarette-smoking patients with or without congestive heart failure when compared to control patients with normal renal function ($p > 0.05$, one-way analysis of variance). Since total body clearance determines the steady-state concentration of a drug after repeated administration, theophylline doses do not need to be reduced in patients with acute renal failure or uremia. (Auth. Abs. Mod.)

82–1651. Begin, R.; Masse, S.; Cantin, A.; Menard, H. A.; Bureau, M. A. **Airway Disease in a Subset of Nonsmoking Rheumatoid Patients.** *American Journal of Medicine* 72(5): 743-750, May 1982.

The site and time course of airflow obstruction was determined in six lifetime nonsmokers, who did not have bronchiectasis, and insight was gained into the pathogenesis of chronic airway disease in this subset of patients with rheumatoid disease. Clinical and histopathologic evidence of the Sjogren autoimmune exocrinopathy was documented in five of the patients, and the sixth patient had lymphoplasmocytic infiltrates of the labial glands without obstruction of the lumen or destruction. By pulmonary function tests and histopathologic examination of four open lung biopsies, the airway disease was found to be located predominantly in the peripheral airways of the lung. On each biopsy, the lesions were in different stages of activity, but on all specimens there was a definite predilection for selective bronchiolar injury. Early stage lesions were characterized by mononuclear cell infiltrates of the peribronchiolar tissue which led to deformation of airway lumen, focal mucosal extension, and ulceration. Subsequently, the inflammatory reaction was replaced by fibroblastic proliferation, and in the end stage of the disease, there was complete obliteration of many bronchioles by collagenized fibroblastic tissue. From regression analyses of serial pulmonary function tests of these patients, it was concluded that (1) the airway disease in nonsmokers progressed inevitably but not uniformly and (2) deterioration of pulmonary functions was more rapid in these patients than it was in the cigarette smokers who had chronic obstructive lung disease. (FVC_1 of 9 percent FVC per year percent decrease than the established progression of the airway disease in cigarette smokers, FEV_1 of 2 percent FVC per year.) Major dysfunctions of the chest wall mechanics which appear to contribute to the restriction of lung volumes in some rheumatoid patients are also documented. (Auth. Abs. Mod.)

82–1652. Carrell, R. W.; Jeppsson, J.-O.; Laurell, C.-B.; Brennan, S. O.; Owen, M. C.; Vaughan, L.; Boswell, D. R.

Structure and Variation of Human α_1-Antitrypsin. *Nature* 298(5872): 329-334, July 22-28, 1982.

Abnormal variants of α_1-antitrypsin (α_1-AT) help explain cigarette smoking's accelerative effect in the development of emphysema. Although α_1-AT deficiency may be compatible with a full lifespan in a healthy environment, with heavy smoking it may result in crippling lung disease and early death. Smoking is the most common cause of emphysema: smoke exposure causes a large increase in phagocytic cells (pulmonary alveolar macrophages) in the lungs. An inactivation of inhibitor accompanies this increase, resulting in half the normal inhibitory capacity of α_1-AT. The subsequent attack on the elastase tissue of the lower lungs results in emphysema. Thus individuals with abnormal α_1-AT are at exceptionally high risk and should not smoke. In this review of the molecular pathology of α_1-AT, the primary structure of the protein is presented in its completed form, aligned with human antithrombin-III and chicken ovalbumin. Its functions, structural variations, and resulting diseases are also discussed.

82–1653. Clonfero, E.; Mastrangelo, G.; Cortese, M. S. **Cross Sectional Study on Chronic Bronchitis and Air Flow Obstruction in Three Italian Aluminum Smelting Plants.** *Medicina del Lavoro* 72(4): 301-305, July-August 1981.

Respiratory symptoms among Italian aluminum industry workers appear to be unrelated to occupational exposures. Smoking habits, age, and work exposures were evaluated by multiple regression analysis for effects on forced expiratory volume in 1 second (FEV_1) and chronic bronchitis symptoms. Mean age and pack-years respectively were 36.5 and 5.9 in controls, 40.6 and 10.3 for the Fusina workers, 43.2 and 12.3 in the Porto Marghera workers, and 46.7 and 16.8 among the Bolzano employees. After adjustment for age and smoking habits, there were no detectable effects on FEV_1 or chronic bronchitis symptoms due to occupational exposures among currently exposed workers. However, the past exposed subjects showed effects of length of exposure on lung function but not bronchitis symptoms. The mean FEV_1 values and chronic bronchitis were correlated to smoking habits and age.

82–1654. Cohen, A. B.; Chenoweth, D. E.; Hugli, T. E. **The Release of Elastase, Myeloperoxidase, and Lysozyme From Human Alveolar Macrophages.** *American Review of Respiratory Disease* 126(2): 241-247, August 1982.

Pulmonary alveolar macrophages (PAM) of cigarette smokers were found to secrete a factor capable of causing release of myeloperoxidase, lysozyme, and elastase from cytochalasin-B-treated neutrophils and from neutrophils in the absence of cytochalasin B. PAM secretions from smokers were harvested after 18 hours of incubation. The medium contained at least two acid-stable factors that could release enzymes from cytochalasin-B-treated human neutrophils. The study focused on the largest of these factors, which had an apparent mass ratio of 5,400 by gel filtration chromatography in 10 percent acetic acid. The high molecular weight (HMW) factor was partially degraded by trypsin. Chymotrypsin completely destroyed the factor, but human neutrophil elastase did not affect it. The factor is partially extractable into chloroform indicating that it is very hydrophobic and may contain a lipid. High concentrations of the HMW factor inhibited the release of lysozyme and myeloperoxidase. Because elastases can cause emphysema when introduced into alveoli of animals, the most important observation may be that the HMW factor was able to release elastase from human neutrophils attached to Millipore membranes in the absence of cytochalasin B. The enzyme-releasing factors may be identical to neutrophil chemotactic factors recently described by others. The contribution of the released elastase to the protease load in the lung may be augmented by the simultaneous release from neutrophils of myeloperoxidase, which can inactivate α_1-antitrypsin. The interaction between alveolar macrophages and neutrophils may have importance in the pathogenesis of emphysema. (Auth. Abs. Mod.)

82–1655. Cohen, A. B.; James, H. L. **Reduction of the Elastase Inhibitory Capacity of Alpha$_1$-Antitrypsin by Peroxides in Cigarette Smoke. An Analysis of Brands and Filters.** *American Review of Respiratory Disease* 126(1): 25-30, July 1982.

Results of experiments involving the effects of cigarette smoke samples on alpha$_1$-antitrypsin suggest ways in which smoke oxidants might damage respiratory health. A procedure for measuring the oxidant content of aqueous condensates of tobacco cigarette smoke is described. The procedure was used in conjunction with analysis of the ability of the smoke solutions to inactivate the elastase inhibitory capacity (EIC) of alpha$_1$-antitrypsin. The ability of the smoke of a brand to inactivate alpha$_1$-antitrypsin correlates well with the known tar and nicotine and with the amount of oxidants as measured using *o*-dianisidine. Filters were found to remove about 73 percent of the oxidants from smoke. Smoke from a commercial nontobacco cigarette was also found to contain a significant amount of oxidants and to also destroy alpha$_1$-antitrypsin. Catalase and superoxide dismutase reduce the effect of solutions containing smoke on the EIC of alpha$_1$-antitrypsin, suggesting that peroxides and superoxide anions in smoke contribute to the oxidant capacity of the smoke. The extent of apparent oxidation by a given quantity of smoke condensate increases for as long as an hour from the time the condensate is collected. The addition of hydrogen peroxide to the smoke solution increases both its oxidant content and its ability to inactivate alpha$_1$-antitrypsin. These data suggest that occurrence of hydrogen peroxide caused by secretion from macrophages found in the small airways of smokers may contribute to a locally damaging environment for alpha$_1$-antitrypsin in the presence of cigarette smoke that could promote the development of centrilobular emphysema. (Auth. Abs. Mod.)

82–1656. Dodge, R. **The Effects of Indoor Pollution on Arizona Children.** *Archives of Environmental Health* 37(3): 151-155, May-June 1982.

The respiratory health of a large group of Arizona schoolchildren who have been exposed to indoor pollutants—tobacco smoke and home cooking fumes—is reported. Questionnaires were completed by the parents and spirometry was performed on 676 third and fourth grade students (approximately 50 percent Mexican American) from schools in Morenci, San Manuel, and Kingman. A significant relationship was found between parental smoking and symptoms of cough, wheeze, and sputum production. Also, children in homes where gas cooking fuel was used had higher rates of cough than children in homes where electricity was used. No differences in pulmonary function or yearly lung growth rates occurred among subjects grouped by exposure to tobacco smoke or cooking fuel. Thus, parental smoking and home cooking fuel affected cross-sectional respiratory symptom rates in a large group of Arizona schoolchildren. Study of pulmonary function, however, revealed no lung function or lung growth effects during the 4 years of study. (Auth. Abs. Mod.)

82-1657. du Bois, R. M.; Townsend, P. J.; Cole, P. J.; Haslam, P. L.; Turner-Warwick, M. **Bronchoalveolar Macrophages in Sarcoidosis and Cryptogenic Fibrosing Alveolitis.** *Clinical Allergy* 11(5): 409-419, September 1981.

Human alveolar macrophages were studied to determine whether blood monocytes and tissue epithelial cells in patients with sarcoidosis are "activated." Bronchoalveolar lavage samples were obtained from 18 patients with sarcoidosis and these were compared with samples from 20 controls and 29 patients with nongranulomatous chronic inflammatory interstitial lung disease (cryptogenic fibrosing alveolitis (CFA)) to identify the functional characteristics intrinsic to macrophages of sarcoidosis patients. Assessment of the functional state of the macrophages was made by measurements of C3b receptor sites on the cell membrane, intra and extracellular lysosomal enzyme (β-D-glucosaminidase) and the degree of spreading of macrophages on glass. C3b receptor sites and intracellular levels of lysosomal enzyme were significantly reduced in sarcoidosis compared to controls; levels of extracellular enzyme in the lavage supernatant fluid and macrophage spreading were similar to controls. Comparing smokers with nonsmokers in each group, significant differences were found only within the CFA group. There were more erythrocyte antibody complement rosettes following zymosan treatment ($p<0.01$) and less spreading ($p<0.05$) in smokers. However, comparing CFA smokers to smoking controls, no significant difference was found in surface receptors for C3b following Zymosan stimulation, but there remained a significantly greater spreading in CFA ($p<0.025$). It is concluded that alveolar macrophages from patients with sarcoidosis are not "activated." By contrast, in CFA, macrophages show a greater extracellular:intracellular ratio of lysosomal enzyme and more spreading, suggestive of "activation." (Auth. Abs. Mod.)

82-1658. Fergusson, D. M.; Horwood, L. J.; Shannon, F. T.; Taylor, B. **Parental Smoking and Lower Respiratory Illness in the First Three Years of Life.** *Journal of Epidemiology and Community Health* 35(3): 180-184, September 1981.

Maternal smoking was found to be associated with childhood lower respiratory illness for the first year of life, but decreased thereafter. The relationships between parental smoking and the rates of lower respiratory illness during the first 3 years of life were examined for a birth cohort of 1,265 New Zealand children. Lower respiratory illness varied significantly with maternal smoking for the first year; there was equivocal evidence of a relationship between maternal smoking and lower respiratory illness in the second year; and by the third year the relationship had clearly disappeared. Paternal smoking had no significant effect on rates of lower respiratory illness at any time. Application of logistic regression showed that for the first year rates of lower respiratory illness were approximately linearly related to maternal smoking: increases of five cigarettes a day resulted in an increase of 2.5 to 3.5 incidents of lower respiratory illness per 100 children at risk. Statistical control for maternal age, education, family size, and family living standards showed that the relationship between maternal smoking and rates of lower respiratory illness was not significantly influenced by these factors. It is suggested that parental smoking may also lead to respiratory symptoms or illness by middle childhood—a passive smoking effect. (Auth. Abs. Mod.)

82-1659. Gishen, P.; Saunders, A.J.S.; Tobin, M. J.; Hutchison, D.C.S. **Alpha$_1$-antitrypsin Deficiency: The Radiological Features of Pulmonary Emphysema in Subjects of Pi Type Z and Pi Type SZ: A Survey by the British Thoracic Association.** *Clinical Radiology* 33(4): 371-377, July 1982.

The non-alpha$_1$-antitrypsin (AT)-deficient form of emphysema is closely associated with cigarette smoking. The habit has a weaker, though still significant, influence in patients with Pi type Z AT deficiency. Radiological data were obtained in 165 subjects homozygous for Pi type Z and in 23 subjects heterozygous for type SZ. Eighty-five percent of all type Z subjects had radiological evidence of pulmonary emphysema, which was commoner in smokers (90 percent) than in those who had never smoked (65 percent), and which predominantly affected the lower zones of the lungs. Smokers also constituted the larger portion of subjects with a subnormal cardiac transverse diameter, a low, flat position of the diaphragm, and larger than normal retrosternal space. An increased pulmonary artery diameter was observed in 19 (12 percent) of the subjects, all smokers. Of the type Z subjects identified through family studies, 66 percent also had emphysema. Emphysema in subjects of Pi type SZ was limited to those who had smoked. Direct radiological signs were observed in 36 percent of the subjects. Specific risk of emphysema in this group is attributable to smoking rather than to phenotype. (Auth. Abs. Mod.)

82-1660. Hall, S. K.; Cissik, J. H. **Effects of Cigarette Smoking on Pulmonary Function in Asymptomatic Asbestos Workers**

With Normal Chest Radiograms. *American Industrial Hygiene Association Journal* 43(6): 381-386, June 1982.

A possible additive effect between cigarette smoke and asbestos exposure is indicated from data obtained from 135 male asbestos workers participating in a comprehensive health screening program. The program consisted of a standardized health and occupational history questionnaire, blood chemistry profile, complete blood count, urinalysis, chest radiograph, and pulmonary function testing. All blood chemistry profiles and urinalysis results were within normal limits. Twenty-two of the asbestos workers had radiologic pulmonary abnormalities as well as severe pulmonary function impairment. The remaining 113 workers were divided into subgroups based on the number of years of exposure to asbestos and their smoking history. The data show a significant difference in pulmonary function between asymptomatic asbestos workers with normal chest radiographs who have less than 20 years' work exposure and those with more than 20 years' exposure. The smokers in each group had lower pulmonary function parameters than the nonsmokers, indicating a possible additive effect. Furthermore, a large constant loss in values is seen to begin early in exposure to asbestos. The loss levels off and actually decreases after 20 years' exposure but the irreversible damage has probably already occurred. (Auth. Abs. Mod.)

82–1661. Holmes, P.; Faulks, L. **Presentation of Pulmonary Tuberculosis.** *Australian and New Zealand Journal of Medicine* 11(6): 651-653, December 1981.

Smoking was one of several possible predisposing factors for pulmonary tuberculosis among 100 hospital patients (97 males, 3 females) in Australia. The patients, aged 42 to 85, were examined according to symptoms and the mode of presentation, chest X-ray data, and possible predisposing factors. It was found that 68 individuals were regular smokers, and 43 were thought to consume alcohol excessively. Other possible predisposing factors included marital problems, earlier partial gastrectomy, use of oral corticosteroid drugs, diabetes mellitus, and the presence of coexisting carcinoma of the lung. It is suggested that middle-aged male smokers with drinking problems and marital difficulties should be carefully examined for the presence of tuberculosis.

82–1662. Hughes, J. A.; Hutchison, D.C.S.; Bellamy, D.; Dowd, D. E.; Ryan, K. C.; Hugh-Jones, P. **The Influence of Cigarette Smoking and Its Withdrawal on the Annual Change of Lung Function in Pulmonary Emphysema.** *Quarterly Journal of Medicine* 51(202): 115-124, Spring 1982.

In 56 patients with pulmonary emphysema the forced expiratory volume in 1 sec and the vital capacity declined at a significantly faster rate in smokers ($n=37$) than in ex-smokers ($n=19$). The patients were followed for a minimum period of 3 years (range 3-13) to observe the effects of smoking and smoking withdrawal. Ex-smokers were defined as those who stopped smoking before entry into the study and did not smoke thereafter. The smokers included 18 patients who smoked throughout the study, 14 who stopped during the study, and 5 who stopped and restarted. Forced expiratory volume in 1 sec and vital capacity declined at a significantly faster rate in those who continued to smoke than in ex-smokers. No significant difference between the two groups was observed for permeability coefficient and arterial oxygen tension. There was a significant difference between the two groups in the annual change in body weight. It is concluded that all patients with emphysema, even those with advanced disease, should be encouraged to stop smoking. (Auth. Abs. Mod.)

82–1663. Jacobs, J. E. **Wideband Acoustic Energy Studies of Pulmonary Airways.** *Bioelectromagnetics* 3(1): 167-177, 1982.

The use of low-level wideband acoustic energy and cross-correlation techniques to obtain the impulse response of the pulmonary system appears to be an attractive method of noninvasively identifying important mechanical parameters of the pulmonary system. Typical correlation records for healthy, young adult male nonsmokers and smokers are presented. In the records for the nonsmokers, a steady repetition of a 2,000 Hz oscillation "A" type is apparent and the differences between the slots are readily recognizable; the amplitude of the peaks decays exponentially with delay for nonsmoking, healthy males. This pattern is almost never found in smokers or subjects with known lung disease. Typical records for apparently healthy smokers can be distinguished from the records of nonsmokers by the lower "B" type oscillation frequency of from 500 to 1,300 Hz, and an absence of a smooth decline in peak amplitude with increasing delay. (Model studies predict the presence of two regions of dominant signals from the basal regions of normal healthy lungs and that the higher frequency wave will not be present in lungs with thickened mucous layers.) A tabulation of the results of record analysis of one group of 39 subjects is also included. It is anticipated that further clinical studies coupled with advanced modeling procedures will establish the basis for more widespread use of this technique. (Auth. Abs. Mod.)

82–1664. Jedrychowski, W.; Flak, E.; Garlinska, J.; Bruzgielewicz, J. **Wystepowanie Objawow Przewleklego Niezytu Oskrzeli i Dychawicy Oskrzelowej wsrod Pracownikow Przemyslu Chemicznego. [Incidence of Chronic Bronchitis and Bronchial Asthma Among Workers in Chemical Industry.]** *Przeglad Epidemiologiczny* 35(4): 479-488, 1981, Polish.

Prevalence of chronic bronchitis (CB) and bronchial asthma (BA) was determined in 4,717 male workers of a Polish chemical combine to establish the effect of exposure to gases and dust at different work posts and that of smoking and age. Using questionnaire and spirometric data, a marked excess of CB and BA cases was found in smokers compared with nonsmokers within each age bracket. In workers under 30 years old, the total percentage of CB and BA cases among the ex-smokers was similar to the smokers. However, for ex-smokers over 50 years it was closer to that of nonsmokers.

Interrelationships between age and smoking were statistically significant in the cases of CB only. The incidence of both CB and BA among the smokers as well as nonsmokers increased with age, but the probability of CB and/or BA increased particularly fast among the smokers compared with the nonsmokers in the age bracket to 29 years. Symptoms of CB were predominant (41.8 percent) among the smokers compared with nonsmokers (22.8 percent) or ex-smokers (26.2 percent).

82–1665. Kauffmann, F.; Drouet, D.; Lellouch, J.; Brille, D. **Occupational Exposure and 12-Year Spirometric Changes Among Paris Area Workers.** *British Journal of Industrial Medicine* 39(3): 221-232, August 1982.

A followup study over 12 years was conducted among 556 men aged 30 to 54 in 1960 and working at that time in factories around Paris (France). Of the nonsmoking workers, 58 percent were exposed to at least one occupational hazard (grain dust, mineral dust, gases, and heat), and 73 percent of ex-smokers, 65 percent of moderate smokers, and 74 percent of heavy smokers were exposed to at least one hazard. The annual rate of decline of forced expiratory volume in one second (FEV_1) during 12 years was estimated for each subject from the measurements in 1960 and 1972. This rate (the FEV_1 slope) was related independently of FEV_1 level (which reflects the loss since the beginning of adult life) and of smoking habits to occupational exposure to dust, gases, and heat. After adjusting for age, smoking, and FEV_1 level (ASLA), the following average slopes were obtained: 44 ml/a (for exposure to none or to only a slight amount of dust, or to gases alone), 51 ml/a (heat), 53 ml/a (noticeable dust), 55 ml/a (noticeable dust and heat), 60 ml/a (noticeable dust, heat, and high concentration of gases). FEV_1 slope was significantly related to inhalation of mineral dust (even in the absence of silica) as well as to grain dust, and the slope was steeper with increased intensity of exposure to dust. The influence of heat on FEV_1 decline showed a clear trend. Results suggest that exposure to gases associated with exposure to dust or heat or both had a deleterious effect. There was no evidence of interaction between tobacco and the various risks. Men more noticeably exposed decreased or quit smoking more often than the others, and the FEV_1 slope is less steep for these men. Those who quit had, before stopping, a lower FEV_1 than other smokers. (Auth. Abs. Mod.)

82–1666. Knowles, M. R.; Buntin, W. H.; Bromberg, P. A.; Gatzy, J. T.; Boucher, R. C. **Measurement of Transepithelial Electric Potential Differences in the Trachea and Bronchi of Human Subjects** ***in Vivo.*** *American Review of Respiratory Disease* 126(1): 108-112, July 1982.

The airway transepithelial electric potential difference (PD), a parameter that reflects passive ion permeabilities and active ion transport across respiratory epithelia, was measured in vivo in human subjects. The ability of this technique to detect the effects of two potential insults to airway epithelium, cigarette smoke and poorly conditioned inspired air (tracheostomy), was assessed. A high impedance voltmeter was used to measure PDs between a fluid-filled and perfused recording bridge located on the airway surface and a reference bridge in the subcutaneous space. Tracheal PD was a relatively high 32 ± 5 mV (lumen negative) in subjects younger than 35 years of age ($n=23$). Tracheal PD declined after 35 years of age at a rate of 6 mV per decade. The PDs in bronchi were lower than in the trachea in subjects of all age groups. Neither the magnitude nor the regional variation of the PD appeared influenced by the chemical agents used in general anesthesia. However, superfusion of the airway surface with a pharmacologic inhibitor of Na^+ absorption, amiloride, significantly reduced PD in the trachea (38.5 ± 7.2 percent, $n=7$) and bronchi (-37.6 ± 6.3 percent, $n=6$). Both cigarette smoking and tracheostomy appeared to reduce tracheal PD when the patients were compared with age-matched control subjects. It was concluded that: (1) the tracheal PD in young human subjects is relatively high, (2) substantial regional variations in the magnitude of human airway PDs can be demonstrated, (3) active Na^+ absorption contributes to the generation of airway PD, and (4) measurements of PD in vivo may help detect early epithelial dysfunction. (Auth. Abs. Mod.)

82–1667. McMillan, G.H.G. **The Health of Welders in Naval Dockyards: Welding, Tobacco Smoking and Absence Attributed to Respiratory Diseases.** *Journal of the Society of Occupational Medicine* 31(3): 112-118, July 1981.

The relationship between absence attributed to respiratory disease, exposure to welding, and smoking habits of three groups of craftsmen in three naval dockyards was examined over a 5-year period. The men studied were welders, shipwrights, and boilermakers intermittently exposed to welding pollutants as neighborhood workers; and joiners, painters, and electrical fitters rarely, if ever exposed. In comparison with the other two groups of men, a higher proportion of welders could be classified as smokers, and a lower proportion as nonsmokers. Welders who are or have been smokers were found to have higher absence indices. Welders who have never smoked have lower absence indices compared with nonsmokers in the other two groups. The presence of welding pollutants in the working environment may prompt welders with respiratory symptoms to take leave although the cause of these symptoms may be unrelated to work. (Auth. Abs.)

82–1668. Melville, G. N.; Kumar, M.; Ismail, S.; Mills, J. L. **Response of the Central and Peripheral Airways to Cigarette Smoking in Humans and Rats.** *Respiration* 43(1): 29-34, January-February 1982.

In both humans and rats tobacco smoke affected the central and peripheral airways when assessed by direct and indirect methods. In humans there were apparent decreases in the 1-sec forced expiratory volume and peak expiratory flow rate, and significant increases in closing volume and closing capacity ($p<0.001$). In rats significant changes were seen in specific airway resistance from the 6th week of exposure onwards.

Similarly, airway luminal diameter decreased markedly in tobacco-exposed animals stimulated with subthreshold concentrations of acetylcholine (10^{-8}M). This decrease was also exposure-time dependent. The increased responsiveness of the respiratory system has been partially attributed to: (1) increased vagal activity; (2) increased mucus production leading to decreased airway lumen; (3) mucosal swelling due to changed ionic constellation; (4) disturbance of the lungs' defense mechanism; and (5) imbalance in and easy accessibility to the adrenoceptors. (Auth. Abs. Mod.)

82–1669. Mollo, F.; Bollati, A.; Bergia, R.; Colombo, A. **Bronchial Mucus Gland Enlargement in "Healthy" Subjects. An Autopsy Survey on 343 Cases of Traumatic Death, in Turin.** *Pathologica* 73(1024): 185-192, March-April 1981.

Bronchial mucus gland enlargement (BMGE) was more common among smokers than nonsmokers in a population sample from Turin, Italy. Bronchial tissue specimens were taken during 343 autopsies performed from 1974 to 1977. The gland to wall ratio (G/W) was measured, and the results classified as normal, borderline, and evidence of BMGE. Individual factors examined included smoking habits, age, sex, and current or most recent occupational activity. Data on smoking were gathered from close relatives for 82 cases (43 smokers, 39 nonsmokers). Smokers were defined as all subjects who had smoked regularly at least until the last year before death. Ex-smokers (excluding individuals who had quit within 12 months before death) were considered to be nonsmokers. Evidence of BMGE was significantly more common for smokers (41.9 percent) than nonsmokers (14.4 percent), and normal measurements were more frequent for nonsmokers (43.6 percent) than for smokers (20.9 percent). Although BMGE was more common in men, the relative risk associated with smoking was much larger for women indicating that smoking is more effective as a BMGE factor for women.

82–1670. Nash, D. R.; Fortson, N. G.; McLarty, J. W.; Hurst, G. A. **Serum Protein Concentrations and Respiratory Tract Abnormalities Following Short-Term Exposure to Amosite: A Comparison Between Former Asbestos Workers and Unexposed Controls.** *Journal of Toxicology and Environmental Health* 7(5): 733-744, May 1981.

Smoking had an adverse effect on the respiratory systems of a group of 176 former asbestos workers evaluated and compared with 76 unexposed controls of similar age for concentrations of serum immunoglobulins, α_1-antitrypsin, carcinoembryonic antigen (CEA), pulmonary function, radiographic appearance of the lungs, rales, cellular atypia and ferruginous bodies in sputum. All study participants were classified as nonsmokers, light smokers, or heavy smokers. The asbestos-exposed group had a much higher percentage of heavy smokers. Test subjects were evaluated as a single group and also according to years of exposure to asbestos. Both test and control populations were independently subjected to parametric and nonparametric correlation analyses. Partial correlations were also determined for both groups after controlling for age, race, sex, and smoking habits. Analysis of variance was used to compare test and control groups. Smoking, particularly heavy smoking, was an important variable influencing abnormalities of the respiratory tract and CEA in former asbestos workers but not in controls, reemphasizing the synergistic influences of smoking in relation to a variety of lung diseases. (Auth. Abs. Mod.)

82–1671. Poe, R. H.; Israel, R. H.; Utell, M. J.; Hall, W. J. **Chronic Cough: Bronchoscopy or Pulmonary Function Testing?** *American Review of Respiratory Disease* 126(1): 160-162, July 1982.

Pulmonary function testing was found to have diagnostic value in an examination of patients with chronic cough as an isolated clinical symptom, but bronchoscopy was of limited value. The records of all patients referred to pulmonary specialists at three community hospitals for evaluation of cough over a 6-year period were reviewed. There were 109 patients (56 smokers, 22 ex-smokers, 53 nonsmokers), 15 to 89 years of age with the isolated symptom of chronic cough and a normal or stable chest roentgenogram. Bronchoscopy was performed in 51 patients (31 smokers/ex-smokers, 20 nonsmokers). All patients but one were followed until the cough disappeared or for a minimum of 1 year. Carcinoma was established as the cause for cough in one patient (a nonsmoker). Asthma was the most frequently observed cause, found in 39 patients (8 smokers, 8 ex-smokers), followed by respiratory infection, bronchitis, and postnasal drip. Based on the finding that the incidence of bronchogenic carcinoma is very small in patients with persistent cough as the only symptom, it is recommended that fiberoptic bronchoscopy be reserved for patients in whom the cause of the cough is not identified by clinical history and pulmonary function testing.

82–1672. Rees, P. J.; Chowienczyk, P. J.; Clark, T.J.H. **Immediate Response to Cigarette Smoke.** *Thorax* 37(6): 417-422, June 1982.

Narrowing of the bronchial airways almost invariably follows inhalation of cigarette smoke. Changes in airway resistance were measured in 12 smokers and 12 nonsmokers using an automated method in the body plethysmograph. Decreases in specific conductance occurred within 7 or 8 seconds after exposure to single inhalations of cigarette smoke. Less than half of the initial change was present 40 seconds after the inhalation. Initial responses were greater in the nonsmokers. Responses recurred with repeated inhalations in smokers and nonsmokers, although the responses decreased with repeated exposure, and failed to return to baseline values between inhalations. Prior administration of salbutamol (800 μg) and ipratropium bromide (0.072-0.54 mg) significantly inhibited the response and this effect appeared to be greater in nonsmokers: a dose of 0.54 mg was required to inhibit airways response in smokers. Sodium cromoglycate inhaled as a dry powder had no effect on the response. (Auth. Abs. Mod.)

82–1673. Sackner, M. A.; Rao, A.S.V.; Birch, S.; Atkins, N.; Gibbs, L.; Davis, B. **Assessment of Density Dependent Flow-Volume Parameters in Nonsmokers and Smokers. Measurement With Spirometry, Body Plethysmography and Respiratory Inductive Plethysmography.** *Chest* 82(2): 137-142, August 1982.

Density-dependent flow-volume information using helium as a test gas alone or compared with air was determined to be a good approach to detection of early lung disease in smokers. Simultaneous spirometry, respiratory inductive plethysmography, and body plethysmography were used to detect small airway disease. The forced vital capacity (FVC) was measured during air, helium-oxygen, and sulfur-hexafluoride breathing to provide a range of gas density influences. Combining flow measured at the mouth with volume referenced to change in alveolar gas volume as measured by body or respiratory inductive plethysmography during helium-oxygen breathing accentuated the differences between nonsmokers and smokers. This was due to the variable degree of alveolar gas compression occurring over the second half of FVC. The volume of isoflow obtained when comparing the helium and oxygen mixture to air also effectively separated nonsmokers from smokers. The utilization of the high density gas mixture, sulfur hexafluoride-oxygen, during the FVC maneuver did not provide useful diagnostic information. (Auth. Abs. Mod.)

82–1674. Sparrow, D.; Silbert, J. E.; Weiss, S. T. **The Relationship of Pulmonary Function to Copper Concentrations in Drinking Water.** *American Review of Respiratory Disease* 126(2): 312-315, August 1982.

Tap water concentrations of copper were found to be unrelated to lung function or plasma copper levels among current or ex-smokers. Copper has been shown to be an important cofactor for certain enzymatic reactions: cross-linking of elastin is inhibited by copper deficiency. In animal models, this inhibition leads to weakened connective tissue and pathologic changes in the lungs consistent with emphysema. To explore the potential relationship of copper exposure to level of pulmonary function in humans, copper concentrations in tap water were examined in the homes of 297 adult male subjects involved in the Normative Aging Study. Forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV_1) were obtained by standard techniques. The relationship of tap water copper concentration to pulmonary function was explored using multiple regression analysis, controlling for age, height, smoking status, and educational attainment. Separate regressions were performed for each of three smoking status groups: never, former, and current. Among never smokers, tap water copper was significantly and positively related to levels of both FVC ($p=0.014$) and FEV_1 ($p=0.027$). No significant trend was found among former or current smokers. (Auth. Abs. Mod.)

82–1675. Tobin, M. J.; Hutchison, D.C.S. **An Overview of the Pulmonary Features of α_1-Antitrypsin Deficiency.** *Archives of Internal Medicine* 142(7): 1342-1348, July 1982.

Extensive research, stimulated by the recognition of an association between α_1-antitrypsin (α_1-AT) deficiency and emphysema, has greatly advanced our understanding of emphysema in general. Since smoking can lead to a deficiency of α_1-AT, it may promote emphysema. Literature is reviewed concerning the basic defect; inheritance; pathogenesis of lung disease; and clinical, physiologic, and roentgenographic findings in patients with severe (Pi Z) and intermediate (Pi SZ) deficiency of α_1-AT. Studies confirm the increased risk of developing lung disease in cigarette smokers. Smoking increases the numbers of macrophages, recruits neutrophils to the lungs, induces the release of proteases from both cell types, and causes a localized deficiency of α_1-AT. Proteases can destroy the alveolar wall. However, the question as to why all cigarette smokers do not have emphysema remains unanswered. (Auth. Abs. Mod.)

82–1676. Wegman, D. H.; Peters, J. M.; Boundy, M. G.; Smith, T. J. **Evaluation of Respiratory Effects in Miners and Millers Exposed to Talc Free of Asbestos and Silica.** *British Journal of Industrial Medicine* 39(3): 233-238, August 1982.

Indications are that talc exposure and smoking each impair respiratory health among talc miners and millers, although an interaction between the two factors was not definitely established. The talc ore was free from asbestos and silica. The study of 116 subjects included the modified British Medical Research Council respiratory symptom questionnaire, simple spirometry, chest radiography, and limited examination of the chest. A 1-year followup evaluation was performed on 103 subjects. Of the 12 subjects whose chest radiographs showed small round or irregular opacities (perfusion 1/0 or greater), five had never smoked cigarettes. Pulmonary function values at the time of the initial study were less than predicted and the rates of loss of forced expiratory volume in 1 sec and forced vital capacity were greater than expected. These differences were in part attributed to cigarette smoking but were greater than predicted from that source alone. There was evidence of an exposure effect after adjusting for cigarette smoking and it is concluded that these workers were affected by their exposures. (Auth. Abs. Mod.)

See also, **82–1569, 82–1738**

CARDIOVASCULAR DISEASES

82-1677. Brenner, M. H.; Mooney, A. **Economic Change and Sex-Specific Cardiovascular Mortality in Britain 1955-1976.** *Social Science and Medicine* 16(4): 431-442, 1982.

A multivariate model of the impact of changes in the socioeconomic and biophysical environments was developed and applied to cardiovascular disease (CVD) mortality rates, by sex, in England, Wales, and Scotland during 1955-1976. The predictive model included factors associated with (1) long-term growth in the economy; (2) deleterious behavioral risk factors loosely associated with economic growth—especially cigarette consumption per capita; (3) economic instability—especially recession as indicated by factors related to unemployment, income loss, and recessional declines in average weekly hours worked in manufacturing industries; (4) health care; and (5) physical environmental disturbances—especially very cold temperatures. The model proved to be an excellent instrument for the statistical explanation of trends and fluctuations in CVD mortality rates for both sexes and both regions in Britain in the post-War period. In general, the overall exponential rate of economic growth is found to be the most powerful factor in the long-term decline in CVD mortality rates. Similarly, disturbances to the national and regional economic situations—especially recessions—have regularly been associated with elevated death rates for all populations observed. Cigarette and unusually high spirits consumption, as well as particularly cold winter temperatures, have also had important deleterious effects on CVD mortality. The proportion of government expenditures devoted to health care is associated with a reduction in CVD mortality in England and Wales. (Auth. Abs.)

82-1678. Brill, R. W.; Jones, D. R. **On the Suitability of Innovar, a Neuroleptic Analgesic, for Cardiovascular Experiments.** *Canadian Journal of Physiology and Pharmacology* 59(11): 1184-1189, November 1981.

The effect of Innovar on the bradycardia and vasoconstrictor response to cigarette smoke (the "nasopharyngeal reflex") was tested in rabbits given 0.2 or 0.5 mL/kg doses. Innovar's effects on arterial pressure, heart rate, respiration rate, ventricular contractility, and arterial oxygen tension were also recorded. In animals given 0.2 mL/kg Innovar, the efficacy of the nasopharyngeal reflex and all cardiovascular variables returned to preinjection levels within 45 minutes. The nasopharyngeal reflex returned to normal within 90 minutes in animals given 0.5 mL/kg Innovar, but the cardiovascular variables (except arterial oxygen tension) were depressed for up to 135 minutes after administration. The calmative and analgesic effects of Innovar, and its only moderate effects on cardiovascular functions and reflexes, make it a suitable neuroleptic analgesic agent for cardiovascular experiments where neither fully conscious nor surgically anesthetized animals can be used. (Auth. Abs. Mod.)

82-1679. Burch, P.R.J.; Tweed, W. A. **Cigarette Smoking and Coronary Heart Disease. (Letters).** *Canadian Medical Association Journal* 127(2): 107, 111, July 15, 1982.

Further arguments on the relationship between cigarette smoking and coronary disease (CD) are presented in which Burch cites omissions in an earlier editorial (*Canadian Medical Association Journal* 126(2): 102, 104, 107, January 15, 1982) by Tweed who argued for the causal relationship. The aspects of the age dependence of fatal CD and the secular trends in cigarette smoking are noted as evidence against finding smoking an important cause of CD. The possibility of counteracting factors is suggested, and it is concluded that any association between smoking and CD is at the constitutional (genetic) level. Tweed's reply questions the validity of the mathematical model used by Burch, since age was the only true variable, and any observed mortality in aging populations could fit some variation of the model. He also questions the constant latency period assumption, since there were striking inconsistencies among the different populations examined. Tweed states that the evidence against smoking is too well documented to be seriously questioned, and that the theory of a decline of a particular influenza virus as the factor in reduced CD rather than documented changes in cigarette smoking, diet, and hypertension control is simply evidence of bias on the part of Burch. See also 82-0750.

82-1680. Clyne, C.A.C.; Arch, P. J.; Carpenter, D.; Webster, J.H.H.; Chant, A.D.B. **Smoking, Ignorance, and Peripheral Vascular Disease.** *Archives of Surgery* 117(8): 1062-1065, August 1982.

The effect of the surgeon's advice to give up smoking was studied in a group of 43 patients with peripheral vascular disease (PVD), using blood carboxyhemoglobin levels to detect deception. Only 7 of 40 patients (17.5 percent) actually stopped smoking, but the remainder appeared fairly honest in reporting their continued habits. Few patients were aware of the possible harmful effect of smoking on the peripheral arteries. The 43 patients with PVD who smoked had significantly higher carboxyhemoglobin levels than a group of 25 smokers without PVD although their cigarette consumption was the same. This suggests that carboxyhemoglobin levels may provide a better indication of the risk of smoking than overall cigarette consumption alone in the development of PVD. (Auth. Abs.)

82-1681. Colman, R. W. **The Role of Platelets in the Genesis of Ischemia.** *Annals of the New York Academy of Sciences* 382: 190-203, 1982.

The way in which coronary artery arteriosclerosis risk factors, such as smoking, stress, and hyperlipoproteinemia, may act in part through platelets, is discussed. There is clinical and laboratory evidence that platelets play a role in sudden death due to coronary artery disease. Two mechanisms appear to enhance the activation of platelets by smoking: (1) products that can be extracted from tobacco smoke can directly activate the zymogen coagulant factor XII to the active enzyme factor XIIa, and factor XIIa can then stimulate the intrinsic coagulation cascade to eventually produce thrombin, a potent platelet agonist; (2) smoking also may act to stimulate the release of amines such as epinephrine, perhaps directly through the action of nicotine on the adrenal glands. Smokers have been found to have increased numbers of platelet aggregates in their circulation. It is noted that smoking, stress, and other risk factors may interact with the various mechanisms to enhance each of the platelet aggregators in the production of intracellular prostaglandins. Trials of antiplatelets indicate that they are capable of preventing sudden death in myocardial infarction, and agents such as aspirin appear to have an effect in the prevention of transient ischemic attacks, although problems exist in the interpretation of the epidemiologic evidence for the efficacy of these drugs. Studies have identified important trends which suggest areas of further research, particularly the interactions of platelets with risk factors and the influence of platelets on the formation of the arteriosclerotic plaque.

82–1682. Criqui, M. H.; Mebane, I.; Wallace, R. B.; Heiss, G.; Holdbrook, M. J. **Multivariate Correlates of Adult Blood Pressures in Nine North American Populations: The Lipid Research Clinics Prevalence Study.** *Preventive Medicine* 11(4): 391-402, July 1982.

The associations of seven variables—age, education, body mass index, alcohol consumption, cigarette smoking, regular exercise, and gonadal hormone use in women—with systolic and diastolic blood pressure were studied in 4,780 white adult men and women in nine North American populations. Smoking caused acute increases in blood pressure, but overall was negatively associated with both systolic and diastolic pressure. This may be due to secondary factors, though little or no changes in blood pressure have been recorded after smoking cessation. Age was positively associated with systolic and diastolic pressure in men and younger women, and with systolic but not diastolic pressure in older women. Education was not consistently associated with blood pressure. Body mass index and alcohol consumption were positively associated with systolic and diastolic pressure. Regular exercise was negatively associated with systolic pressure in women and diastolic pressure in men. Gonadal hormone use in younger women, mostly oral contraceptives, was positively associated with systolic pressure. Gonadal hormone use, mostly postmenopausal estrogens, showed a weak positive association with systolic pressure in older women but this association was not statistically significant. It is noted that, with the exception of age and possibly education, these variables are all amenable to preventive intervention. (Auth. Abs. Mod.)

82–1683. Dyer, A. R.; Stamler, J.; Shekelle, R. B.; Schoenberger, J. A.; Stamler, R.; Shekelle, S.; Berkson, D. M.; Paul, O.; Lepper, M. H.; Lindberg, H. A. **Pulse Pressure—I. Level and Associated Factors in Four Chicago Epidemiologic Studies.** *Journal of Chronic Diseases* 35(4): 259-273, 1982.

In cross-sectional analyses, the association between adjusted pulse pressure and age, serum cholesterol, relative weight, heart rate, glucose, cigarette use, serum uric acid, and hematocrit was examined. Data from four Chicago epidemiologic studies were used to determine whether or not factors that have been shown to be related to blood pressure and hypertension are related to pure systolic hypertension or the "classical" hypertension. For cigarette use the coefficients in the multivariate analyses were computed without heart rate in the equation, because cigarette smoking increases heart rate. Cigarette use appeared to be positively related to pulse pressure, particularly among white males; in seven of nine groups of white males, cigarettes/day had a significant positive association with adjusted pulse pressure in the multivariate analyses. There was also a significant positive association in two of the five groups of white females and in one of four groups of black males. However, there was a significant negative association for black females aged 45-64 years. Cigarette use also showed positive associations with adjusted pulse pressure at all ages, with no marked change in the association with age. The association between cigarettes and adjusted pulse pressure may reflect an increased peripheral resistance among smokers, or the presence of aortic atherosclerosis and arterial inelasticity, since smoking is a risk factor for the atherosclerotic diseases. Overall results suggest that glucose, heart rate, and cigarette use are related to pure systolic hypertension, while hematocrit, serum cholesterol, serum uric acid, and relative weight are related to "classical" hypertension, although relative weight may be related to pure systolic hypertension in young adult men and women. (Auth. Abs. Mod.)

82–1684. Dyer, A. R.; Stamler, J.; Shekelle, R. B.; Schoenberger, J. A.; Stamler, R.; Shekelle, S.; Berkson, D. M.; Paul, O.; Lepper, M. H.; Lindberg, H. A. **Pulse Pressure—II. Factors Associated With Follow-Up Values in Three Chicago Epidemiologic Studies.** *Journal of Chronic Diseases* 35(4): 275-282, 1982.

In both cross-sectional and prospective analyses, cigarette use was found to be related more strongly to systolic than diastolic blood pressure. In this, the second in a series of reports on pulse pressure and pure systolic hypertension, the association between the initial values and the changes in five variables (serum cholesterol, relative weight, heart rate, glucose, and cigarette use) were examined in prospective analyses. Data from three Chicago epidemiologic studies were used to determine whether variables known to be related to blood pressure and hypertension are related to pure systolic hypertension or "classical" hypertension. In these analyses, followup pulse pressure, which was measured from 2 to 5 years after the initial measurement of the other variables, was redefined so that the association between the initial value or change and pulse pressure indicated whether the initial value or change was more strongly related to followup systolic or diastolic blood pressure.

Only the initial value for cigarette use had a consistent positive association with followup pulse pressure, suggesting that cigarette use is related to pure systolic hypertension, rather than "classical" hypertension. Change in heart rate was generally positively related to followup pulse pressure, while the initial value was not. For relative weight and serum cholesterol, both the change and the initial value tended to be negatively related. For glucose, the association was not consistent for either the initial value or the change. (Auth. Abs. Mod.)

82–1685. Freis, E. D. **Should Mild Hypertension Be Treated?** *New England Journal of Medicine* 307(5): 306-309, July 29, 1982.

According to epidemiologic investigations, such as the Framingham Study, the risk of myocardial infarction (MI), the most frequent complication in mild hypertension, varies markedly, depending on the number of other risk factors present. Among adults having diastolic pressures of 90 to 99 mmHg (about one fifth of the population), and no other cardiovascular risk factors, the potential toxic and financial consequences outweigh the benefits of possible infarct prevention. With multiple risk factors (cigarette smoking, electrocardiographic evidence of left ventricular hypertrophy, hypercholesterolemia, and glucose intolerance) the risk in men is 26.6 percent with a 165 mmHg systolic pressure and 20.0 percent with 135 mmHg. These are considerable risks, and the increment in risk due to hypertension alone is 6.6 percent. Therefore, if hypertensive treatment is beneficial in reducing MI risk, it is most beneficial in these patients. Parenthetically, stopping smoking is nearly as beneficial as lifelong drug treatment; however, the National Health Interview Survey showed only one in three hypertensive smokers had been advised by a doctor to quit. A compromise position is suggested: patients with diastolic pressures of 90-99 mg at three visits are to be treated or not, according to the number of risk factors present; those with fewer risk factors are to be given reducing or low-sodium diets and advised to quit smoking, but no drugs.

82–1686. Herman, B.; Leyten, A.C.M.; van Luijk, J. H.; Frenken, C.W.G.M.; Op de Coul, A.A.W.; Schulte, B.P.M. **An Evaluation of Risk Factors for Stroke in a Dutch Community.** *Stroke* 13(3): 334-339, May-June 1982.

Smoking was not found to be a significant risk factor for stroke in an epidemiological study of the Dutch community of Tilburg. A total of 126 stroke patients and 212 controls were examined. Among the risk factors surveyed were smoking, hypertension, diabetes mellitus, myocardial infarction, cardiac arrhythmia, other heart conditions, overweight, possible transient cerebral ischemic attack symptoms, and extent of physical activity. Stroke was not significantly associated with smoking vs. not smoking, the quantity of cigarettes smoked daily, the duration of smoking, and the combination of these factors. The adjusted relative risk for smokers of any quantity of cigarettes as compared with nonsmokers was 1.2. No trend in stroke risk with amount of cigarettes consumed was found.

82–1687. Hutchinson, R. R.; Emley, G. S. **Nicotine Ingestion Reduces Elevated Blood Pressures in Rats and Squirrel Monkeys.** *Life Sciences* 28(7): 801-809, 1981.

Nicotine tartrate (0.002-5.0 mg/kg/day) was added to the home cage drinking water of albino rat and squirrel monkey subjects for periods from 1 to 10 weeks. Rats received the drug for approximately 10 weeks and were then implanted with a chronic aortic cannula, allowed to recuperate, and blood pressure measured before and following exposure to a mild tail pinch procedure. All subjects receiving nicotine showed reduced blood pressure elevations to noxious stimulation compared to water control subjects, and these reductions were greater at higher drug doses. Squirrel monkeys exhibiting widely differing resting blood pressures were administered chronic dosages of nicotine. Subjects having high blood pressures showed dose—dependent blood pressure decreases, whereas subjects possessing low resting pressures demonstrated little or no pressure change during drug intake. A followup study with high blood pressure subjects measured effects of progressive increases in drug followed by a return to drug-free water drinking solutions. Here again a dose—dependent decrease in both systolic and diastolic blood pressures was observed. Upon termination of drug intake, pressures rapidly increased to predrug levels. The results are concordant with previous studies demonstrating that oral nicotine ingestion causes reductions in aggressive behavior, and suggest that nicotine is selectively influential in altering the biological state of anger, decreasing both its behavioral and somatic components. (Auth. Abs.)

82–1688. Jarrett, R. J.; McCartney, P.; Keen, H. **The Bedford Survey: Ten Year Mortality Rates in Newly Diagnosed Diabetics, Borderline Diabetics and Normoglycaemic Controls and Risk Indices for Coronary Heart Disease in Borderline Diabetics.** *Diabetologia* 22(2): 79-84, February 1982.

Mortality rates from coronary heart disease and from all causes were ascertained over 10 years in three groups of people participating in the Bedford Survey—newly-diagnosed diabetics, borderline diabetics, and control subjects with normal glucose tolerance. Age-corrected mortality rates, from all causes and coronary heart disease, were highest in the diabetics and intermediate in the borderline diabetics and in both groups were similar in men and women. During the 10-year followup of the Bedford borderline diabetics, coronary heart disease morbidity and mortality rates were similar in men and women. Age at entry to the study was the major independent and significant predictor of mortality from all causes. The level of systolic blood pressure and current cigarette smoking at baseline were statistically significant predictors only of mortality due to coronary heart disease. (Auth. Abs. Mod.)

82–1689. Kannel, W. B.; Thomas, H. E., Jr. **Sudden Coronary Death: The Framingham Study.** *Annals of the New York Academy of Sciences* 382: 3-21, 1982.

The occurrence of sudden death (within 1 hr of onset of symptoms) over a 26-year followup period in the Framingham Study was examined. Of the 5,209 subjects, 131 men and 49 women died suddenly and unexpectedly, 60 percent without prior indications of overt coronary heart disease (CHD). The incidence of these sudden deaths increased with age, averaging 35/1,000. This incidence was related to cigarette smoking, blood pressure, serum cholesterol, glucose intolerance, left ventricular hypertrophy, heart rate, and relative weight. It is now widely acknowledged that cigarette smoking predisposes to coronary heart disease. Data from the Framingham Study continue to show that cigarette smokers are at significantly higher risk of CHD and of sudden death in particular. Risk of sudden death is increased almost three-fold in cigarette smokers compared with nonsmokers. There is also a positive trend in the proportion of CHD deaths that are sudden that increases with the amount of cigarette smoking. Smoking cessation may, therefore, be one way to help reduce the incidence of sudden coronary death. (Auth. Abs. Mod.)

82–1690. Kennelly, B. M.; Gersh, B. J.; Lane, G. K.; Beck, W. **The Relationship Between Angiographic Findings and Risk Factors in Young Men With Myocardial Infarction.** *South African Medical Journal* 61(14): 508-512, April 3, 1982.

Coronary angiography was performed and risk factors were evaluated 2-4 months after myocardial infarction in 50 men aged 40 years or less. There was a high incidence of heavy cigarette smoking (62 percent smoked at least 1 pack cigarettes per day), obesity, and hyperlipidemia among these young men. Single-vessel disease (≥ 70 percent coronary obstruction) was found in 52 percent, double-vessel disease in 22 percent, and triple-vessel disease in 20 percent, the right coronary artery being more frequently involved (≥ 70 percent obstructed) or totally occluded than the left anterior descending or left circumflex coronary arteries, in that order. One of the two patients with a normal coronary arteriogram had left ventricular angiographic evidence of previous infarction. There was no apparent difference in the distribution of coronary artery disease in this group of young men from the reported distribution found in older subjects. Neither was there any significant correlation of any single major coronary risk factor or combination thereof with the extent or severity of coronary artery obstruction, although well-recognized risk factors such as elevated serum lipid levels, cigarette smoking, hypertension, diabetes, and obesity are common in patients presenting with acute myocardial infarction at an early age. (Auth. Abs. Mod.)

82–1691. Lamm, G. **The Cardiovascular Disease Programme of WHO in Europe.** World Health Organization, Regional Office for Europe, Copenhagen, Public Health in Europe 15, 1981, 143 pp.

In 1968, the Regional Office for Europe of the World Health Organization launched a long-term program on the study and control of cardiovascular disease (CVD). This review of the program, written chiefly for health professionals, includes chapters on: (1) the background and various components of the program; (2) its weaknesses and specific achievements; (3) a survey of established knowledge and unresolved questions; and (4) considerations for the future. An early achievement of the program was the basic confirmation of the association of CVD with the four major risk factors: smoking, overweight, high blood pressure, and high cholesterol levels. In multifactorial prevention trials, it was found that relatively cheap and simple methods, concentrating on the major risk factors, could intervene in CVD. In rehabilitation, new secondary prevention measures were instituted, correlating with primary risks: stopping smoking, exercise, and controlling blood pressure and diet. In the light of multiple-risk theory, in contrast to individual risk factors, the differences between populations such as the United States and Europe are better understood. For instance, the population effect of cigarette smoking depends on the extent of the habit in the population, and on the presence and magnitude of other risk factors and differences in the chemical content of the tobacco. It must be considered that smoking is involved not only in CVD, but in other diseases as well, especially cancer and chronic bronchitis. Thus the key concept for the future is integration of specific disease programs into total health action. Seven appendices exploring the details of coronary care are included.

82–1692. Layde, P. M.; Ory, H. W. **Oral Contraceptives, Smoking, and Cardiovascular Disease: Response to** F. M. **Sturtevant.** *International Journal of Fertility* 27(1): 14-15, 1982.

Sturtevant's conclusion that the effect of oral contraceptives (OCs) on the risk of cardiovascular disease is similar in smokers and nonsmokers (*International Journal of Fertility* 27(1): 2-13, 1982) is not supported by the data when the data are analyzed more critically. (1) Terms such as potentiation, synergy, and interaction are confusing and users of these terms disagree on their meaning. (2) Although in some situations there appears to be a greatly increased risk in users of OCs over nonusers, the baseline risk may be so low that the high relative risk from OC use produces only a small excess (or attributable) increased risk in absolute terms. (3) Data from cohort studies, such as the Royal College of General Practitioners study, should be more heavily weighted than studies based on small numbers of cardiovascular disease because the failure to demonstrate a higher risk in a particular population could be due to chance. Despite confusing terminology and inconsistencies, the weight of existing evidence points clearly to one conclusion: women 35 years and older who smoke are at higher risk of OC-associated cardiovascular disease than are younger women and those who don't smoke. See also 82-1708.

82–1693. Loscutoff, S. M.; Jaffe, R. A.; Hilton, D. I.; Phelps, D. W.; Carr, D. B.; Wehner, A. P. **Dosimetry and Cardiopulmonary Function in Rats Chronically Exposed to Cigarette**

Smoke. *Toxicology and Applied Pharmacology* 64(2): 335-352, June 30, 1982.

Groups of 80 female F-344 rats were exposed to smoke from three types of research cigarettes: high-tar, low-nicotine (HT-LN); low-tar, high-nicotine (LT-HN); and high-tar, high-nicotine (HT-HN) at a rate of approximately 10 puffs during each of eight daily exposure sessions, 7 days/week for 24 months. Additional groups were sham-exposed on the same schedule, or served as untreated "shelf" controls. Heart rate and breathing pattern (tidal volume, respiration rate, and minute volume) were measured both during exposure and nonexposure intervals. During exposure, smoke-exposed rats inhaled 75 percent less air and had 40 percent lower heart rates than sham-exposed rats. Results were similar for all smoke-exposed groups, and differences between sham- and smoke-exposed groups persisted throughout the 24-month exposure. When not being exposed, heart rate and breathing pattern were also different between smoke- and sham-exposed groups; tidal volumes were higher and respiration and heart rates were lower in smoke-exposed than in sham-exposed animals. In general, these differences between smoke- and sham-exposed groups developed during the first 12 months of exposure and diminished thereafter. Of the smoke-exposed groups, rats exposed to LT-HN cigarettes showed the greatest changes, and rats exposed to HT-LN cigarettes showed the least changes, in heart rate and breathing patterns. Arterial blood pressure was consistently higher in smoke-exposed than in sham-exposed animals following 18 and 24 months of exposure. This study showed that cardiopulmonary function in rats was significantly affected by chronic cigarette-smoke exposure. (Auth. Abs. Mod.)

82–1694. Nevius, E.; Bennett, R.; Sobel, S. **Critique of an Article by F. M. Sturtevant Entitled "Smoking, Oral Contraceptives, and Thromboembolic Disease."** *International Journal of Fertility* 27(1): 16-20, 1982.

Sturtevant's contention (*International Journal of Fertility* 27(1): 2-13, 1982) that smoking is a strong risk factor for some forms of cardiovascular disease and does not interact with oral contraceptives (OCs) use is criticized for placing the emphasis in the wrong direction. The conclusion should be that the independent risk of OC use is the important one in regards to deep venous thrombosis and cerebral artery disease, whereas, combined smoking and OC use is the important consideration in ischemic heart disease. A problem exists in the perception of the terms "synergism" and "relative risk." A reappraisal of the same case—control studies leads to somewhat different observations. In the case of deep vein thrombosis, there is strong evidence of increased risk due to OC use. The evidence that smoking is a risk factor or that there is an interaction between OC use and smoking is ambiguous. There is insufficient evidence to conclude that an interaction exists between OC use and smoking for either thrombotic or hemorrhagic stroke. Strong evidence has been found in both types of stroke of an increased risk due to OC use. In addition, smoking has been implicated as a risk factor for hemorrhagic stroke. Sturtevant's conclusion that cigarette smoking appears to be a significant risk factor for myocardial infarction is reasonable but his conclusion that OC use does not appear to be a risk factor is not. There is a good deal of evidence for implicating OC use and smoking together as producing a high risk for ischemic heart disease. See also 82- .

82–1695. Okada, M.; Ueda, K.; Omae, T.; Takeshita, M.; Hirota, Y. **The Relationship of Serum Uric Acid to Hypertension and Ischemic Heart Disease in Hisayama Population, Japan.** *Journal of Chronic Diseases* 35(3): 173-178, 1982.

No correlation was found in a Japanese study population between serum uric acid (SUA) level and the amount of tobacco smoked in an examination of the association of SUA with hypertension, ischemic ECG change, diabetes mellitus, and drinking and smoking habits. A multivariate analysis was made of cross-sectional data collected from 1,871 residents aged 40 and over in Hisayama, Japan. Blood pressure level and history of hypertension did not correlate with an increased level of SUA when other factors such as serum creatinine, obesity, beta-lipoprotein, and leucineaminopeptidase were taken into account. Antihypertensive medication was a significant factor influencing SUA. Glycosuria was negatively correlated with SUA, while alcohol ingestion had positive correlation. (Auth. Abs. Mod.)

82–1696. Palac, R. T.; Meadows, W. R.; Hwang, M. H.; Loeb, H. S.; Pifarre, R.; Gunnar, R. M. **Risk Factors Related to Progressive Narrowing in Aortocoronary Vein Grafts Studied 1 and 5 Years After Surgery.** *Circulation* 66(2, Part II): I-40—I-44, August 1982.

Smoking was not found to be directly associated with late narrowing of aortocoronary vein grafts. Repeat aortocoronary bypass graft angiography was performed in 34 patients at 13±5 months and 61±13 months after surgery. Progressive graft narrowing occurred in 15 of 34 patients (44 percent). Risk factors (incidence of smoking, diabetes mellitus, hypertension, and triglyceride and cholesterol levels) in these 15 patients were compared with those in the 19 patients whose grafts were unchanged between 1 and 5 years. The incidence of smoking, diabetes mellitus (fasting blood sugar ≥ 110 mg percent or 2-hour postprandial sugar ≥ 140 mg percent), and hypertension (systolic blood pressure >140 mmHg or diastoic pressure $\geq$ 95 mmHg) were 46 percent, 27 percent, and 33 percent, respectively, in the patients with progressive graft narrowing and 68 percent, 16 percent, and 26 percent in the patients whose grafts were unchanged. The average mean triglyceride levels were 617±785 mg percent (±SD) and 195±86 mg percent, respectively ($p<0.05$). The average mean cholesterol levels were 279±53 mg percent and 234±35 mg percent, respectively ($p<0.01$). In patients who have had aortocoronary bypass surgery, total graft occlusion is rare between 1 and 5 years after surgery. Progressive narrowing of grafts is common and appears to be associated with elevated blood lipids. (Auth. Abs. Mod.)

82-1697. Patrick, C. H.; Palesch, Y. Y.; Feinleib, M.; Brody, J. A. **Sex Differences in Declining Cohort Death Rates From Heart Disease.** *American Journal of Public Health* 72(2): 161-166, February 1982.

Changing trends in cigarette smoking may help account for the recent decline observed in heart disease (HD) mortality rates among American men. Cohort mortality from HD at ages 40 and over was examined for white men and women in the United States between 1945 and 1975. For each successive birth cohort from 1886 to 1890 and 1906 to 1910, female HD mortality rates exhibited a continuous decline with parallel slopes which showed no sign of abating in recent years. Among men, cohort HD mortality rates were increasing prior to 1965; since 1965, there has been a reversal of prior trends, i.e., each successive cohort has shown a decrease in HD mortality rates. The change in male patterns may come chiefly from a decrease in ischemic heart disease mortality. This development may be due to reduced cigarette smoking among men, although the exact impact of such a reduction is uncertain. (Auth. Abs. Mod.)

82-1698. Petersen, C. C. **Simulation of Alternative Designs of a Cardiovascular Risk Reduction Program for the U.S. Air Force.** *Journal of Medical Systems* 6(2): 149-164, April 1982.

Smoking, systolic blood pressure, and serum cholesterol are used as the prime cardiovascular disease risk factors in a screening and intervention program (HEART) being tested for implementation by the U.S. Air Force. The design and application of a simulation model to select the best system of organization and method of program delivery is described in detail. The model includes the effect of turnover of personnel, the effect of treatment on the therapy group, and the effect of aging on blood pressure and cholesterol level. The model determines how the overall incidence of cardiovascular disease in the U.S. Air Force may be expected to change each year through a 20-year period under each of several design policies. Results are given to justify the choice of several design options, and the scope of future improvements in the simulation is outlined. It appears possible to achieve a reduction of one-third in the calculated risk of cardiovascular disease, and this more than justifies the risk reduction program, based on a cost-effectiveness study performed earlier. The predicted percentage of smokers (60 percent at all age levels) was examined using various operating strategy options (therapy group size, screening, therapy treatment effectiveness). The results appear somewhat optimistic and ignore the fact that a smoker who does not stop smoking as a result of his first time in therapy is not likely to comply during subsequent stays in therapy. Other ways of specifying smoking compliance are being considered. All design decisions have not been made but will await the result of a demonstration implementation. The demonstration will produce more accurate data on initial distribution of risk factors and effect of therapy, which in turn will permit a sharpening of these preliminary analyses. (Auth. Abs. Mod.)

82-1699. Phillips, N. R.; Havel, R. J.; Kane, J. P. **Serum Apolipoprotein A-I Levels: Relationship to Lipoprotein Lipid Levels and Selected Demographic Variables.** *American Journal of Epidemiology* 116(2): 302-313, August 1982.

Serum apolipoprotein A-I (apo A-I) and high density lipoprotein (HDL) cholesterol were strongly correlated with one another in 289 subjects of northern California from 1974 to 1976. Both were inversely correlated with very low density lipoprotein (VLDL) triglycerides. Apo A-I level was unrelated to age, but increased with ethanol consumption and decreased with adiposity. An inverse relationship was found between apo A-I level and cigarette use. The correlation was only significant, however, among the women not taking exogenous sex steroids. Adjustment for body mass index and tobacco use had no effect on the association between apo A-I level and alcohol consumption. The decrease in HDL-cholesterol with increasing VLDL-triglycerides was relatively much larger than the concomitant decrease in apo A-I, cholesterol, and triglycerides, suggesting that the decreasing HDL-cholesterol:apo A-I ratio with increasing VLDL-triglycerides is due in large part to reciprocal transfer of cholesteryl esters for triglycerides between HDL and VLDL. Mean apo A-I level was 16 mg/dl higher in women not taking exogenous sex steroids than in men, and 31 mg/dl higher in women taking estrogens without progestins. (Auth. Abs. Mod.)

82-1700. Salonen, J. T. **Primary Prevention of Sudden Coronary Death: A Community-Based Program in North Karelia, Finland.** *Annals of the New York Academy of Sciences* 382: 423-437, 1982.

The North Karelia project was started in the county of North Karelia, Finland, in 1972 as a comprehensive community-based program to control cardiovascular diseases (CVDs). The main program objective was to reduce the population's CVD mortality and morbidity. Although middle-aged men were emphasized, the intermediate objectives were a reduction of smoking, serum cholesterol levels, and elevated blood pressure levels among the whole population. At program onset, smokers comprised 52 percent of the population; the mean serum cholesterol level was 269 mg/dl, and the mean blood pressure was 147/91 mm hg. A county-wide campaign to reduce or eliminate cigarette smoking in the population was launched in 1972. Smoking was banned in all public vehicles and public buildings. This action was supplemented by antismoking posters in public places, lectures in schools, and antismoking advertisements in newspapers and on radio and television. Special efforts were made to reduce smoking among health personnel, teachers, and among those individuals identified by screening programs as having CVD or being at high risk of developing CVD. Group therapy sessions for confirmed smokers were also established. The dietary program sought to reduce the total fat consumption in dairy products, sausages, and other high-fat meat and to encourage consumption of fresh vegetables, seldom seen in the North Karelian diet. Family vegetable plots were also recommended. A county-wide screening program to detect individuals with elevated arterial blood pressure was undertaken. Hypertensive persons were listed in a special register and usually placed on antihypertensive therapy. Current smokers decreased by 10 percent over the intervention period, and overall consumption also dropped considerably. Serum cholesterol levels diminished by a net of 16 percent among men and 9 percent among women. Net reduction in the prevalence of elevated blood pressure was substantial for both men (34 percent) and women (32 percent). Age-adjusted annual incidence of acute myocardial infarction decreased by 25 percent from 1973 to 1979 among men. No changes occurred among women, or in ischemic heart disease prevalence. During the followup period, an estimated reduction in coronary heart disease risk of 17 and 12 percent occurred among men and women, respectively. (Auth. Abs. Mod.)

82-1701. Salonen, J. T.; Puska, P.; Tuomilehto, J.; Homan, K. **Relation of Blood Pressure, Serum Lipids, and Smoking to the Risk of Cerebral Stroke. A Longitudinal Study in Eastern Finland.** *Stroke* 13(3): 327-333, May-June 1982.

The impact of blood pressure, serum cholesterol and triglycerides, and smoking on the risk of cerebral infarction and other stroke was studied by a longitudinal design. A random sample of the population aged 35-59 years in two counties of eastern Finland was examined in 1972, with a participation rate of 92 percent. In subjects (both sexes) standardized epidemiological measurements on blood pressure, height, weight, and serum lipids were made and questionnaire data were obtained on smoking. The cohort was followed for 7 years by means of national hospital discharge and death certificate registers. During the followup 77 men and 65 women had a cerebral stroke. Based on multiple logistic risk function analysis, age, smoking, blood pressure, and history of previous stroke and diabetes turned out to be independent predictors of both cerebral infarction and other strokes in men. (Auth. Abs. Mod.)

82-1702. Schlant, R. C.; Forman, S.; Stamler, J.; Canner, P. L. **The Natural History of Coronary Heart Disease: Prognostic Factors After Recovery From Myocardial Infarction in 2,789 Men. The 5-Year Findings of the Coronary Drug Project.** *Circulation* 66(2): 401-414, August 1982.

Findings of the Coronary Drug Project (CDP) on the long-term prognosis of men after recovery from clinically documented myocardial infarct (MI) suggest that lowering cholesterol and cessation of smoking can be beneficial even after recovery from one or more MIs. The natural history of coronary heart disease was studied in 2,789 men who had sustained at least one MI at least 3 months previously, and who were enrolled in the CDP between 1966 and 1969 and were allocated to receive placebo medication during a 5-year followup period. Forty characteristics determined at entry were selected for univariate and multivariate analysis, with four end points: death from all causes, sudden cardiovascular death, coronary death, and coronary death or definite nonfatal myocardial infarction. The baseline characteristics included demographic, historical, clinical, biochemical, and electrocardiographic data. The mean age at the time of enrollment was 52.4 years; the median interval since the last myocardial infarction 1.98 years. During 5 years of followup, 591 men died from all causes. The baseline characteristics that were statistically more related to subsequent mortality were those that reflected the amount of myocardial damage and loss of ventricular function. Baseline cholesterol and smoking were positively related to the risk of all four end points, whereas serum triglyceride was not related to 5-year prognosis in univariate analyses and was inversely related by multivariate analyses. There was a possible weak association between moderate or vigorous physical activity and decreased mortality during the next 5 years.

82-1703. Schoenenberger, J. C. **Smoking Change in Relation to Changes in Blood Pressure, Weight, and Cholesterol.** *Preventive Medicine* 11(4): 441-453, July 1982.

Quitters emerged as a group of men who demonstrated a positive response to intervention across multiple modalities in this study of the relationship of change in cigarette smoking status to change in body weight, systolic and diastolic blood pressure, and blood serum cholesterol levels during the first 3 years of the Multiple Risk Factor Intervention Trial (MRFIT). Over 4,000 of the 6,428 special intervention men were retrospectively categorized as hypertensive and nonhypertensive smokers, reducers, quitters, and nonsmokers. Comparisons among the groups of men were completed using analysis of covariance. The results indicated no evidence for a relationship of change in smoking status to change in systolic or diastolic blood pressure. Hypertension medication use and decreases in body weight were related to decreases in systolic and diastolic blood pressure. Body weight decreased significantly in smokers, nonsmokers, and reducers in contrast to quitters for whom the average weight change was minimal. For blood serum cholesterol, quitters and nonsmokers showed a greater reduction than smokers and reducers; and the expected positive relationship between change in body weight and serum cholesterol level occurred. (Auth. Abs. Mod.)

82-1704. Simons, L. A.; Jones, A. S. **Coronary Risk Factor Screening.** *Medical Journal of Australia* 1(6): 278-279, March 22, 1980.

A 3-year followup of subjects in the Sydney Coronary Heart Disease Prevention Programme (SCHDPP) revealed significant improvements of coronary risks as a result of physician intervention. At first screening, those with isolated risk factors were designated as controls, and those with multiple risks were referred to physicians and considered as under treatment. The smoking habits of the two groups were similar. Subjects who were successfully referred to general practitioners sustained significant improvement in the factors of hypertension, hyperlipidemia, and obesity, while control subjects not referred showed no significant change with respect to hyperlipidemia and obesity, and a lesser improvement in blood pressure. Both groups showed a slight decline in cigarette intake: 25 percent of the smokers were using fewer or no cigarettes. The extent of improvement observed could lead to an appreciable reduction in the risk of coronary heart disease. (Auth. Abs. Mod.)

82–1705. Sinzinger, H.; Kefalides, A. **Passive Smoking Severely Decreases Platelet Sensitivity to Antiaggregatory Prostaglandins. (Letter).** *Lancet* 2(8294): 392-393, August 14, 1982.

The risks of atherosclerosis from passive smoking were not mentioned in a previous editorial and correspondence (*Lancet* 1(8271): 548-549, March 6, 1982). Blood platelet sensitivity to the antiaggregatory prostaglandins (E_1, I_2, D_2) is a major determinant of hemostatic regulation and may be responsible for changes preceding atherosclerosis. Passive smoking's effects on platelet sensitivity were measured in 12 smokers and 9 nonsmokers, age 24 to 30, before, during, and after 15 minutes of exposure in a smoke-filled room. The baseline values were significantly higher in nonsmokers than in smokers, and reduction of sensitivity was more severe in nonsmokers. Platelet sensitivity began to return to normal values 20 minutes after exposure, and increased more rapidly in nonsmokers. This may be an important risk for nonsmokers exposed to smoke over long periods of time. See also 82-1070.

82–1706. Smith, T. **Chestnuts, Fats, and Fibre.** *British Medical Journal* 285(6335): 116-117, July 10, 1982.

A meeting on the science and strategy of coronary disease prevention produced some old "chestnuts" and the big news that Britain, since the mid-1970s, has joined Australia and the United States in showing a declining mortality from coronary heart disease (CHD) in blacks and whites, men and women, mostly in the 35-44 year age range, but also evident in the 45-54-year-olds; however, little change has occurred in those over 55. Though the change appears to follow a drop in the rate of fats and cigarette consumption, the decline in mortality is attributable mainly to changes in eating habits as has been shown by the relatively CHD-free Japanese, who despite heavy smoking, show that tobacco is no great threat to an otherwise healthy heart. The dietary key appears to be a lowered serum concentration of cholesterol with fiber not very helpful in lipid lowering, but with beta carotene a necessary factor for bowel cancer prevention when serum cholesterol is lowered. Though secondary to diet, smoking remains a factor in CHD mortality; however, smoking cessation remains a difficult task in spite of health education. Nicotine addicts may be treated with nicotine chewing gum, snuff, or possibly by a liquid preparation of pure nicotine for nasal inhalation.

82–1707. Stocksmeier, U. **Verhinderung des Rauchens als Teil Umfassender Praevention. [Smoking Prevention as a Part of Comprehensive Prevention Effort.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 36-49, German.

Cigarette smoke inhalation is discussed in a review of human health behaviors for which intervention measures are recommended. Because smoking is considered a primary risk factor for mortality and morbidity due to myocardial infarction and stroke, prevention for these diseases should begin at 10 to 13 years of age. Regular physical exercise is indicated as a means of countering the negative effects of smoking, including infarction and stroke. Also, a study of 1,200 patients revealed that more than half did not recognize their intensive cardiac problems until too late: thus public education is also recommended as a preventive step. The role of smoking in cardiovascular and other disease etiology is discussed in relation to stress. Smoking is judged to be more hazardous than momentary psychosocial stress because the reduction in plasma levels of "good cholesterol," combined with oxygen deprivation, may result in arteriosclerosis. Screening is also urged as a way to reduce mortality; research indicates that semiannual checkups could result in the lowest myocardial infarction incidence rates in Europe.

82–1708. Sturtevant, F. M. **Smoking, Oral Contraceptives, and Thromboembolic Disease.** *International Journal of Fertility* 27(1): 2-13, 1982.

After reviewing several studies it is concluded that little convincing evidence exists to indicate a serious risk of cardiovascular side effects from cigarette smoking and oral contraceptive (OC) use. Conclusions are presented in three broad categories: (1) In the case of deep vein thrombosis, the evidence is against cigarette smoking as a significant, positive risk factor in either users or nonusers of OCs. On the contrary, suggestive evidence exists for smoking as a negative, or at least a neutral factor, in certain groups of subjects. (2) For stroke, isolated studies have reported smoking and OC use to be risk factors for thrombotic stroke, but there is no indication of any interaction between the two. The evidence is somewhat stronger for implicating smoking in hemorrhagic stroke, while the evidence concerning OC use and possible interactive effects is weak. (3) In the case of ischemic heart disease, cigarette smoking appears to be a significant risk factor for myocardial infarction but OC use does not, when allowance is made for the presence of other risk factors such as age and smoking. Evidence for an interaction between smoking and OC use has been reported but appears weak. However, it is concluded that, for the present, OC users should forgo smoking cigarettes.

82–1709. Theorell, T.; Vigborg, A.; Kallner, A.; Dahlen, G.; Walinder, O. **A Comparison Between Finnish Immigrants and Native Swedes in the Greater Stockholm Region—Social and Medical Risk Indicators of Ischemic Heart Disease.** *Scandinavian Journal of Social Medicine* 8: 105-115, 1980.

Smoking was more common among Finnish immigrants to Sweden than among native Swedes in an analysis of environmental and genetic risk indicators of ischemic heart disease (IHD) in the two groups. A random sample of 71 male Finnish immigrants, aged 35 to 59, in the greater Stockholm region was compared with 77 age- and sex-matched native Swedes from the same area. Subjects responded to questions related to personal health (sickness), lifestyle (diet, tobacco and alcohol consumption), environment (living and job conditions), and family (marital status, number of children), and provided blood samples at a health examination. Smoking habits and high-density lipoprotein cholesterol were the "conventional" risk indicators that most strikingly differentiated the groups from one another. Significantly more native Swedes than immigrants had never smoked. Also, current cigarette smokers among the immigrants show a nonsignificant tendency to smoke more cigarettes/day than do the native Swedes who smoke.

82-1710. Turker, R. K.; Ercan, Z. S.; Ersoy, A.; Zengil, H. **Inhibition by Nicotine of the Vasodilator Effect of Bradykinin: Evidence for a Prostacyclin-Dependent Mechanism.** *Archives Internationales de Pharmacodynamie et de Therapie* 257(1): 94-103, May 1982.

In urethane-anesthetized rabbits, bradykinin caused a dose-dependent hypotension. This hypotensive response was not altered in magnitude but was inhibited in duration after pretreatment with nicotine. Nicotine failed to inhibit the hypotensive effect of eledoisine, prostacyclin, isoprenaline, and papaverine. Aspirin pretreatment, however, caused a marked inhibition in both magnitude and duration of the hypotensive effect of bradykinin without altering that of eledoisine, isoprenaline, papaverine, and prostacyclin. Bradykinin also produced a concentration-dependent fall in perfusion pressure of the isolated perfused guinea pig heart. Addition of nicotine to the perfusion medium caused a significant inhibition in both magnitude and duration of the vasodilator response to bradykinin. Nicotine did not alter the vasodilation induced by prostacyclin. Nicotine also failed to abolish the vasodilator effects of isoprenaline, histamine, papaverine, and eledoisine in the coronary circulation. Other antagonists such as atropine, propranolol, and cimetidine did not alter the vasodilator effect of bradykinin. These results were taken as the evidence indicating that the vasodilator action of bradykinin is mediated through the release of prostaglandins; the peptide selectivity increases the release of prostacyclin and this prostacyclin contributes to the bradykinin-induced vasodilation in guinea-pig coronary vessels. (Auth. Abs.)

82-1711. U.K. Department of Health and Social Security. **Prevention and Health: Avoiding Heart Attacks.** London, Her Majesty's Stationery Office, 1981, 69 pp.

Nearly every investigation into the cause of coronary heart disease has concluded that smoking is a powerful risk factor, particularly at younger ages. This overview of the size of the heart disease problem in the United Kingdom includes a definition of coronary disease, and means of avoiding it. Of every three men who are heavy smokers at age 40, one will have a major heart attack before age 65. Among nonsmokers, six out of seven will reach that age without an attack. At least two components of cigarette smoke, nicotine and carbon monoxide, damage the heart by depriving its tissues of oxygen. The risk of coronary disease increases with the amount smoked: it is detectable in those smoking 5 to 10 cigarettes per day, and may be 20 times as great in those smokers consuming over 40 per day. This risk is nearly eliminated 5 years after quitting. Filters do not remove carbon monoxide from smoke, and have no protective value where coronary disease is concerned. Also discussed are other heart disease risks: blood pressure, dietary factors, exercise, oral contraceptives, inherited and environmental influences, stress, and personality.

82-1712. Vajpayee, A.; Chadda, V. S.; Jain, N. C.; Mishra, S. N.; Sulemani, A. A.; Singhvi, D. R. **Serum Lipids and Smoking Habit in First Degree Relatives of Patients of Ischaemic Heart Disease.** *Indian Heart Journal* 33(6): 274-276, November-December 1981.

Serum cholesterol, triglyceride profile, and smoking habits of 81 first-degree relatives of 57 patients of documented ischemic heart disease (IHD) in the age group of 21 to 40 years were studied and compared with 30 healthy individuals. Both groups were divided into two subgroups of smokers and nonsmokers (smokers were defined as someone who smoked five or more cigarettes daily in the preceding 12 months). The diagnosis of hypercholesterolemia was made when total serum cholesterol was more than 260 mg percent and hypertriglyceridemia when serum triglyceride level was more than 150 mg percent. There was a significant difference in serum cholesterol levels between the relatives of IHD patients and controls ($P<0.01$) while serum triglyceride levels in the two groups were not significantly different ($P>0.05$). Smoking did not affect serum cholesterol and triglyceride significantly ($P>0.05$). Hypercholesterolemia was observed in 52.5 percent of smoker and 48.78 percent of nonsmoker relatives of IHD patients while only in 6.66 percent and 0 percent, respectively, of control individuals. Hypertriglyceridemia was present in 7.5 percent and 7.3 percent of smoker and nonsmoker relatives of IHD patients while in none of the control individuals. (Auth. Abs. Mod.)

82-1713. Voors, A. W.; Webber, L. S.; Berenson, G. S. **Resting Heart Rate and Pressure-Rate Product of Children in a Total Biracial Community. The Bogalusa Heart Study.** *American Journal of Epidemiology* 116(2): 276-286, August 1982.

Resting heart rates were ascertained during the 1978-1979 school year in 3,590 children aged 5-17 years in the biracial geographic population of Bogalusa, Louisiana. These rates were found to be relatively independent of the method of measurement, whether supine by physician's stethoscope or dressed sitting radial pulse taken by a nurse, and in either case adhering to a strict measurement protocol. Apart from the known influences of age and sex, a small but consistent racial influence was found, with whites having 3-4 beats/min higher rates than blacks. After controlling for age, heart rate correlated positively with blood pressure in whites and with subscapular skinfold thickness in boys. No consistent relation between heart rate and amount of cigarettes smoked was observed.

Reported smoking was negatively related to heart rate in girls, who showed a weak correlation with statistical significance. It is noted that the amount of smoking reported never reached the level of one pack a day. (Auth. Abs. Mod.)

82–1714. Wilhelmsen, L. **Risk Factors for Disease According to Population Studies in Goteborg, Sweden.** In: Bostrom, H.; Ljungstedt, N. (Editors). *Medical Aspects of Mortality Statistics.* Stockholm, Almqvist and Wiksell International, 1981, pp. 73-88.

Smoking is identified as a major risk factor for coronary heart disease (CHD) in Swedish morbidity and mortality studies of male myocardial infarct (MI) and stroke patients who were compared with general population samples. In addition, a primary prevention trial was conducted with 7,455 men, 47 to 54 years of age at entry to the trial in 1970, and 20,000 controls. Morbidity and mortality risk factors, particularly for CHD, which has accounted for slightly more than 35 percent of deaths to date, have been identified in followups of both groups. Mortality was found to be about three times higher among the nonparticipants in the prevention trial, and certain variables, including alcohol use, differed markedly between the two groups. The relative mortality among nonparticipants was even more evident when it was related to the risk increase associated with increased smoking, blood pressure, and serum cholesterol levels. For each combination of two of these risk factors, a low level of one apparently "protects" from the risk of high levels of the other. However, even moderately elevated levels of two or more of the factors are associated with high risk for MI or sudden death. Risk increases to high levels are more often due to such moderation of several factors than to great elevation of a single factor, a finding which has important implications for any planned CHD prevention programs.

82–1715. Wilhelmsen, L.; Wilhelmsson, C.; Vedin, A.; Elmfeldt, D. **Effects of Infarct Size, Smoking, Physical Activity and Some Psychological Factors on Prognosis After Myocardial Infarction.** *Advances in Cardiology* 29: 119-125, 1982.

The typical myocardial infarct (MI) patient is more often a smoker, and commonly has diabetes mellitus, aberrations in plasma lipoprotein patterns, and higher blood pressure. A review of long-term survival factors indicates about a 50 percent reduction in mortality among those who stop smoking compared to those who don't. These effects were seen as early as the second year after the acute infarction, and applied also to those who had suffered more than one MI. The short-term factor with strongest predictive capacity is the extent of myocardial damage, such as left heart failure, hypotension, and shock during the acute phase, arrhythmias, and a previous history of coronary heart disease. Physical training is probably of slight beneficial effect on survival prognosis, and definitely improves well-being and the psychological status of many patients. Stress and personality variables appear to play a role, but no conclusions can yet be drawn.

See also, **82–1618**

PREGNANCY AND INFANT HEALTH

82–1716. Amankwah, K. S.; Weberg, A. D. **Ultrastructural Changes in Preputial Neural Tissues: Effects of Maternal Smoking.** *Gynecologic and Obstetric Investigation* 13(3): 164-176, 1982.

Results of an examination of 14 newborns suggest that degenerative and regenerative processes are increased in the neural tissues of the fetal prepuce when the mother smoked during pregnancy. Prepuces from 10 newborns whose mothers smoked (≥ 20 cigarettes/day) throughout pregnancy and from 4 newborns of nonsmoking mothers were examined by transmission electron microscope. Significant ultrastructural changes were seen in the neural tissues of the newborns of smoking mothers. The ultrastructural changes observed were: (1) local aggregation of mitochondria, (2) many myelin bodies, and (3) dilated Golgi apparatus and dilated endoplasmic reticulum of the Schwann cell. Similar changes have been reported in neuropathies due to various drugs, chemicals, and deficiency states. (Auth. Abs. Mod.)

82–1717. Andresen, B. D.; Ng, K. J.; Iams, J. D.; Bianchine, J. R. **Cotinine in Amniotic Fluids From Passive Smokers. (Letter).** *Lancet* 1(8275): 791-792, April 3, 1982.

In commenting on the hazards of passive smoking, a study of cotinine in pregnant women is cited. The amniotic fluids of 24 nonsmoking women exposed to sidestream smoke at work or at home were examined for cotinine. Two samples were found with concentrations in the 5 to 10 ng/ml range, both from women whose husbands smoked and were home much of the time. A third sample had only trace elements: the amount of exposure was unknown. This suggests that prolonged exposure to sidestream smoke is necessary for appreciable concentrations of cotinine to be found in amniotic fluids. It is possible that the more acidic nature of amniotic fluid in the third trimester acts as an ion trap for tobacco smoke components.

82–1718. Carruth, B. R. **Smoking and Pregnancy Outcome of Adolescents.** *Journal of Adolescent Health Care* 2(2): 115-120, 1981.

Among 67 pregnant adolescents, smoking was not found to be significantly correlated with maternal weight gain, infant birth weight, and dietary inadequacy. The interrelationship between dietary intake, smoking behavior, and weight gain during pregnancy was studied and compared with pregnancy outcomes of infant birth weight and Apgar scores. Nonsmokers $(n=28)$ and smokers $(n=39)$ were volunteers attending secondary schools in an urban area. Both groups received prenatal and tertiary care at free or reduced fees and participated in free lunch and other assistance programs at their schools. Smoking was not statistically related to maternal weight gain, infant birth weight, dietary inadequacy, or Apgar scores, but was significantly related to low pregravid weight. An adequate diet, prenatal care, and maternal weight gain appeared to overcome the risk of smoking. (Auth. Abs. Mod.)

82–1719. Dalaker, K.; Sundfor, K.; Skuland, J. **Early Complications of Induced Abortion in Primigravidae.** *Annales Chirurgiae et Gynaecologiae* 70(6): 331-336, 1981.

The early postabortion complication rate in primigravidae is analyzed in relation to age, gestation length, abortion technique, and contraceptive practice after abortion. Of the 461 women who returned for followup, smoking data were obtained for 453, 67 percent of whom were smokers. There was a statistically significant difference between the smoking habits of women under 19 years and those over. The incidence of pelvic inflammatory disease after the abortion was highest in this group of patients who, in addition to having a high frequency of smokers, were characterized by low age of menarche and low rate of married or cohabit status.

82–1720. Gunasegaram, R.; Peh, K. L.; Loganath, A.; Karim, S.M.M.; Ratnam, S. S. **Inhibition of Second Trimester Human Placental Cholesterol C-20,22-Desmolase Activity by Soluble Constituents of Cigarette Smoke.** *IRCS Medical Science: Biochemistry* 10(4): 322-323, 1982.

The in vitro effect of cigarette smoke extract on the ability of C-20,22-desmolase to cleave 26-^{14}C cholesterol between C-20 and 22 to ^{14}C isocaproic acid, and presumably pregnenolone, was examined. Concentrations of ^{14}C isocaproic acid were measured in incubations of second trimester placental (obtained from nonsmoking mothers immediately after therapeutic abortion) homogenates with 26-^{14}C cholesterol and with or without serial dilutions of cigarette smoke extract (equivalent to 1/2, 1, 2, and 4 standard American filtered cigarettes) which were introduced to the test flasks throughout the series. A dose—dependent pattern of ^{14}C isocaproic acid formation was observed. The suppression of enzymic conversion of 4 dose levels was 29.1 percent to > 95.4 percent. Evaluation of this overall effect suggests that placental C-20,22-desmolase efficiency was slowed dramatically by increasing concentrations of chemical compounds present in the extract. Damped sensitivity to cytochrome P-450 of the cholesterol oxydase system could explain this effect. Clearly, the unusual sensitivity probably reflects an impairment in the supply of pregnenolone for the formation of progesterone and the synthesis of androgen sulphates that account for the bulk of estrogen production in normal pregnancy.

82–1721. Mau, G. **Rauchen Waehrend der Schwangerschaft—Risiken fuer das Kind. [Smoking During Pregnancy. Risks for the Child.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, 139-141, German.

An unborn child of a smoking woman is considered an involuntary passive smoker and, as such, is exposed to all potential risks from cigarette smoke. A lower than normal birth weight is the most frequent consequence of maternal smoking. This retardation of the fetal growth process is dose-dependent, i.e., depends on the number of cigarettes smoked. According to the most recent data, this defective development is attributed to the combined effects on fetal metabolism of carbon monoxide and nicotine in the mother's blood. In addition to smoking, maternal consumption of stimulants, particularly alcohol, undernourishment, and work and social stresses play an important role in that defective development of the fetus. Less evident is the effect of mother's smoking on perinatal mortality and malformations such as cleft lip, palatine cleft, and heart disease in the newborn. Small somatic and psychometric differences were found between smoke-exposed and nonexposed 11-year-old children. It is concluded that pregnant women must be convinced to stop smoking during pregnancy as cigarette smoking presents the most frequent hazard to a growing fetus.

82–1722. Paige, D. M.; Cordano, A.; Mellits, E. D.; Baertl, J. M.; Davis, L. **Nutritional Supplementation of Pregnant Adolescents.** *Journal of Adolescent Health Care* 1(4): 261-267, June 1981.

Statistically significant differences in birth weight were noted for the offspring of nonsmoking nutritionally supplemented pregnant adolescents compared with those of smoking nonsupplemented adolescents in a study of 157 pregnant black teenagers. Adolescents, all enrolled in a Baltimore public school for pregnant teenagers, were studied to determine the impact of a nutritional supplement on pregnancy outcome. Seventy-eight students (23 smokers) voluntarily agreed to receive a nutritional supplement; 79 comparably matched students (31 smokers) did not receive the supplement. The effects of smoking and nutritional supplementation were analyzed jointly. Within the smoking and nonsmoking groups, no significant differences in birth weight in supplemented and nonsupplemented women were noted. The mean birth weight of the 55 infants born to the nonsmoking supplemented teenagers was 3136.4 (± 431.9) g, whereas the mean birth weight of the 31 infants born to smoking nonsupplemented teenagers was 2907.8 (±546.2) g. (Auth. Abs. Mod.)

82–1723. Pelkonen, O.; Karki, N. T.; Tuimala, R. **A Relationship Between Cord Blood and Maternal Blood Lymphocytes and Term Placenta in the Induction of Aryl Hydrocarbon Hydroxylase Activity.** *Cancer Letters* 13(2): 103-110, July 1981.

Correlations between lymphocyte aryl hydrocarbon hydroxylase (AHH) inducibility in cord blood and in maternal lymphocytes and placental AHH activity were studied in 15 smokers and 11 nonsmokers. The mothers who smoked consumed 3 to 25 cigarettes daily, with an average of 11. Placental AHH activity was extremely low in the nonsmokers regardless of the lymphocyte AHH induction ratio, but was elevated to a variable extent in the smokers, in whom it showed a statistically significant correlation with cord blood lymphocyte AHH inducibility. The correlation between maternal lymphocyte AHH inducibility and placental AHH activity was not significant. These findings suggest that AHH induction in man may be "systemically" regulated and that the genetic background will determine the extent of induction at a given level of exposure to polycyclic aromatic hydrocarbons. (Auth. Abs. Mod.)

82–1724. Peters, M. A.; Ngan, L.L.E. **The Effects of Totigestational Exposure to Nicotine on Pre- and Postnatal Development in the Rat.** *Archives Internationales de Pharmacodynamie et de Therapie* 257(1): 155-167, May 1982.

Totigestational administration of low nicotine doses in female rats impaired behavioral performance among their offspring. Chronic administration of varying doses of nicotine (1.5, 3.0, and 6.0 mg/kg per day) to female Fischer 344 rats started 1 week prior to mating and continued until parturition. Because the group receiving 6.0 mg/kg had such an increased perinatal mortality rate, only data involving the 1.5 mg/kg and 3.0 mg/kg groups were used. An initial dose-related weight reduction was seen during the first week. However, following mating the weight gain of the females and the birth weight of the offspring were not different from controls. Treatment effects in the newborn were observed in the righting reflex, temperature regulation, adherence to the inclined screen, and in organ/body weight ratios for brain, heart, lung, liver, and kidney. In the adult female offspring treatment-related effects were seen as a prolonged time required and an increase in number of mistakes made during food maze testing and an increased brain protein content. Treated offspring required three learning sessions to reach the 50 percent shock avoidance level while control offspring required only two. Nicotine administered chronically in low doses throughout gestation has little effect on gross structural development, but does cause subtle neurological changes which are manifested as behavioral alterations in the newborn and adult offspring. (Auth. Abs. Mod.)

82–1725. Seshadri, R.; Baker, E.; Sutherland, G. R. **Sister-Chromatid Exchange (SCE) Analysis in Mothers Exposed to DNA-Damaging Agents and Their Newborn Infants.** *Mutation Research* 97(2): 139-146, April 1982.

Maternal smoking by itself did not increase the sister-chromatid exchange (SCE) rate in either mothers or their newborn infants. The relationship of parental smoking habits, alcohol consumption, possible exposure to DNA-damaging agents, and the incidence of SCE in the lymphocytes of mothers and their infants was analyzed. The results showed that the SCE rate in the newborns is significantly less than that of their mothers. The mothers in the smoking group (n=23) had an antenatal cigarette consumption ranging from 5 to over 15 cigarettes daily. No difference was found in the SCE rate between the control mothers and smoking mothers. Mothers who consumed alcohol (n=9) had a significantly increased SCE rate compared to control mothers. But the fact that all of the mothers in the alcohol group also smoked over 15 cigarettes daily complicated the findings. These maternal habits did not affect, however, the SCE rate of their infants. Neonates with neural tube defects showed a significantly increased SCE rate compared to normal babies. (Auth. Abs. Mod.)

82–1726. Stanhope, J. M.; Prior, I. M. **Alcohol, Smoking and Pregnancy Outcome Among New Zealand Maori.** *Australian Alcohol/Drug Review* 1(1): 74-76, January 1982.

An investigation of obstetric history among 333 Maori women revealed a relationship between smoking and miscarriage, and between alcohol consumption, miscarriage, and stillbirth. No evidence of an interaction between the two substances was found. Questionnaires were administered in 1962 and again in 1974 to Maori women from 15 to 44 years of age. Heavy smokers and heavy drinkers had more pregnancies and more miscarriages than expected; however, neither stillbirth nor child mortality was significantly related to smoking. When all smokers were compared to nonsmokers, the relative risks were significant for miscarriage and stillbirths but not for child death. Pooling the data into consumption categories abolished the relationships between number of pregnancies and drinking or smoking, but confirmed the original correlations between smoking, drinking, miscarriage, and stillbirth.

See also, **82–1658**

OTHER DISEASES AND CONDITIONS

82-1727. Adena, M. A.; Gallagher, H. G. **Cigarette Smoking and the Age at Menopause.** *Annals of Human Biology* 9(2): 121-130, 1982.

In a sample of 15,464 apparently healthy Australian women, the mean age at natural menopause of women who smoke 10 or more cigarettes a day was 1-3 years lower than that of other women. These distributions were well described by logistic distributions with the same variances. A parallel nonparametric analysis confirmed that heavy smokers reached menopause earlier than other women, and also showed that the women accurately recalled their age at menopause. Logistic regression analyses were used to show that obesity, alcohol intake, and regular use of aspirin and other analgesics, sedatives, tranquillizers, or antidepressants did not affect the distribution of the age at natural menopause. The age-specific probability of natural compared with surgically induced menopause was shown to be independent of smoking history and obesity. (Auth. Abs. Mod.)

82-1728. Alvesalo, I.; Reisine, S.; Hay, J.; Bailit, H. L. **Effects of Fluoride and Regular Dental Care on Personal Dental Expenditures of Young Adults in Finland.** *Community Dentistry and Oral Epidemiology* 10(1): 15-22, February 1982.

An analysis of the smoking habits of 555 adults in Finland confirms earlier findings of a significant and independent effect for smoking behavior on oral health status. The relationship among presence of fluoride in the drinking water, oral health, and dental expenditures in four towns in Finland was analyzed. A two-stage least-squares model was used to determine the effect of fluoridated water and other independent variables on the number of missing teeth and per capita annual dental service expenditures. The results show that regularity of dental visits is the only significant factor explaining per capita expenditures. In contrast, many factors affect the number of missing teeth; most importantly, the number of dentists per capita, level of fluoride, age, education, use of services, and smoking habits. It is suggested that smoking may affect tooth loss by way of inefficient toothbrushing. The toothbrushing variable in the regression is not significant but this finding is probably due to the measure's insensitivity to plaque removal efficiency. Further study with a more precise measure of toothbrushing effectiveness in the context of smoking behavior is recommended. (Auth. Abs. Mod.)

82-1729. Anttonen, T.; Tenovuo, J. **Crevicular Thiocyanate and Iodide Ions: Cofactors of the Antimicrobial Peroxidase System in Leucocytes.** *Proceedings of the Finnish Dental Society* 77(6): 318-323, 1981.

The presence of thiocyanate (SCN^-) and iodide (I^-) ions, the auxiliary cofactors of peroxidase-mediated antimicrobial reactions in human crevicular fluid was studied in 26 (13 males, 13 females, mean age 28.3 years, 50 percent smokers) subjects. Average crevicular concentration of SCN^- and I^- were 37 ± 24 and 4.3 ± 1.9 μM, respectively. Comparison between salivary and crevicular concentrations showed that the crevicular amount of SCN^- was only about 2.5 percent of the respective concentration in whole saliva, while in the case of I^- the corresponding value was about 40 percent. No changes in crevicular I^- were found in relation to gingivitis or smoking whereas the crevicular SCN^- concentration increased slightly as a function of inflammation and smoking. The results indicate that antibacterial cofactors of leukocyte peroxidases exist in crevicular fluid. Earlier studies have shown that peroxidases, most probably of leukocytic origin, are present in crevicular fluid. In conclusion, it seems that a peroxidase-mediated antimicrobial system can control the growth of bacteria in gingival sulcus. (Auth. Abs. Mod.)

82-1730. Bauchinger, M.; Hauf, R.; Schmid, E.; Dresp, J. **Analysis of Structural Chromosome Changes and SCE After Occupational Long-Term Exposure to Electric and Magnetic Fields From 380 kV-Systems.** *Radiation and Environmental Biophysics* 19(4): 235-238, 1981.

Smoking status made no significant difference in the frequency of sister-chromatid exchanges (SCEs) among workers exposed to electric and magnetic fields. Chromosome analyses were carried out in lymphocytes of 32 workers occupationally exposed for more than 20 years to 50 Hz alternating electric and magnetic fields in 380 kV switchyards. The exposed workers comprised 12 moderate smokers (< 10 cigarettes/day) or heavy smokers, 4 who had quit within the last 4 years, and 16 nonsmokers. As compared with a control group of 22 workers (10 smokers, 12 nonsmokers) of similar age and occupation but without field exposure, neither the yields of structural chromosome changes nor the SCE-frequencies were increased. Parameter-free analysis of variance was used to compare the SCE frequencies in the control group and field-exposed group taking into account a subdivision in smokers and nonsmokers. No significant differences could be demonstrated. (Auth. Abs. Mod.)

82-1731. Chujoh, C.; Nakazawa, S. **Inenmaku Shogai ni Kansuru Jikkenteki Kenkyu Toku ni Sakusan Kaiyo no Chiyu ni Oyobosu Kitsuen no Eikyo ni Tsuite. [Experimental Study for the Gastric Mucosal Damage.]** *Japanese Journal of Gastroenterology* 78(12): 2285-2294, December 1981, Japanese.

To elucidate the effect of cigarette smoking on the gastric mucosa, cigarette smoke or subcutaneous nicotine was administered to rats bearing an experimental acetic acid ulcer of the stomach. Microangiography was performed to measure the diameter of the gastric mucosal vessels. In all groups which were loaded with cigarette smoke, the healing course of the acetic acid ulcer was prolonged. A similar result was shown in the groups which were given subcutaneous injection of nicotine (250 μg/kg or 500 μg/kg). The results of microangiographic study disclosed that the diameter of metarteriole, precapillary, and capillary blood vessels decreased after cigarette smoking as well as nicotine administration, and this contraction persisted for 60 min. It is concluded that cigarette smoking exerts an ill influence upon the healing course of the gastric ulcer, and this effect is hypothesized to be due to the reduction of the gastric mucosal blood flow. (Auth. Abs. Mod.)

82–1732. Dale, B.; de Santis, A.; Hagstrom, B. E. **The Effect of Nicotine on Sperm-Egg Interaction in the Sea Urchin: Polyspermy and Electrical Events.** *Gamete Research* 5(2): 125-135, 1982.

Some of the effects of nicotine on sea urchin eggs, spermatozoa, and their interaction were studied using electrical recording techniques and fertilization-rate experiments. Pretreating eggs with nicotine enhanced the fertilization rate, whereas this drug had an inhibitory effect on spermatozoa. Pulse-treated eggs or eggs fertilized in the presence of nicotine gave rise to attenuated step depolarizations, which may be attributed to a decrease in membrane resistance (Rm) of the egg or, in the latter case, to an alteration to the spermatozoon. Concurrently, with the change in the step depolarization there was a reduction in amplitude of the fertilization potential (FP) suggesting that the cortical reaction is in some way altered. Nicotine had no effect on the Rm of fertilized eggs or oocytes, where there are no cortical granules. It is suggested that nicotine alters the cortex of sea urchin eggs—possibly by causing a partial dissolution of cortical granules—which renders the eggs more receptive to spermatozoa. The reductions in amplitude of the step depolarization and the FP are consequences of this alteration. (Auth. Abs.)

82–1733. Fung, W. P.; Mahoney, D. P.; Beilin, L. J. **Effect of Cigarette Smoking on Gastric Secretion of 6-Keto Prostaglandin $F_{1\alpha}$.** *Australian and New Zealand Journal of Medicine* 12(2): 206-208, April 1982.

Smoking does not appear to promote peptic ulceration by inhibition of prostacyclin secretion. The effect of cigarette smoking on gastric secretion of 6-keto prostaglandin $F_{1\alpha}$ was studied in 31 volunteers, 18 of whom smoked three cigarettes after basal collection of gastric juice. The output of 6-keto $PGF_{1\alpha}$ was unaffected by smoking three cigarettes in 1 hour. Furthermore, basal levels of 6-keto $PGF_{1\alpha}$ prior to smoking or "control" were similar in results between smokers and nonsmokers. Cigarette smoking has been associated with delayed healing of peptic ulcers during treatment with antacids or placebo. However, these results do not support the hypothesis that smoking may predispose to the development of peptic ulcer by suppression of gastric prostacyclin secretion. (Auth. Abs. Mod.)

82–1734. Godwin-Austen, R. B.; Lee, P. N.; Marmot, M. G.; Stern, G. M. **Smoking and Parkinson's Disease.** *Journal of Neurology, Neurosurgery and Psychiatry* 45(7): 577-581, July 1982.

In a case—control study of the relationship between smoking habits and Parkinson's disease a negative association was demonstrated with a relative risk of 0.52. A history of smoking up to 20 years earlier was associated with a risk of developing Parkinson's disease equal to about half that in nonsmokers. The type of disease, age of onset, and rate of progression were associated with a similar reduction in risk implying that in respect of smoking history the disease is homogeneous. The positive correlation of degenerative vascular disease with smoking is further evidence that arteriosclerosis is not involved in the causation of Parkinson's disease. The negative association between Parkinson's disease and smoking is confirmed and is independent of other associated factors. The known and serious risks of smoking far outweigh the possible benefit of lowered risk of Parkinson's disease. (Auth. Abs.)

82–1735. Gray, R. S.; Starkey, I. R.; Rainbow, S.; Kurtz, A. B.; Abdel-Khalik, A.; Urbaniak, S.; Elton, R. A.; Duncan, L.J.P.; Clarke, B. F. **HLA Antigens and Other Risk Factors in the Development of Retinopathy in Type 1 Diabetes.** *British Journal of Ophthalmology* 66(5): 280-285, May 1982.

Smoking was more prevalent among type 1 diabetics with diabetic retinopathy than those with no or minimal retinopathy. Factors possibly influencing the development of diabetic retinopathy were studied in 112 randomly selected type 1 diabetics having no or minimal retinopathy (group A) and in 82 type 1 diabetics with known severe diabetic retinopathy. The latter comprised those with severe background retinopathy (group B, $n=17$) and those having proliferative retinopathy without (group C, $n=38$) and with (group D, $n=27$) diabetic nephropathy. Nonretinopaths (group A) were of similar sex ratio, body weight, and age at diagnosis of diabetes but had been diabetic longer and were thus older than retinopaths (groups B-D). The distribution of histocompatibility antigens (HLA antigens) of the A, B, and C loci was similar in nonretinopaths and retinopaths with the exception that HLA B7 showed a reduced prevalence in the retinopaths (6 percent vs. 17 percent), and was singularly underrepresented in group D, where no patients had this antigen. Mean postprandial plasma glucose and HbA_1 concentrations were higher and cigarette smoking was more prevalent in the retinopathy groups B-D than in Group A. Although retinopaths (groups B-D) included more ($p<0.01$) smokers (60 percent) than nonretinopaths (38 percent), this difference was mainly accounted for by the high prevalence of smoking in group D (69 percent), being significantly greater than in the other three groups of diabetics (44 percent). It is concluded that the risk of development of diabetic retinopathy in type 1 diabetes may be related to HLA-associated genetic factors and to cigarette smoking. (Auth. Abs. Mod.)

82–1736. Gregory, J. **Office Air Quality, "Tight Buildings," and Job Stress—The Impact on Women Office Workers' Occupational Health.** Presented at a conference of the American Industrial Hygiene Association/American Conference of Government Industrial Hygienists, Portland, Oregon, May 25, 1981, 11 pp.

Problems with office air quality and job stress emerged as the complaint most frequently cited by 960 respondents to a Working Women survey. Clerical workers, 80 percent of whom are women, typically work in areas where office air is the worst. Nonsmoking office workers are often exposed to the equivalent of 10 cigarettes per day. The level of particulate matter in office buildings where smoking is allowed is 10 to 100 times higher than permissible limits for outside air. Smoke suspends other airborne contaminants, makes it harder and more costly to remove indoor pollutants, and compounds the effects of many other contaminants. Complaints of irritating fumes were reported by 25 percent (244) of the respondents, involving fumes from photocopying or laboratory equipment and chemically treated paper, as well as smoke. Recent building trends which contribute to the passive smoking problem include lowered ventilation and circulation to cut energy costs, sealed "weatherless" offices, faulty ventilation and humidifying systems which recycle smoke and other contaminants, overcrowding, and lack of no-smoking areas. It is noted that the comments on smoke were write-ins; a much higher response would have been expected if the category had been present on the questionnaire. (Auth. Abs. Mod.)

82–1737. Gupta, P. K.; Frost, J. K. **Cytologic Changes Associated With Asbestos Exposure.** *Seminars in Oncology* 8(3): 283-289, September 1981.

Cigarette-smoking subjects with asbestos exposure show more marked bronchial epithelial atypia, suggesting that there may be an additive effect. Ferruginous bodies may be seen in the sputum but can not be readily associated with asbestos exposure since many with known exposure do not demonstrate these bodies while some smokers without known asbestos exposure do have ferruginous bodies seen in their sputum. Further correlation efforts are, therefore, needed to determine if the "false positives" had an unknowing exposure to asbestos and whether there is a dose or specific fiber type which leads to ferruginous bodies. These findings would support the epidemiologic observations that smoking and asbestos exposure are associated with a higher incidence of bronchogenic carcinoma. (Auth. Abs. Mod.)

82–1738. Heckman, C. A.; Dalbey, W. E. **Pathogenesis of Lesions Induced in Rat Lung by Chronic Tobacco Smoke Inhalation.** *Journal of the National Cancer Institute* 69(1): 117-129, July 1982.

Lesions were induced in the lungs of specific-pathogen-free F344 rats by chronic tobacco smoke exposure. Animals exposed to seven cigarettes/day were killed after 1, 1.5, or 2 years of exposure. Parallel lifetime exposures induced pulmonary tumors in 9 percent of the animals. In serially killed animals, four types of lesions were found: (1) perivascular or peribronchiolar accumulation of lymphoreticular cells, (2) fibrotic and cellular enlargement of peribronchiolar septa, (3) type II cell hyperplasia with septal fibrosis, and (4) air-space enlargement (emphysema). However, emphysema occurred only in animals exposed to a higher (10 cigarettes) dose of tobacco smoke. Ultrastructural studies showed all of the focal lesions to be infiltrated by cells typical of the inflammatory response. The type II hyperplastic and peribronchiolar alveolar lesions involved larger portions of the parenchyma in fibrotic changes but differed in structure, location, and frequency. The incidence of the peribronchiolar alveolar lesions was temporally related to tumor incidence. (Auth. Abs.)

82–1739. Hirsch, J.-M., Thilander, H. **Snuff-Induced Lesions of the Oral Mucosa—An Experimental Model in the Rat.** *Journal of Oral Pathology* 10(5): 342-353, October 1981.

An experimental white rat model, developed to study the influence of snuff on oral mucosa, is described. A test canal in the lower lip, with one orifice buccally to the incisors and one on the lip side, was created by surgical means. The connection between the canal and the oral cavity was made to ensure the presence of saliva in the canal so that physiological conditions resembling those of the oral cavity were obtained. The canal was filled with snuff morning and night 5 days a week. The mean value for the maximal retention time of the snuff was 6 hours. The total quantity of snuff administered to the rats daily was calculated to be 1 g/kg body weight. This is about five times greater than the corresponding dose in humans. The animals tolerated the dose and time of exposure without signs of severe toxic symptoms. However, the test animals did show an increase in activity compared to control rats, following each snuff injection and throughout the test period. Histological examination of the canals after 9 months of exposure to snuff showed a mildly to moderately hyperplastic epithelium with hyperorthokeratosis. Locally deep proliferations of epithelium with acanthotic rete pegs could be seen. In the stratum basale hyperplasia with disturbed polarity and hyperchromatic nuclei and single mitosis were noted. It is concluded that the model is well suited for further studies of the factors which influence and modify the pathogenesis of snuff lesions. (Auth. Abs. Mod.)

82–1740. Ibrahim, A. S.; Fatt-Hi, A. S. **Cigarette Smoking and Hearing Loss.** *World Smoking and Health* 7(2): 42-45, Summer 1982.

Smoking was found to increase the incidence and severity of both conductive and perceptive deafness in a comparison of hearing loss in 150 smokers and 150 nonsmokers. Smokers and nonsmokers were matched by age, educational level, and social class. Average hearing loss in smokers (25.3 percent) was significantly higher than in nonsmokers (8.8 percent). Hearing loss was significantly correlated with age in both groups and with duration of smoking among smokers. Also, duration of smoking was correlated with age. Therefore, a partial correlation analysis was done which showed that duration of smoking had more of an effect on hearing loss than, and was independent of, age.

82-1741. Klemp, P.; Staberg, B.; Madsbad, S.; Kolendorf, K. **Smoking Reduces Insulin Absorption From Subcutaneous Tissue.** *British Medical Journal* 284(6311): 237, January 23, 1982.

Smoking contributes to metabolic instability in insulin-dependent patients by influencing the rate of absorption. In nine ketosis-prone, insulin-dependent diabetics, an average decrease of 113 percent occurred in insulin absorption during smoking, and a decrease of 31 percent was found 30 minutes after smoking. Diabetic patients who smoke have been shown to need 15 to 20 percent more insulin than nonsmokers, and heavy smokers may need up to 30 percent more. Smoking both increases the concentration of catecholamines in the blood, and causes peripheral vasoconstriction, possible mechanisms for interfering with insulin absorption.

82-1742. Korman, M. G.; Hansky, J.; Merrett, A. C.; Schmidt, G. T. **Ranitidine in Duodenal Ulcer. Incidence of Healing and Effect of Smoking.** *Digestive Diseases and Sciences* 27(8): 712-715, August 1982.

Smoking had a significantly adverse effect on the healing rate of duodenal ulcer achieved with ranitidine. Fifty patients with endoscopically proven duodenal ulcer were randomly assigned to ranitidine 150 mg twice daily or placebo for 4 weeks. Endoscopic examination at this time showed that 20 of 25 patients (80 percent) on ranitidine healed compared to 4 of 25 patients (16 percent) on placebo. Smoking adversely affected the incidence of healing: 70 percent of nonsmokers healed compared to 30 percent of smokers. Additionally, the number of cigarettes smoked did not correspond with healing or failure to heal. Review at 6 months after cessation of therapy showed relapse of duodenal ulcer in 10 of 20 patients (50 percent) healed with ranitidine and 1 of 4 patients (25 percent) healed with placebo. Thus, ranitidine (300 mg/day) produces similar healing rates to those reported for cimetidine (1,000 mg/day); also like cimetidine, the incidence of healing on ranitidine is adversely influenced by smoking and the relapse rate on cessation of therapy is high. (Auth. Abs. Mod.)

82-1743. Lasnitzki, I.; Bollag, W. **Prevention and Reversal by a Retinoid of 3,4-Benzpyrene- and Cigarette Smoke Condensate-Induced Hyperplasia and Metaplasia of Rodent Respiratory Epithelia in Organ Culture.** *Cancer Treatment Reports* 66(6): 1375-1380, June 1982.

An aromatic analog of vitamin A, etretinate, inhibited or reversed the effects of 3,4-benzpyrene and cigarette smoke condensate in fetal mouse lung and neonatal rat tracheas grown in organ culture. In both tissues, 3,4-benzpyrene as well as cigarette smoke condensate induced a striking increase of epithelial mitosis within 12-14 days of treatment. The increase was associated with a loss of secretory activity and of ciliary function. These changes persisted in the absence of benzpyrene or smoke condensate in explants transferred to control medium. Treatment with etretinate alone did not affect the normal epithelial growth rate or normal differentiation. If combined with either benzpyrene or smoke condensate, the aromatic compound inhibited the increase in cell division and prevented the loss of secretory activity or ciliary function. In explants pretreated with 3,4-benzpyrene or cigarette smoke condensate, etretinate reduced the carcinogen- or smoke condensate-induced increase in mitotic activity to normal levels and restored secretory differentiation and ciliary function. The mechanism of action involved in the anticarcinogenic activity of the retinoid is not yet fully understood. (Auth. Abs. Mod.)

82-1744. McLean Ross, A. H.; Smith, M. A.; Anderson, J. R.; Small, W. P. **Late Mortality After Surgery for Peptic Ulcer.** *New England Journal of Medicine* 307(9): 519-522, August 26, 1982.

Excess mortality among male patients who had successfully undergone a curative peptic ulcer operation is attributed to smoking-associated diseases. The records of 779 patients (83 percent smokers) who had undergone ulcer surgery between 1947 and 1965 were reviewed to determine the mortality rate and causes of death. By the end of 1979, 360 patients had died. There was an excess mortality in the three major age groups (30 to 39, 40 to 49, and 50 to 59 years at operation), as compared with the general population, with a mean shift of 9.1 years in the survival curve. Surgery was not the direct cause of death. In particular, the eight deaths from carcinoma of the stomach were no more than expected. Mortality due to smoking-associated disease accounted for 200 deaths. It is concluded that substituting highly selective vagotomy for gastrectomy will not lead to improved survival, because 80 percent of patients now having ulcer operations are heavy cigarette smokers. (Auth. Abs. Mod.)

82-1745. Portheine, F. **Blutuntersuchungen bei Rauchern im Rahmen der Krebsfrueherkennung (Tumorantigen CEA). [Blood Tests in Smokers in the Framework of Early Detection of Cancer (Carcinoembryonic Antigen CEA).]** In: Deutsche Haupstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 134-138, German.

Preliminary results of a blood test for carcinoembryonic antigen (CEA) as an early diagnostic method for lung cancer are reported. The tumor antigen of the CEA type had been determined in blood of over 1,000 smokers and its evolution had been followed up in regular smokers and ex-smokers. In agreement with other research, 3 to 5 ng of CEA/ml of blood was found in 22 percent, 5.1 to 10.0 ng/ml in 9 percent, and 10.1 to 20.0 ng/ml in about 1 percent of heavy smokers. The normal CEA content in blood of healthy persons is less than 2.5 ng/ml. This CEA distribution pattern is interpreted as indicative of a precancerous state in smokers. In a high proportion of smokers, the CEA content slowly decreased over a several month period after total abstinence from smoking. These research findings should encourage even heavy smokers to quit, in recognition of the fact that 5-year survival after operation is achieved in only 20 to 25 percent of lung cancer patients, if detected early enough.

82–1746. Rodriguez-Rigau, L. J.; Smith, K. D.; Steinberger, E. **Cigarette Smoking and Semen Quality.** *Fertility and Sterility* 38(1): 115-116, July 1982.

No association between cigarette smoking and compromised semen quality was found in the analysis of semen from 159 male partners of infertile couples screened for confounding factors (101 nonsmokers and 58 cigarette smokers, average age approximately 31 years). Of the smokers, 24 smoked <20 cigarettes/day, and 34 smoked >20 cigarettes/day. Semen analysis indicated 44.7 percent and 44.8 percent average percentage of normal spermatozoa for nonsmokers and cigarette smokers, respectively. Average sperm mobility, mean sperm count, and frequency distribution of sperm count also were not statistically different for smokers and nonsmokers. Subdivision of smokers into groups who smoked <20 cigarettes/day and >20 cigarettes/day produced similar results. A parallel investigation of 97 men with varicocele found no significant differences in the semen quality of 59 nonsmokers compared with 38 cigarette smokers. When compared with the 159 men without varicocele, a significantly higher percentage of men with varicocele had sperm counts of <20 million/ml.

82–1747. Shephard, R. J. **The Risks of Passive Smoking.** New York, Oxford University Press, 1982, 195 pp.

A simple but critical examination of the risks of passive smoking, as perceived in the energy-deficient 1980s, is presented. The key constituents of tobacco smoke are noted, and the pollutant yield is discussed for various species of such smoke. Observations are related to epidemiological information on the levels of key pollutants observed in public places. Carbon monoxide accumulation is discussed in relation to possible disturbances of psychomotor function and exacerbation of cardiac disease. The acute effects of smoke upon the respiratory tract and on other body systems are considered, and long-term disturbances of health are reviewed in relation to both the general population and sensitive target groups (young children, asthmatics, and the elderly). Problems encountered by the fetus and the infant born to a smoking mother are also explored. Final chapters discuss the current attitudes of smokers and nonsmokers to the issue of passive smoking, along with possible remedies that will afford a greater measure of protection to the nonsmoker. (Auth. Abs.)

82–1748. Sonnenberg, A.; Huesmert, N. **Effect of Nicotine on Gastric Mucosal Blood Flow and Acid Secretion.** *Gut* 23(6): 532-535, June 1982.

The effects of an intravenous infusion of nicotine at a dose of 2.5, 5.0, 7.5, or 10.0 $\mu g\ kg^{-1}h^{-1}$ on pentagastrin-stimulated gastric mucosal blood flow and acid secretion were investigated in eight healthy male nonsmokers. Each dose was tested in two males. Gastric neutral red clearance served as a measure of mucosal blood flow. Nicotine reduced volume secretion, acid secretion, and neutral red clearance in a dose-dependent manner. In five healthy male smokers, smoking five cigarettes in 2 hrs induced similar changes to the intravenous infusion of 5 $\mu g\ kg^{-1}h^{-1}$ nicotine. As volume secretion was inhibited more than neutral red clearance, it is concluded that nicotine increases blood supply to the gastric mucosa relative to the reduced gastric secretion. Nicotine is either not associated with the development of peptic ulcers, or it exerts its ulcerogenic action via other mechanisms than change of acid secretion and gastric mucosal blood flow. (Auth. Abs.)

82–1749. Wheeler, L. A.; Saperstein, M. D.; Lowe, N. J. **Mutagenicity of Urine From Psoriatic Patients Undergoing Treatment With Coal Tar and Ultraviolet Light.** *Journal of Investigative Dermatology* 77(2): 181-185, August 1981.

The possible percutaneous absorption of mutagens from patients receiving crude coal tar (CCT) and ultraviolet light was investigated. Urine samples were collected from nonsmoking volunteers, and smoking and nonsmoking psoriatic patients. Patients were treated with 1 percent CCT United States Pharmacopeia or 1 to 10 percent CCT in petrolatum in the evening. The following morning, patients received coal tar baths and then ultraviolet light (mainly 290-320 nm). Nonpolar organics in urine samples were extracted by adsorption onto XAD-2 resin and the extracted organics assayed in the Ames *Salmonella*/Microsome test. TA98 was the most sensitive bacterial strain to detect mutagenicity. Except for smoking psoriatic patients, the addition of liver homogenate was necessary to see mutagenicity. No increase in the number of revertants was observed when B-glucuronidase was added to the assay. Of 14 patients studied, 12 had at least one mutagenic urine sample. Typical values for nonsmoking psoriatics treated with CCT ranged from 42 to 496 his^+/20 ml of urine after the subtraction of spontaneous his^+ counts (26 ± 6). Two nonsmoking normal volunteers were found to excrete mutagenic urines. Smoking psoriatic patients ranged from 213 to 1,100 his^+/20 ml urine. This study demonstrates the percutaneous absorption of mutagens from CCT and indicates that its effects may not be limited to the skin. (Auth. Abs.)

82–1750. World Health Organization. **Health Aspects Related to Indoor Air Quality.** World Health Organization, Regional Office for Europe, Copenhagen, EURO Reports and Studies 21, 1979, 32 pp.

Tobacco smoke is the major and most common source of indoor contaminants. Smoking results in high levels of carbon monoxide, aldehydes, nitrogen dioxide, and various particulates in indoor air. Other sources of indoor pollution include outdoor air, since there is continuous air exchange with the exterior. Contaminants associated with the building involve construction, sealing, and fireproofing materials such as formaldehyde, radon, and asbestos. Human activity such as smoking, cooking, and hobbies add carbon oxides, nitrogen oxides, water vapor, and compounds from consumer products. Special environments, such as airplanes, trains, buses, cars, terminals, and public buildings, where smoking is a serious contaminant, are also discussed. In most societies, people spend an average of 71 percent of their time indoors. Adoption of current energy-saving proposals whereby the present minimum ventilation rates in building codes would be lowered, eventually becoming maximum rates, will aggravate the existing problems in indoor air quality. As the major threat to indoor air quality, it is recommended that smoking be curtailed if ventilation rates are to be reduced.

82–1751. Yesner, R. **The Effect of Tobacco Smoke on the Non-Smoker.** *Connecticut Medicine* 45(7): 470-471, July 1981.

The effects of sidestream smoke on the passive smoker are both acute and chronic. Cough, red eyes, asthmatic attacks, conjunctivitis, and respiratory distress are well-known. Even Raynaud's phenomenon has been reported in passive smokers. Smoking is the major source of ambient carbon monoxide: the carboxyhemoglobin level in exposed nonsmokers averages 1.2 percent. Electrocardiogram changes occurred in 15 dogs passively exposed to smoke, and some showed scarring of the myocardium. Rabbits exposed to smoke and kept on a high-fat diet had atheromatosis far more developed than controls: some of the animals displayed necrosis and subendocardial hemorrhages. Passive smoking increases the risk of respiratory illness, and reduces small airways function in children of smoking parents and in those exposed occupationally for more than 20 years. A study in Japan showed that nonsmoking wives of smoking husbands develop lung cancer 4.6 times as often as wives of nonsmokers.

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

82–1752. Benwell, M.E.M.; Balfour, D.J.K. **Effects of Chronic Nicotine Administration on the Response and Adaptation to Stress.** *Psychopharmacology* 76(2): 160-162, 1982.

The effects of chronic nicotine administration (0.4 mg/kg for 40 days) and its withdrawal on the adrenocortical response to acute and repeated exposure to stress have been examined and related to changes in brain 5-hydroxyindole levels. No significant effects on the response to acute stress were observed. Repeated exposure to the stressful procedure resulted in complete adaptation of the adrenocortical response and the development of a significant positive correlation between the plasma corticosterone and hippocampal 5-HT concentrations. In nicotine-treated rats, complete adaptation did not occur and the plasma corticosterone showed a significant negative correlation with hippocampal 5-HT. Nicotine withdrawal was not associated with any reduction in plasma corticosterone, but did abolish its relationship with hippocampal 5-HT. (Auth. Abs.)

82–1753. Biener, K. **Motivationen zum Tabakkonsum der Jugend. [Motivation for Smoking by Youth.]** In: Deutsche Haupstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 96-106, German.

Results of a survey of 2,656 male and female teenage apprentices in large industries of North Switzerland show that most smokers are motivated by pleasure and taste, while 26 percent of the older male smokers from urban areas are motivated by habit or addiction. In contrast to smokers, all nonsmokers indicated a concrete motive for not smoking, such as a reduction in sports performance, given as motivation by about one quarter of the older male apprentices from urban areas. About one half of all answering teenagers did not perceive any advantage from smoking. In contrast, 87 and 93 percent of all younger (16 to 17 years) and all older (18 to 19 years) apprentices considered smoking as hazardous, including about 5/6 of the smokers. Lung cancer is given as the principal hazard by 63 percent, cancer in general by 27 percent, and heart disease by 5 percent of all apprentices. In a sample of 404 apprentices, 35 percent of the younger but only 4 percent of the older ones would refuse an offered cigarette; 21 percent of the 16- to 19-year-old girls would do the same. Cigarette smoking does not look impressive to two-thirds of all apprentices among either the smokers or nonsmokers.

82–1754. Black, D.; Morris, J. N.; Smith, C.; Townsend, P. **Inequalities in Health.** *World Health Forum* 3(1): 68-73, 1982.

Differences in health status between the social classes were found to be related to smoking, overcrowding, and work accidents, which are strongly class-related in Great Britain. Despite a decline in infant mortality rates, considered to be reflective of general social conditions, the health experience of classes V and IV (unskilled and semiskilled manual classes) relative to class I (professionals) did not improve, and in some respects deteriorated throughout the 1960s and early 1970s. A gap also exists in the utilization of health services. These inequalities in health are multicausal in nature, and three objectives are presented for health and social policy: a better start in life for children, preventive and educational action toward public health, and improved quality of life for the disabled. Recommendations include promotion of changes in smoking, drinking, dietary, and exercise behaviors.

82-1755. Borgman, R. F.; Peeler, H. J. **Relationship of Nutrition, Caffeine Consumption, and Cigarette Smoking in Problem Drinkers: A Prospective Study in South Carolina.** *Journal of the South Carolina Medical Association* 78(8): 426-430, August 1982.

The problem drinker differs from the general population in smoking habits, nutritional status, drug abuse, caffeine consumption, and food intake habits. Interviewers at the Charleston County Detoxification Center administered questionnaires to problem drinkers who were nonalcoholics, and also to alcoholics. Among the problem drinkers, smoking, number of years smoking, and total nonalcoholic liquid consumption were all positively correlated to ethanol consumption. Drinking 10 cups of coffee or more was common. Among the alcoholics, smokers consumed moderately more ethanol than nonsmokers, and 88 percent of the subjects smoked compared with 50 percent in the general population. Irregular breakfast eating and caffeine consumption were also positively correlated. Ethanol consumption did not differ by race, but men consumed more than women. A preference for salt was also noted with increasing beer consumption. The use of alcoholic beverages and cigarettes early in adolescence is associated with use in adulthood. This suggests one substance predisposes to the other, and may provide a potential intervention area.

82-1756. Carlhoff, H.-W. **Rauchgewohnheiten bei Schulern des 4. Schuljahres. [Smoking Habits of the Fourth-Grade Students.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 88-95, German.

The smoking habits of 9- and 10-year-old students of the Baden-Wurttemberg, Germany, elementary schools were surveyed. Questionnaires were administered to rural students (119 boys and 112 girls), students from small and medium-sized towns, and from the city of Mannheim (298 boys, 322 girls). Results of the survey are summarized as follows: (1) smoking by parents decisively affects cigarette smoking by their children; (2) on the average, parents from the big city (72.4 percent) smoke more often than those from the country (51.1 percent); (3) smoking by children is independent of their place of residence, with an increasing trend toward cigarette use in rural and small-town areas; (4) in the big city, the percentages of nonsmoking boys (26.5) and girls (33.5) of smoking parents are significantly high; (5) 40.9 and 23.5 percent of all surveyed children from the rural and big city areas, respectively, indicated curiosity as their principal motivation for smoking; (6) peer pressure was a factor in cigarette use for only 15.0 and 7.1 percent of those surveyed, respectively; and (7) already at that early age, children are looking for justification for their habit, such as pleasure and status, in spite of dubious taste and knowledge of smoking's health hazards (97.2 percent of all children).

82-1757. Chait, L. D.; Griffiths, R. R. **Smoking Behavior and Tobacco Smoke Intake: Response of Smokers to Shortened Cigarettes.** *Clinical Pharmacology and Therapeutics* 32(1): 90-97, July 1982.

When given shortened cigarettes, smokers increased the number of cigarettes consumed and changed their manner of smoking. The response of four cigarette smokers to full-length and three different types of half-length cigarettes was examined in a naturalistic laboratory environment. During daily 100-minute sessions, subjects smoked ad libitum: (1) full-length (100 mm) cigarettes, (2) the distal half of cigarettes, (3) the proximal half of cigarettes, or (4) the proximal half of previously smoked cigarettes. As a group, subjects smoked 75 percent more half-length cigarettes than full-length cigarettes. Subjects also puffed at a higher rate (i.e., had shorter interpuff intervals) on half-length than on full-length cigarettes. Mean puff duration (seconds/puff) was higher when subjects smoked the distal-half cigarettes than when they smoked the proximal-half cigarettes, and subjects spent proportionately more time puffing on the distal-half cigarettes than on the other three types. Through a combination of smoking more half-length cigarettes and modifying the way they smoked half-length cigarettes, subjects maintained the same intake of smoke (as measured by expired air carbon monoxide) during sessions as when they smoked full-length cigarettes. These results demonstrate that smokers make complex adjustments in their smoking behavior in response to changes in cigarette length. (Auth. Abs. Mod.)

82-1758. Christen, A. G.; Glover, E. D.; Adams, E. E. **Six Persistent Myths About Tobacco Use.** *Health Education* 13(4): 35-37, July-August 1982.

Six myths concerning tobacco consumption are presented, and the inaccuracies of each are explained. The first myth is that tobacco is not really very harmful, especially if consumed moderately, although much research shows otherwise. The second myth assumes that it is impossible to give up smoking, but millions of people have successfully done so, and a variety of community resources are available to help. The third myth is the belief that smoking is not a very pleasurable habit, but smoking or other tobacco use can be meaningful and pleasant to the user, becoming very much a part of the user's life. The fourth myth is that there is a single correct way to give up smoking. Each individual's smoking habit is unique, however, and there are various methods for quitting to choose from. The fifth myth assumes that failure to quit totally thus indicates personal failure. Various individuals may not quit altogether, but progress in reducing intake or consumption is a positive accomplishment. The final myth assumes that pressuring or frightening people can be effective in smoking cessation, but such methods may actually be counterproductive.

82–1759. Cooperstock, R.; Thom, B. **Health, Smoking and Doctors' Advice.** *Journal of the Royal College of General Practitioners* 32(236): 174-178, March 1982.

One hundred and sixty-seven individuals with chronic conditions were interviewed about their smoking history and current habits as part of two larger studies on health care. The interviews permitted individuals to comment freely on reasons for stopping or continuing smoking and the role of doctors' advice in these decisions. Only 22 percent had never smoked. The youngest and males were most likely to have smoked. At time of interview, 51 percent of those who had ever smoked were still smoking. Those who continued to smoke gave several reasons for doing so. Women most often described smoking as a way of coping, while men were most likely to give hedonism as a reason. Other reasons given included habit, claiming that smoking was beneficial to the respondent, denial, fatalism, and claiming that smoking was harmless to the respondent. Those with circulatory disorders, in contrast to respiratory or musculoskeletal disorders, were most likely to have stopped. Among those who had quit, a majority described health factors as their major reason. Smaller percentages of respondents gave reasons such as abstinence during illness or hospitalization, alleging that smoking made them ill, cigarette prices, and social or religious motives. Perceptions of doctors' advice varied by both age and diagnosis, with the elderly claiming to have received little medical advice. Those with histories of circulatory disorders reported receiving advice more frequently. Doctors may have advised these individuals more frequently because there was threat to life, but it is also possible that individuals with life-threatening disorders are more likely to recall such advice. Doctors who wish to change patients' smoking behavior might consider the patients' experiences, beliefs, and perceptions. (Auth. Abs. Mod.)

82–1760. Creswell, W. H., Jr. **Quo Vadis—Health Education?** *Health Education* 13(4): 12-17, July-August 1982.

The development of the field of health education is reviewed in the perspective of future trends. Health education grew from the Kellogg project, which began as a program to train high school students in a domestic science class to be nurses' aides. Other events, such as Sputnik, the curriculum revolution, and the 1964 Surgeon General's Report on Smoking and Health, have influenced contemporary programs. Recent publications emphasize the role of personal habits, such as smoking, drinking, eating, overuse of medications, lack of exercise, and others, in preventing disease and promoting health. In health behavior change programs, the traditional knowledge-attitude-behavior consistency model is used widely, but is not always appropriate: many people, as in the case of smokers, are already aware of dangers to their health. Most smokers want to quit, and rather than an exhortation, need to "learn the skill of not smoking." An analysis of public health habits indicates people are adopting healthier lifestyles. The 1964 Surgeon General's Report and subsequent antismoking campaigns are credited with causing a significant reduction in adult smokers, thereby contributing to a reduction in cardiovascular disease. Recommendations for increasing research and developing a unified theory of health education are included.

82–1761. Eysenck, H. J. **Schenk and the Personality of Smokers.** *Personality and Individual Differences* 3(2): 217-218, 1982.

Schenk's study of personality and smoking points out that where the drug in question, i.e., nicotine, is socially acceptable, personality variables are less important. Thus as smoking becomes less acceptable, the personality factors involved in the habit will become more crucial. Schenk divided his subjects, 19- and 20-year-old German army recruits, into nonsmokers, experimenters, ex-smokers, moderate smokers, and heavy smokers. Questionnaires were used to assess extraversion, neuroticism, general aggressiveness, political attitudes, experience-seeking, and sensation-seeking. The precise amount smoked; intention to quit, reduce, or continue consumption; and the estimated ease of quitting were also analyzed. The various groups did not lie along a continuum, but rather multiple factors were required to explain the intergroup variations on the personality scales. Social and political attitudes, as well as extraversion and neuroticism, were profitable in characterizing different types of smokers.

82–1762. Fagerstrom, K.-O. **Tobacco Smoking, Nicotine Dependence and Smoking Cessation.** Doctoral Dissertation, Uppsala University, Uppsala, Sweden, Almqvist and Wiksell International, 1981, 36 pp.

The development and validation of a questionnaire (the Tolerance Questionnaire) for measuring nicotine dependence among tobacco smokers are described and the relationship of the questionnaire to smoking cessation, smoking withdrawal symptoms, and regulation of nicotine intake is reported. Four studies were conducted to test: (1) the questionnaire's validity in relation to body temperature pre- and postsmoking withdrawal and tolerance defined as heart rate increase contingent on smoking tobacco; (2) the effectiveness of a nicotine chewing gum in smoking cessation; (3) the placebo effect of nicotine instruction; and (4) nicotine regulation through compensatory smoking behavior. Results show that nicotine dependence differs greatly among smokers with the most highly dependent having more withdrawal symptoms and less successful outcomes. Symptoms seemed to decrease when the abstainers had access to a nicotine chewing gum and the highly dependent smokers almost doubled their chance of being abstinent at 6 months with this device. It is recommended that, in the case of low-yield cigarettes, less tar and carbon monoxide are positive features; however, to reduce these substances to the lowest possible levels, nicotine should be kept at a medium or high level to avoid self-regulation through compensatory smoking.

82–1763. Golding, J.; Mangan, G. L. **Arousing and De-Arousing Effects of Cigarette Smoking Under Conditions of Stress**

and Mild Sensory Isolation. *Psychophysiology* 19(4): 449-456, July 1982.

The arousing and de-arousing effects of smoking a 1.3 mg nicotine delivery cigarette, measured by changes in electrodermal, heart rate, and electroencephalogram (EEG) alpha responding, were examined under conditions of stress, induced by aversive white noise, and mild sensory isolation. The subjects were divided into three groups (n=8 each)—"Real Smoking," where each individual smoked a whole cigarette; "Sham Smoking," where the individual smoked an unlit cigarette while smoke floated about from a second, lit cigarette in an ashtray; and "Situation Control," where the individual did nothing. Compared with sham smokers and the situation control group, smokers showed significant arousal elevations in all response systems under sensory isolation conditions, but mixed stimulant (heart rate response) and depressant (EEG, skin conductance response) effects under stress conditions. Thus, to some extent, situation differentially determines the significance and direction of smoking effects in the response system examined. (Auth. Abs. Mod.)

82–1764. Haussmann, G. **Die Rauchfreie Zone "Schule" als Paedagogische Notwendigkeit. [The Nonsmoking Zone "School" as Pedagogic Necessity.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive and Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 78-81, German.

The nonsmoking zone "school" is defined in terms of space and persons. It includes all school premises and all persons occupied in school. Active participation of teachers and students is a prerequisite for successful establishment of the nonsmoking zone. Intervention of school authorities with pedagogical planning and behavior, personal example and formal nonsmoking instruction by teachers, and activity which will build a new group consciousness among nonsmoking students are cited as the necessary measures. The role of responsible authority is seen as elaboration of the health curriculum for teachers, as is abolition of smoking rooms in schools, and introduction of "smoking" as a compulsory subject. The task of teachers is to show a personal example by voluntary cessation of smoking, to communicate necessary information to students in the course of teaching various subjects, and to encourage students' active participation in smoking control by initiating other antismoking activities. Successful examples of such undertakings in West German schools are given. Prohibition of advertising for tobacco products is called for to make the nonsmoking zone "school" a complete success.

82–1765. Ho-Yen, D. O.; Spence, V. A.; Moody, J. P.; Walker, W. F. **Why Smoke Fewer Cigarettes?** *British Medical Journal* 284(6333): 1905-1907, June 26, 1982.

A reduction in the number of cigarettes consumed by 16 volunteers led to compensatory changes in their inhalation behavior. The volunteers were tested when smoking their own brand of cigarettes normally and when smoking half their usual number of cigarettes. While smoking half their usual amount, the subjects changed their inhalation behavior. Over this period the percentages of carboxyhemoglobin were not significantly different from steady-state values whereas plasma nicotine concentrations rose significantly. With the reduction in cigarettes there were significant falls in hemoglobin concentration, packed cell volume, and red cell count. These findings suggest that the advice given to patients to smoke fewer cigarettes should be accompanied by a warning against increasing inhalation. Patients who say that they have reduced their smoking but who have unaltered carboxyhemoglobin concentrations should not be discredited. (Auth. Abs. Mod.)

82–1766. Jamrozik, K.; Fowler, G. **Anti-Smoking Education in Oxfordshire General Practices.** *Journal of the Royal College of General Practitioners* 32(236): 179-183, March 1982.

A postal questionnaire survey of 360 Oxfordshire general practitioners (GP) and health visitors (HV) on the subject of antismoking education was conducted in May-June 1980. Two mailings produced a response rate of 87 percent. For both the doctors and the health visitors in the survey, the proportion who did not smoke currently (GP=78 percent, HV=93 percent) was very similar to the proportion who agreed that their profession has a role to play in setting a nonsmoking example (GP=84 percent, HV=94 percent). Involvement in antismoking education was felt to be more relevant for the doctors than for the health visitors. Health visitors thought that health education officers had a major role to play; they were also more likely than doctors to use literature as an aid in counseling smokers. In general, the mass media were not thought to be effective in helping individual smokers to give up the habit. Both doctors and health visitors were in favor of their professional organizations exerting pressure on Parliament, but only one respondent had ever written to a member of Parliament about smoking. (Auth. Abs. Mod.)

82–1767. Kozarevic, D.; Racic, Z.; Gordon, T.; Kaelber, C. T.; McGee, D.; Zukel, W. J. **Drinking Habits and Other Characteristics. The Yugoslavia Cardiovascular Disease Study.** *American Journal of Epidemiology* 116(2): 287-301, August 1982.

In two large general populations of Yugoslav men from Tuzla, Bosnia, and Remetinec, Croatia, examined in 1964-1965, it was found that men who drank more tended to smoke more. As part of the Yugoslavia Cardiovascular Disease Study, the drinking histories of 10,641 men were obtained and the relationship of these habits to other characteristics reported or measured was determined. In relating smoking to other characteristics, it was found that the number of cigarettes smoked per day was positively associated with pulse rate and liver size and negatively associated with blood pressure, weight, and blood glucose. Unlike alcohol consumption, which did not vary significantly with age, the number of cigarettes smoked per day was significantly greater in young men than in old.

Data are provided on demographic variations and prevalence rates for certain conditions.

82–1768. Kozlowski, L. T.; Rickert, W. S.; Pope, M. A.; Robinson, J. C.; Frecker, R. C. **Estimating the Yield to Smokers of Tar, Nicotine, and Carbon Monoxide From the "Lowest Yield" Ventilated Filter-Cigarettes.** *British Journal of Addiction* 77(2): 159-165, June 1982.

Results of interviews designed to assess the extent of blocking the holes of low-yield, ventilated-filter cigarettes, and results of a modified smoking machine which better estimates the yield of such cigarettes when they are smoked intensively, are presented. Interviews with 46 low-yield smokers show 52 percent admitting to having blocked the ventilation holes on these cigarettes at some time with either lips, fingers, or tape. Although only three smokers admitted blocking the holes at the present time, 41 percent (of the 39 who were observed smoking) gave evidence (by direct observation and by inspection of characteristic stain patterns on the filter) of currently blocking the holes. A modified smoking machine assay (a 2.4 sec, 47 ml puff, with a 44 sec interval, vent-holes blocked) demonstrated how much tar, nicotine, and carbon monoxide (CO) an average smoker might derive from the lowest yield cigarettes in the United Kingdom, Canada, and the United States. Compared with standard assays, tar yield increases from 15- to 39-fold, nicotine from 8- to 19-fold, and CO from 10- to 43-fold when ventilation is stopped. It is suggested that smokers be advised of the risks of blocking the holes of ventilated-filter cigarettes and taught how to detect whether they are doing it. The construction of low-yield cigarettes that will ensure low yields to smokers requires careful attention to the behavior of smokers. (Auth. Abs. Mod.)

82–1769. Lambert, C. A. **Prevalence of Tobacco and Alcohol Use Among Massachusetts Students, Ages 10 to 18.** In: National Institute on Drug Abuse. *Drug Abuse Trends and Epidemiologic Issues.* Rockville, Maryland, National Institute on Drug Abuse, Division of Data and Information Development, Forecasting Branch, 1982, pp. II-124—II-130.

Tabulations of data from four eastern Massachusetts Smoking and Alcohol Health Education/Risk Reduction intervention projects located in affluent, blue-collar, and Chinese-American communities indicate that (1) big increases in smoking incidence occur in the 6th, 7th, and 8th grades, a critical period for initiation; (2) males experiment earlier than females (at the 6th grade level 44 percent boys, 32 percent girls), however, females rapidly close the gap by the 7th grade and by the 8th grade lead the males, 72 percent to 64 percent; (3) by the 12th grade, over 4/5 of the students have tried smoking; (4) current prevalence (smoking past month) shows a big jump in the 7th and 8th grades (8 percent in the 6th grade to 31 percent in the 8th), a peak is reached in the 10th grade at 40.8 percent (and almost 53 percent for 10th grade girls) and a return to 33 percent by the 11th and 12th grades; (5) proportions of students who claim to smoke from 5 to 15 packs/week rise slowly until 10th grade when the rate doubles from 3 to 6 percent. Females generally double the male rate at this point reaching a 9 to 11 percent level in the 11th and 12th grades. Alcohol consumption figures are also tabulated. It is recommended that the significant characteristics of students who appear free of peer pressure be defined.

82–1770. Lauer, R. M.; Akers, R. L.; Massey, J.; Clarke, W. R. **Evaluation of Cigarette Smoking Among Adolescents: The Muscatine Study.** *Preventive Medicine* 11(4): 417-428, July 1982.

The smoking behaviors of parents and peers are important factors in the decisions of children aged 12 to 18 to smoke or not to smoke according to a survey conducted in Muscatine, Iowa. The smoking behaviors and related factors were studied in 2,156 schoolchildren using data collected through a confidential questionnaire, a "randomized question response," and by saliva thiocyanate analyses. The proportion reporting smoking on the questionnaire and the anonymous randomized question response were not significantly different. Those reporting the greatest frequency of smoking had the highest levels of saliva thiocyanate. No difference in self-reported smoking was observed in subjects who, prior to completing the questionnaire, were told their saliva would be analyzed for tobacco products and those who were not. These data suggest that children under certain circumstances may honestly and validly complete a questionnaire about their smoking behavior. Of respondents who report both parents as nonsmokers and whose best friends are nonsmokers, 80 percent have never smoked, and 3 percent are regular smokers. Of those with both parents and best friends who smoke, only 11 percent have never smoked and 74 percent are frequent smokers. (Auth. Abs. Mod.)

82–1771. Mangan, G. L. **The Effects of Cigarette Smoking on Vigilance Performance.** *Journal of General Psychology* 106(1): 77-83, 1982.

Cigarette smoking promoted vigilance performance in a group of 24 individuals who completed an auditory vigilance task after smoking either a "low" nicotine cigarette (n = 12) or a "middle" nicotine cigarette (n = 12). Each subject served as his own control in an identical session 1 week before or after the test session. The smoking was followed by improved vigilance in both groups as compared with the control performance, but came about in different ways for the two groups. The low-nicotine group recorded improved detection, while the middle-nicotine group had a reduced false positive rate. These effects are attributed to the known differential, biphasic effects of nicotine on performance. (Auth. Abs. Mod.)

82–1772. Matarazzo, J. D. **Behavioral Health's Challenge to Academic, Scientific, and Professional Psychology.** *American Psychologist* 37(1): 1-14, January 1982.

Smoking is one of four behavioral health challenges discussed in relation to the reduction of human and financial costs associated with a number of preventable dysfunctions. Although the percentage of male adult smokers decreased from 52.6 percent in 1955 to 36.1 percent in 1975, there was a 4.9 percent increase in adult female smokers during the same period. Data on teenage smoking show that the time of greatest susceptibility to becoming a regular smoker is around age 12. Recent decreases in teenage smoking provide evidence that smoking behavior is amenable to influence. National polls have repeatedly shown that the majority of regular smokers would like to quit. In addition, since 1964 some 30 million U.S. adults have successfully stopped smoking. Both of these statistics indicate an atmosphere for success for psychologists and other workers in this health promotion field (preventing or ceasing smoking). The three other health challenges reviewed relate to alcohol use, health diets and related risk reduction, and automobile and motorcycle accidents.

82–1773. McCaul, K. D.; Glasgow, R.; O'Neill, H. K.; Freeborn, V.; Rump, B. S. **Predicting Adolescent Smoking.** *Journal of School Health* 52(8): 342-346, August 1982.

Contrary to the findings of other surveys, parental smoking was found to be a poor predictor of adolescent smoking. The significant predictors among 297 white, middle-class seventh grade students (143 males, 154 females) were intent to smoke, number of friends who smoke, and percent of siblings who smoke. The students were surveyed in October 1979 and again a year later at which time a collection of saliva for thiocyanate analysis was made to enhance the validity of self-reported smoking status. Predictor variables included 10 survey items assessing the smoking behavior of students' friends and family, students' school behavior and beliefs about smoking, and students' intentions to smoke in the future. Approximately 6 percent of both males and females in the seventh grade sample reported being current cigarette smokers and 10 percent of the eighth grade students reported current smoking. Univariate analyses indicated that smokers differed from nonsmokers on a number of measures and that there were few sex differences on either the survey variables or on smoking status. Stepwise discriminant function analyses revealed that it was possible to accurately predict the onset of adolescent cigarette smoking by combining the survey variables. It is suggested that programs to prevent adolescent smoking might profitably focus on (1) teaching adolescents to cope with peer pressure, (2) modifying future intentions to smoke, (3) teaching adolescents the harmful short-term effects of smoking, (4) emphasizing the difficulty of quitting smoking, and (5) modifying the norms governing smoking behavior. (Auth. Abs. Mod.)

82–1774. McCusker, K.; NcNabb, E.; Bone, R. **Plasma Nicotine Levels in Pipe Smokers.** *Journal of the American Medical Association* 248(5): 577-578, August 6, 1982.

Plasma nicotine levels in pipe smokers illustrate differing inhaling patterns of smokers which may explain the lower morbidity and mortality of pipe smokers compared with cigarette smokers. Plasma nicotine levels were determined in three groups of pipe smokers: (1) primary pipe smokers, (2) persons who smoke both pipes and cigarettes, and (3) secondary pipe smokers, i.e., former cigarette smokers who now smoke pipes only. Primary pipe smokers inhaled minimally, as evidenced by low plasma nicotine concentrations after pipe smoking. In contrast, persons who smoke cigarettes and also pipes had high plasma nicotine levels after pipe smoking (mean, 22 ng/mL) and, hence, must have inhaled. Secondary pipe smokers (former cigarette smokers) had mostly low levels of plasma nicotine (mean, 6 ng/mL), suggesting that not all cigarette smokers continue to inhale when converting to pipe smoking. Pipe smoking may be safer than cigarette smoking in certain individuals. (Auth. Abs. Mod.)

82–1775. McManus, I. C.; Weeks, S. J. **Smoking, Personality and Reasons for Smoking.** *Psychological Medicine* 12(2): 349-356, May 1982.

Contrary to the usual findings, smoking did not relate to extraversion but to psychoticism in this study of the relationships between smoking, reasons for smoking, and personality in 98 cigarette smokers. It is suggested that this relationship is the true one, and previous findings have been due to a contaminated measure of extraversion. Correlations were also discovered between extraversion and tea and coffee intake as well as alcohol consumption. Neuroticism was related to the amount smoked, and particularly to a willingness to give reasons for smoking. There was no relationship between the Reasons for Smoking Questionnaire results and attempts or success at giving up smoking. Smokers who rolled their own cigarettes were more introverted than others. No measure showed any significant relationship with the nicotine or tar content of cigarettes. (Auth. Abs. Mod.)

82–1776. McNabb, M. E.; Ebert, R. V.; McCusker, K. **Plasma Nicotine Levels Produced by Chewing Nicotine Gum.** *Journal of the American Medical Association* 248(7): 865-868, August 20, 1982.

Nicotine chewing gum is designed to maintain plasma nicotine levels during a smoking cessation effort while the individual copes with the loss of the repetitive behavioral components of smoking. The plasma nicotine levels obtained with hourly gum chewing were compared with levels obtained with cigarette smoking in nine patients with lung disease. There was less variation in plasma nicotine level with hourly use of the chewing gum than with cigarette smoking. Chewing 2- and 4-mg nicotine gum hourly produced mean steady-state plasma nicotine levels of 11.8 and 23.2 ng/mL, respectively. This compares with a mean plasma nicotine trough level during usual smoking of 15.7 ng/mL and a mean trough level of 18.3 ng/mL with hourly smoking of a cigarette with a nicotine yield of 1.1 mg. The difference may be due to slow release of nicotine from the gum, as well as slower nicotine intake from the mouth vs. the lung. Few side effects were seen with the use of either the 2- or 4-mg gum. In a short-term study, nicotine gum proved an acceptable source of nicotine for aiding smoking cessation. (Auth. Abs. Mod.)

82–1777. Pederson, L. L.; Lefcoe, N. M. **Multivariate Analysis of Variables Related to Cigarette Smoking Among Children in Grades Four to Six.** *Canadian Journal of Public Health* 73(3): 172-175, May-June 1982.

For present and past smokers among schoolchildren in grades 4 through 6, the most important determinant of smoking was peer smoking; parental smoking and attitude toward smoking were also strongly related. The questionnaire responses of 3,000 children in London, Ontario, were analyzed to determine the relative predictive power of variables relating to cigarette smoking. In this sample, 76 percent had smoked, and 7.1 percent were current smokers. Among those who had smoked and quit, attitude was the most important determinant; peer smoking and grade were also strongly related. These results are similar to those found among teenagers and have implications for preventive education programs. Education programs should be aimed at the peer group as well as at the individual and should attempt to make cigarette smoking unacceptable to the group as a whole. (Auth. Abs. Mod.)

82–1778. Practitioner. **Girls Outsmoke Boys.** *Practitioner* 226(1369): 1201, July 1982.

A consequence of "equality" is that relative to smoking, teenage girls either caught up or passed teenage boys in Italy, Greece, Uruguay, Switzerland, Belgium, France, New Zealand, Denmark, Canada, Federal Republic of Germany, Sweden, Netherlands, Norway, and the United States. The ratio for girls to boys varied from 55:51 percent in Italy to 19:16 percent in the United States. Boys continued to outsmoke girls in Bulgaria, Australia, United Kingdom, Finland, Papua New Guinea, Ethiopia, Nigeria, and India. Despite advertising links between smoking and sports, the majority of athletes do not smoke. The more individualistic the sport, the less likely was the competitor to smoke: 40 to 50 percent of the football players smoked while none of the cyclists surveyed did. None of the French athletes selected for the Olympic games smoked, and 75 percent of international athletes are nonsmokers. Perhaps their example will better dissuade youth from the habit than warnings of ill health.

82–1779. Rawbone, R. G.; Guz, A. **Cigarette Smoking Among Secondary Schoolchildren 1975-79.** *Archives of Diseases in Childhood* 57(5): 352-358, May 1982.

Smoking patterns found among schoolchildren in the London borough of Hounslow suggested that awareness of several smoking-associated health risks was not altogether effective in reducing smoking. A questionnaire relating to smoking habits, respiratory symptoms, and health attitudes was administered to schoolchildren aged 11 to 17 throughout a defined geographical area in both 1975 and 1979, with a valid response from 10,498 and 12,002 young people, respectively. Each cohort was almost entirely different. The results suggest that although the prevalence of regular smoking has decreased in boys from 16 to 13 percent, it has increased in girls from 13 to 14 percent, and that at all ages more girls smoke than boys. However, despite the fall in the prevalence of regular smoking in boys there has been an overall increase in cigarette consumption. Young people who are regular smokers predominantly smoke middle-tar cigarettes while among experimental smokers there is a high incidence of low-tar smoking, which might suggest that such cigarettes facilitate the taking up of the habit by children. The previously described relationships between smoking and respiratory symptoms were confirmed. During the 4-year study period young people's knowledge of the association between lung cancer and smoking remained high. Their knowledge of the associated links between smoking and heart disease and stroke increased appreciably. For "chestiness" and bronchitis, the reported knowledge remained steady. It is suggested that specific health education during the years 1975-1979 has not been successful, and there is the need for research. (Auth. Abs. Mod.)

82–1780. Russell, M.A.H.; Sutton, S. R.; Iyer, R.; Feyerabend, C.; Vesey, C. J. **Long-Term Switching to Low-Tar Low-Nicotine Cigarettes.** *British Journal of Addiction* 77(2): 145-158, June 1982.

Puffing patterns and blood measures of smoke intake were studied in 12 subjects before and after switching to low-tar (10.9 mg) low-nicotine (0.7 mg) cigarettes for 12 weeks. Puff rate, puff volume, total volume puffed per cigarette, and the number of cigarettes smoked were not changed significantly after switching to the lower yield cigarettes. The reduction in mouth-level nicotine intake (calculated from butt content) was identical to the 47 percent reduction in the machine-smoked nicotine yields. This is in keeping with the measures of puffing in showing the absence of any tendency to compensate for the reduced yields by increasing the intensity of puffing. In contrast, plasma nicotine and cotinine levels were reduced proportionally less than the nominal yields indicating that the subjects compensated to some extent by increasing inhalation. The average reductions in plasma nicotine and cotinine were identical (30 percent). Plasma thiocyanate and carboxyhemoglobin levels were not significantly different after switching to the lower yield cigarettes but the estimated tar intake to the lungs was reduced by about 15 percent. Although the low-yield cigarettes were rated as slightly too weak, there was no significant loss of satisfaction. The fact that subjects tolerated a 30 percent reduction in plasma nicotine without loss of satisfaction and without puffing more intensively suggests that the increase in inhalation was not determined by a need to maintain nicotine intake. This and the discrepancies between natural and experimental switching studies are discussed. (Auth. Abs.)

82–1781. Tobin, M. J.; Sackner, M. A. **Monitoring Smoking Patterns of Low and High Tar Cigarettes With Inductive Plethysmography.** *American Review of Respiratory Disease* 126(2): 258-264, August 1982.

Smokers exhibit different puffing and inhalation patterns when smoking high tar content (HTC) as compared with low tar content (LTC) cigarettes and when using a cigarette holder with either. Using an inductive plethysmograph coil around the cheeks to measure puff volume, 10 subjects smoked an HTC and an LTC cigarette, each with and without a cigarette holder. The mean puff volume from LTC of 52±15 ml was significantly higher than HTC of 39±10 ml. The number of puffs per cigarette (8.4±2.1 for LTC, 8.3±2.0 for HTC), the volume of air inhaled (841±517 ml for LTC and 748±323 ml for HTC), and the duration of inhalation and associated breathhold (5.5±2.6 seconds for LTC and 5.6±2.4 seconds for HTC) as measured by respiratory inductive plethysmography were comparable. The perception of intensity of inhalation by the subjects bore little relationship to the objective measurement. A cigarette holder increased the number of puffs per cigarette to 12.6±4.8 for LTC and to 11.9±3.6 for HTC and increased the puff volume for HTC to 50±12 ml and the duration of inhalation to 6.6±2.5 for LTC. These results show that (1) smokers who switched acutely from HTC to LTC draw larger puffs, nullifying the benefits of smoking LTC, and (2) cigarette holders modify smoking pattern and hence if used with an incorporated flowmeter to monitor the natural pattern of smoking might give spurious information. (Auth. Abs. Mod.)

82–1782. Tuyns, A. J.; Hu, M. X. **Changing Smoking Patterns in Calvados (France).** *British Journal of Addiction* 77(2): 167-183, June 1982.

Results of a population survey show that at some time during their lives, 80 percent of males and 20 percent of females in Calvados, France, indulged in smoking while 54 percent and 14 percent, respectively, are currently smoking. The average number of cigarettes smoked per day is 16.6 for males and 11.4 for females. No difference was found between urban and rural areas. Cigarettes are the most widespread product smoked with the handrolled variety still used by males of older age groups, particularly in rural areas; however, these are progressively being replaced by commercial cigarettes. The proportion of ex-smokers increases with age, probably due to antismoking campaigns, but the resulting decrease in consumption is compensated by an earlier starting age. Among females, the contrast between age groups is more dramatic; while smoking remains rare over age 55, at 25-34 years of age, 21.2 percent indulge in smoking and below that age, 40.7 percent smoke. Though the differential behavior of males and females toward smoking contrasts with attitudes observed in the United Kingdom and the United States, recent changes occurring in young females indicate a trend toward a more homogeneous distribution, as in other countries, with similar prospective health implications. It is toward the younger population that protective measures need to be taken. (Auth. Abs. Mod.)

82–1783. Wake, F. R. **A Psychologist's Perspective on the Public Health Objectives.** *Cancer Forum* 5(3): 144-147, September 1981.

Four elements of behavior must be understood in predicting or modifying a particular behavior: action, target, context, and time. The most important public health behavior, and the one with the highest potential return on money and effort, is cigarette smoking. In applying these elements to smoking cessation, the action with the best record of success is strong educational programs. These have succeeded in both slowing the onset of smoking and lowering the actual number of smokers. However, an international strategy is needed to counter the tobacco industry's own international efforts. A variety of targets have been given priority: children, adolescents, pregnant women, then young adults and the middle-aged. The context of antismoking programs should be presented in differing forms, differing points of view, and a variety of visual and verbal presentations to avoid boredom while providing reinforcement. The timing is of small concern, except these messages should be delivered around the clock.

See also, **82–1680, 82–1687, 82–1803**

SMOKING PREVENTION AND INTERVENTION

82–1784. American Cancer Society. **National Conference on Smoking or Health. Developing a Blueprint for Action. (Proceedings).** Proceedings of the National Conference on Smoking or Health, Developing a Blueprint for Action, New York, November 18-20, 1981, 320 pp.

Representing a broad spectrum of voluntary and government interests, 21 national organizations met to plan and develop the National Conference on Smoking or Health. Obstacles to overcoming smoking-related morbidity and mortality and strategies for smoking control were considered in developing the Blueprint for Action. Physicians, scientists, broadcasters, journalists, government officials, legislators, business leaders, hospital administrators, school officials, public relations professionals, and public educators were invited to join the working teams in groups of 20. Ten key aspects of the antismoking campaign were formulated and assigned to working groups—each with the charge to come up with realistic recommendations. The groups were: Prevention of Smoking through Comprehensive School Health Education; Use of the High Risk Concept in Smoking Control; Advancing the Role of Hospitals in Reducing Smoking; Smoking Control in the Workplace; High Priority Federal Government Initiatives; High Priority State and Local Government Initiatives; Tobacco-Related Research; Countering Cigarette Advertising and Supporting the Rights of Nonsmokers Through the Media; Motivating Smokers to Assume Personal Responsibility for Quitting on their Own; and Future Strategies for the Changing Cigarette. The blueprint will be implemented as the chairman of the conference planning committee contacts those organizations best able to develop a model and provide support. (Auth. Abs. Mod.)

82–1785. Buchholz, E. H. **Verhuetung des Rauchens als Aufgabe der Krankenkassen. [The Task of Health Insurance in Smoking Prevention.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 50-54, German.

A 6-year (1974-1980) media campaign by the West German health insurance industry is credited with 36.7 percent of smokers and 63.3 percent of nonsmokers recognizing the health hazards of smoking. Industry attitudes during this time evolved from an emphasis on curing disease to that of disease prevention through health maintenance. Some of the techniques used to disseminate health information include brochures, pocket-sized books, pamphlets, posters, and a newspaper ("Stay Healthy," 9 million copies published bimonthly)—all addressed to specific target groups. A smoking cessation radio program received a great many requests by youth for information and quitting packets. The health insurance companies also made behavior modification counseling and cessation seminars available, claiming a 40 to 50 percent success rate. Nonsmoking education is being planned for all regions of the West German Federation, and six health clubs are currently developing teaching tools for 4- to 10-year-old children.

82–1786. Burenkov, S. P.; Glasunov, I. S. **USSR: The Preventive Approach in Public Health.** *World Health Forum* 3(1): 54-57, 1982.

An experimental program of preventive medicine, which highlights antismoking campaigns, rationalization of the diet, and campaigns against obesity and hypodynamia, is proposed to reduce the morbidity and mortality rates associated with noncommunicable diseases in the Soviet Union. According to data from the Soviet Central Statistical Board, 51 percent of all deaths in 1978 were from cardiovascular diseases and 14 percent were from oncological diseases. Data from a study directed by the All-Union Cardiological Scientific Centre shows that an average of 47 percent of 40- to 59-year-old men i six Soviet cities smoke at least one cigarette a day. Further, in a district of Moscow it was found that 10 percent of boys aged 12 to 13 smoke, and by the time they are a year older the figure is 30 percent. Evidence that behavioral risk factors can be combated is provided by (1) a survey in Moscow which showed that 66 percent of men and 64 percent of women regarded smoking as harmful and wished to quit, and (2) an antismoking program which resulted in 19 percent of the male participants being nonsmokers a year later.

82–1787. Clutterbuck, D. **Persuading Employees To Break the Smoking Habit.** *International Management* 36: 27-29, April 1981.

The importance of company-sponsored antismoking programs, which could save billions of dollars each year in health costs and lost workdays, is discussed. The program initiated by Johns Manville in 1976, following the demonstrated synergistic cancer risk of asbestos exposure and smoking, is outlined, from the first no-smoking ban in two plants which caused some problems as to employee rights, to the current use of plant smoking cessation programs (Smokenders) for all employees who smoke. Following the cessation programs, a smoking ban is imposed for all employees (and visitors), and the success of the program is shown in the percentages (virtually 100 percent) of employees who are nonsmokers, at least during the workday. The cost of the program is completely covered for employees who remain abstinent for a period of 9 months. The company also utilizes reinforcement techniques: posters, employee discussions, and doctor's counseling. Programs used by other companies, including strictly monetary incentive types, are also described, although the most common solution to the smoking problem is to segregate smokers from nonsmokers. Nonsmokers are now demanding a smoke-free environment, however, and the problem of passive smoking has become an issue, with nonsmoking employees being granted compensation for passive smoking-related illnesses. It is concluded that the average company must at some stage in the future find an equitable compromise between the interests of smokers and those of the nonsmokers.

82–1788. Coe, R. M.; Crouse, E.; Cohen, J. D.; Fisher, E. B., Jr. **Patterns of Change in Adolescent Smoking Behavior and Results of a One Year Follow-Up of a Smoking Prevention Program.** *Journal of School Health* 52(8): 348-353, August 1982.

A smoking prevention program for adolescents conducted in two public middle schools focused on resisting peer pressure to smoke and understanding the intent of commercial cigarette advertising. One class in each school participated in the program group and one served as a control group. The program consisted of eight sessions and was conducted by first-year medical students. Data on smoking behavior and related information were obtained from self-administered questionnaires at baseline, at the conclusion of the program, and 1 year later. It was found that the prevalence of smoking increased with age. Other factors associated with adolescent smoking were smoking among parents and friends, a negative viewpoint about nonsmoking and parental authority, and performing other "risk-taking" behaviors. One year after the program was concluded, the proportion of nonsmokers was higher among those who had participated in the program than among the controls. This suggests that routine implementation of smoking prevention programs in conventional school settings may be productive in reducing the prevalence of cigarette smoking. (Auth. Abs. Mod.)

82–1789. Cohen-Solal, J.-M. **Antismoking Actions in France: Five Year Results.** *World Smoking and Health* 7(2): 20-23, Summer 1982.

Five-year results of an antismoking campaign launched in France in 1976 in conjunction with the adoption of a strong antismoking law are summarized. Generally, the French think that smoking represents a serious health hazard and in 1981, 52 percent of the adult smokers and 31 percent of the young smokers wanted to quit smoking. Between 1976 and 1981, the number of smokers, aged 12 years and older, decreased from 44.2 percent of the population to 38.7 percent, with the most important decrease occurring among male adults. Although cigarette use among youngsters has remained at the same overall level, slightly more girls (45 percent) are smoking than boys (43 percent). School smoking and health education programs are described and the need to coordinate school and adult programs is emphasized. The main provisions of the 1976 antismoking law are listed.

82–1790. Daube, M. **Strategies To Control Tobacco Disease.** *Cancer Forum* 5(3): 157-167, September 1981.

A strategy for a comprehensive tobacco control program is outlined, and includes: (1) a ban on all forms of advertising; (2) prohibition of sales to minors; (3) a firm health warning on packets and such advertising as is permitted; (4) an economic-incentive low-tar program; (5) a taxation strategy; (6) restrictions on smoking in public; (7) governmental funding for health education; and (8) realistic cessation and primary prevention programs. Obstacles to progress include political pressure by the tobacco industry, as well as its commercial opposition. Some objections are made on philosophical grounds, as in the public fight against cholera a century ago. The tobacco industry consistently denies the harmful effects of smoking, and blatantly promotes high-tar products in third world countries where there is no opposition. Governments have no overall commitment to health promotion: it is a low political priority, and underfunded. Market subsidies to the tobacco regime far exceeded the total budget for health education around Europe. Although the media has given publicity to the dangers of smoking, coverage of the issue is only token; advertising revenue has affected editorial judgment. Norway's tobacco control program is discussed as an example.

82–1791. Gray, N. **Review of UICC Program on Smoking and Lung Cancer.** *Cancer Forum* 5(1): 15-18, March 1981.

In 1976, the UICC (Union Internationale Contre le Cancer) began the special project designated as the Program on Smoking and Lung Cancer. A small workshop was held in Lyon to devise a system for measuring smoking rates, and one in Geneva to create clear policy objectives and write a basic manual for workers involved in smoking control. In 1980, the project was redesignated as a formal program of the UICC. Currently, familiarization visits and workshops are being organized in Budapest, Kuwait, Sri Lanka, Japan, and Singapore. The project has become an important international focal point for activities related to smoking-associated cancer. It has contributed substantially to uniformity of policy among public health workers and general antismoking activity in Europe and other regions where workshops have been held. The next 2- to 3-years' activity will be focused on promoting smoking control in developing countries.

82–1792. Hodgkinson, P. **Slow Motion Suicide.** *Nursing Mirror* 154(1): 30-32, January 6, 1982.

In the United Kingdom, about 21,000 men and 4,000 women, age 35-64, die annually of lung cancer or heart disease with the incidence of lung cancer rising in proportion to the rise in cigarette consumption. Ironically, nurses, who are in daily contact with these tobacco-related diseases, remain among the greatest consumers of cigarettes. The role of nicotine in catecholamine production and the circulation of free fatty acids is discussed as are the effects of carbon monoxide on the oxygen transport system. Below the age of 45 years smokers visit their doctors 40 percent more often than nonsmokers, presenting nonfatal illnesses such as high blood pressure; cancer of the mouth, larynx, and throat; peptic ulcers; and pneumonia. Initiation occurs at an age when social factors such as modeling of smoking by parents and peers and feelings of independence, maturity, and rebelliousness are paramount. However, once smoking becomes regular, the social reward decreases and the pharmacological reward becomes greater, making cessation difficult. The roles of tranquilizers, cue control, and motivation in cessation or reduction of smoking are analyzed as are the use of low-tar products for those who can't quit. Cost effectiveness in relation to smoking cessation programs is the focus of the discussion.

82–1793. Kornhuber, H. H. **Primaerpraevention des Zigarettenrauchens. [Primary Prevention of Cigarette Smoking.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 126-133, German.

Because of the carbon monoxide and nicotine in cigarette smoke, inhalation is considered to be the primary health hazard associated with tobacco use. Switching to light (filter) cigarettes would not be less dangerous because of deeper inhalation and an increased frequency of smoking. Antismoking campaigns which advocate abstinence from all tobacco use are considered erroneous because of their failure to recognize that the danger lies in inhalation. Cigar and pipe smoking, which is noninhaling, should be regarded as a reasonable alternative. The attempt to lower the carbon monoxide and nicotine content of cigarette smoke is considered futile. Instead, use of pipe, cigars, or cigarillos is suggested, or more simply, a legal prohibition of using artificially dried and fermented tobacco in cigarettes. In the absence of such prohibition, health education must at least make a clear distinction between inhaling cigarette smoke and not inhaling cigar or pipe smoke. Children age 10 or younger should be taught, preferably, not to smoke at all, and in no case smoke cigarettes. To optimize teaching, feedback from children by means of anonymous questionnaires should be obtained.

82–1794. Kuntzleman, C. T. **Feelin' Good.** *Health Education* 13(4): 24-25, July-August 1982.

The Feelin' Good program aims to reduce future coronary artery disease in children by behavioral intervention through reduction of smoking, aerobic exercise, nutrition, weight control, and stress control. Currently, children learn passivity in health care: parents decide when to see a physician, receive and carry out instructions for health care, and serve as role models. The fundamental objective of the Feelin' Good program is to encourage young people to assume responsibility for their own health and quality of life, and celebrate the joy of being alive. The program attempts to provide scientific explanations for fitness and disease-resistant bodies; help children understand cause, effect, and risk factors in coronary disease; and build value systems with respect to health. Beyond education of the children, reports from teachers indicated that they too had quit smoking or made other modifications as a result of the program. Extensive testing and evaluation of the program is underway.

82–1795. Kunze, M. **Vorschlaege zur Bearbeitung des Tabakproblems aus Oesterreichischer Sicht. [Austrian Suggestions for Treatment of Smoking Problems.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlags gesellschaft mbH, 1982, pp. 24-28, German.

The present status of the "Smoking and Health" problems in Austria is described, health policy goals of the Government are defined, and the trends within the population with respect to these problems are evaluated. The economic dimension of the problem is discussed, and future economic measures for smoking prevention are outlined. Means of educating the general public and schoolchildren about health problems connected with smoking are suggested. Recommendations concerning smoking cessation include the necessity of considering smoking cessation as an integral part of therapy, use of the risk program (20 x 20 program) by high-risk smokers, establishment of special smoking counseling centers, consideration of educational and quality control measures, and development of an optimum smoking cessation method for each individual smoker. Modification of tobacco products to reduce smokers' exposure to noxious substances and control of the production process and products is urged, as well as modification of tobacco pricing policy. Implementation of the suggested measures would require complex interventions on a national level, and international cooperation.

82–1796. Mueller, A. **Rauchen und Schule. Primaerpraevention des Rauchens als Aufgabe der Gesundheitserziehung in Schulen. [Smoking and School: Primary Prevention of Smoking as the Task of Health Education in School.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 63-67, German.

The relative failure of smoking control in West Germany is attributed partly to a wrong approach by the schools to the smoking problem, and partly to school-independent factors. In the past, smoking prevention in schools consisted of prohibition and punishment, which provoked a defiant reaction in students. Also, teachers often ignored the old pedagogical principle of education by example, while they smoked in school. At the present time, parental smoking, the willingness of youngsters to risk sickness and death, and the expansion of addiction to include illicit drugs (heroin, cocaine) and alcohol are cited as factors aggravating the task of teachers in smoking prevention. Smoking intervention by the schools is seen as an interdisciplinary integration of efforts, with the primary task given to biology courses and supportive roles to religion, German literature, and sports. Biological and religious (protestant and catholic) teaching programs for the 5th through 10th grades illustrate the right approach to the smoking problem in schools.

82–1797. Nader, P. R.; Perry, C.; Maccoby, N.; Solomon, D.; Killen, J.; Telch, M.; Alexander, J. K. **Adolescent Perceptions of Family Health Behavior: A Tenth Grade Educational Activity To Increase Family Awareness of a Community Cardiovascular Risk Reduction Program.** *Journal of School Health* 52(8): 372-377, August 1982.

Adolescents' perceptions of and influences on their families' health behaviors were assessed as part of a high school-based educational project. Tenth grade students (n=168) were asked a series of questions related to their own and their families' health practices and requested to encourage their families to watch a countywide Heart Health Telecast. Among the adolescents, 21 (12.5 percent) had smoked tobacco within 24 hours. Although the students did not judge smoking cessation to be a personal health need for themselves, 11.9 percent chose it for their fathers and 10.7 percent for their mothers. Seven percent of the boys and 6 percent of the girls reported chain-smoking as a way of handling stress. Other factors examined included adolescent consumption of alcohol and marihuana, adolescent and parental body weight, eating, and exercise. Adolescents were also questioned about their state of satisfaction with friends, family, education, and individual health, and how confident they were that they could assist parents in altering health behavior. It is concluded that adolescents (1) are interested in health behavior and in the relationships they detect within their families; (2) have ideas already formed about family health behavior and need; and (3) do not see themselves in the role of health advocate for other family members.

82–1798. Noack, K.-A. **Moglichkeiten und Grenzen Schulischer Gesundheitserziehung auf dem Sektor des Rauchens. [Possibilities and Limitations of the School in Smoking and Health Education.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 74-77, German.

A school has at least theoretical influence on the health behavior of the largest enclosed population group. Difficulties are associated with the fact that smoking behavior is the result of a very complex and long learning process affected by a number of psychological and social factors. For instance, the effectiveness of smoking education is limited by the competing requirements of teaching other subjects (drugs and alcohol abuse, nutrition, personal hygiene, etc.), time allocation, teachers' competence, and children's perception of the subject matter as remote from their lives. Practical possibilities of smoking education in elementary and high schools are outlined in teaching materials offered by the Federal Center for Health Information in Koeln, West Germany. The importance of addressing the personal concerns of students in each age group, being aware of motivation and causes of smoking, analyzing actual cases of smoking dependence, discussing advertisement methods and economic aspects, sensitizing students to environmental hazards of smoking, building a positive image of the nonsmoker, staging role-playing by students, and other activities by the teachers are stressed. The attitude of the teacher is also very important.

82–1799. Ramstroem, L. M. **Fortschritte gegen das Rauchen in Schweden. [Anti-Smoking Measures in Sweden.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlags gesellschaft mbH, 1982, pp. 17-23, German.

Swedish governmental and private antismoking activities since 1963 are reviewed. The 4-point, 25-year program is aimed primarily at reducing smoking among young adults. Priority items included in the program published in 1973 are: informing pregnant women, the parents of small children, educators in kindergartens, and school teachers about smoking and health; helping persons working in health and education fields to give up smoking; giving support to market control, e.g., prohibiting advertisement of tobacco products; and influencing public opinion by long-term coordinated campaigns. Examples of suggested information activities are given. To implement the program, two Swedish laws were enacted in 1977 and 1979: one making health warning labels on tobacco packages compulsory, and the other strictly limiting advertisement of tobacco products. A third law for the protection of nonsmokers in public places has been prepared. The results of the antismoking actions are as follows: a continuous decrease in the percentage of adult (18- to 70-year-old) male smokers, and a leveling off of the percentage of adult female smokers since 1970; a decrease in smoking among 16-year-old boys from 41 percent in 1971 to 21 percent in 1979, and from 47 to 34 percent among girls of the same age.

82–1800. Rose, G.; Hamilton, P.J.S.; Colwell, L.; Shipley, M. J. **A Randomised Controlled Trial of Anti-Smoking Advice: 10-Year Results.** *Journal of Epidemiology and Community Health* 36(2): 102-108, June 1982.

Ten-year results are reported from a randomized controlled trial of antismoking advice in 1,445 male smokers, aged 40-59, at high risk of cardiorespiratory disease. After 1 year, reported cigarette consumption in the intervention group (714 men) was one-quarter that of the "normal care" group (731 men); over 10 years the net reported reduction in the intervention group averaged 53 percent. The intervention group experienced less nasal obstruction, cough, dyspnea, and loss of ventilatory function. Over 10 years their mortality from coronary heart disease was 18 percent lower than controls (49 and 62 deaths), and that for lung cancer was 23 percent lower (18 and 24 deaths). Deaths from nonlung cancers were higher in the intervention group (28 vs. 12 deaths). This unexpected difference was due about equally to an excess in intervention and a deficiency in normal care men. It showed no site specificity, and it was unrelated to change in smoking habit. The findings suggest that it is more likely to have been due to chance than to intervention. The total number of deaths were 123 in the intervention group and 128 in normal care (95 percent confidence limits of difference -22 percent to +23 percent). The policy of encouraging smokers to give up the habit should not be changed. (Auth. Abs.)

82–1801. Schenk, J.; Strieder, M. **Die Soziale Bewertung der Zigarettenwerbung. [Social Value of Cigarette Advertisement.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 55-62, German.

Attitudes toward cigarette advertising vs. dealing in marihuana were explored in a random sample of West Germans. A questionnaire was distributed to employees of a large government agency, asking them to evaluate the following: a hypothetical man engaged in a cigarette advertising business with or without giving away free marihuana (cases 4 and 3, respectively); a man engaged in newspaper advertising ("normal" or baseline case 1); or a man in the same business but giving away free marihuana (case 2). Each subject got only one case for evaluation. Slightly over 50 percent of the 236 answering subjects were smokers. Evaluation of differences in attitudes to the described cases led to conclusions that (1) social distance to the cigarette advertising manager is not greater than that of the newspaper advertising manager; (2) the profession of cigarette advertising is not held against the man; (3) the man in case 3 is regarded as well as the man in case 1; and (4) the cigarette business is tolerated (case 3) but dealing with marihuana (case 4) is not.

82–1802. Shephard, R. J.; Corey, P.; Cox, M. **Health Hazard Appraisal—The Influence of an Employee Fitness Program.** *Canadian Journal of Public Health* 73(3): 183-187, May-June 1982.

Among volunteer employees who participated in a Health Hazard Appraisal fitness program, four of five categories of men and three of five categories of women showed a statistically significant decrease in average cigarette consumption. The proportion of smokers in the program was substantially less than the general Canadian population (28.8 percent of the men and 29.2 percent of the women). The sample also had a below-average composite risk, but there remained a substantial residual difference between the appraised age and the compliance age (possible through adoption of an optimum lifestyle). The initial composite risk was correlated with cigarette smoking and a high exercise heart rate, but also with a large lean mass, large lung volumes, large predicted maximum oxygen intake (Jette prediction, women only), and a strong handgrip (men only). After 9 months of the program, subjects at the test company tended to an improvement of appraised age. Several other risk variables, including increased physical activity, decreased alcohol consumption, and reduced blood pressure, contributed to the risk reduction. (Auth. Abs. Mod.)

82–1803. Wake, R. **Teenagers and Tobacco.** *Cancer Forum* 5(1): 25-27, March 1981.

In a discussion of the problem of adolescent smoking, factors in the initiation of smoking are reviewed, including (1) viewing smoking as a sign of maturity and sophistication by the adolescent anxious to assert his/her independence; (2) little concern about the long-range health effects, even though the hazards may be known intellectually; (3) influence of parents and/or friends who smoke; and (4) advertising. Early educational prevention efforts are examined with emphasis on the importance of parental role models for young children, and the need for more effective measures for adolescents, such as the "natural leader" approach. The peer leader concept is now considered the most promising approach to prevention for young teenagers: it not only delivers the message in understandable style, but also teaches coping skills through role-playing, to impart confidence to the would-be nonsmoker. Another approach which may prove valuable for adolescent girls stresses the negative effects on pregnancy, since motherhood is a near and important event, compared to the more long-term health effects of smoking. The problem of adolescent addiction is also addressed, and the major role of tobacco advertising in the initiation of smoking by adolescents, since it often focuses on "young adult" models, is explored.

See also, **82–1766, 82–1783**

SMOKING CESSATION METHODS

82–1804. Fagerstrom, K.-O.; Storm, H.-G. **The Effects of Different Instructions on Smoking Cessation for Individuals With Different Degrees of Dependence.** *Behavioural Psychotherapy* 9(4): 310-315, 1981.

Placebo chewing gum proved more effective in a smoking cessation program if the subjects, especially the more nicotine-dependent, were told that the gum contained nicotine. The smoking cessation rate was studied after administration of a placebo smoking withdrawal chewing gum. Two groups from two factories were recruited and given instructions that they were to receive an effective chewing gum for smoking cessation. One group was told that the gum contained nicotine while the other was told that it worked through its special taste. At followup periods of 1, 3, and 8 weeks, those given the nicotine instructions achieved results superior to those given the taste instructions, although not significantly so. When nicotine dependence was taken into consideration it was found that the more nicotine-dependent subjects responded significantly better to the nicotine instructions as compared to the less dependent subjects. The less dependent subjects, however, had a greater overall success rate as measured at 8 weeks. It seems possible to conclude that instructions and expectations of nicotine content have a powerful "placebo" effect when administered to high nicotine-dependent subjects. (Auth. Abs. Mod.)

82–1805. Moyle, J. **The First Puff Is the Deepest?** *Nursing Mirror* 154(10): 26-27, March 1982.

It is proposed that the answer to cigarette addiction is to simply stop inhaling. The smoker may still enjoy the incidental actions and sensations which are involved in lighting a cigarette, while in effect not smoking. An abrupt break of an old ingrained habit requires a large effort of will. However, a small effort is sufficient if it does not entail breaking a routine. By not inhaling, the smoker avoids the dangers which accompany the custom, while the routine remains intact. The only effort required is to remember not to inhale. After the practice of inhaling has ceased, the power of the habit is broken and the smoker may quit altogether.

82–1806. O'Connor, K. P.; Stravynski, A. **Evaluation of a Smoking Typology by Use of a Specific Behavioural Substitution Method of Self-Control.** *Behaviour Research and Therapy* 20(3): 279-288, 1982.

Twenty-four volunteer smokers were scored on a Situational Smoking Questionnaire and divided into high- and low-activity groups on the basis of main cravings. High-activity smokers were further classified into those who smoked either under emotional stress or to aid concentration, while low-activity smokers were subdivided into those who smoked to relieve boredom or to relax. Smokers were then allocated at random to one of three treatment groups: (1) a behavioral substitution group where alternative relaxation, emotional coping, and distraction activities were rehearsed according to hierarchy of situational targets; (2) a generalized coverant approach where emphasis was placed on generalized beliefs about smoking effects rather than situational ones; and (3) a no-treatment control group, where subjects only self-monitored their smoking over the treatment period. Smokers completed weekly diaries for a baseline session, four treatment sessions, and 2- and 8-month followup periods. Both treatment groups showed a reduction in smoking levels over treatment sessions, but the behavioral group maintained a significantly lower level on session 4, and at followup. Those smokers initially classified as high-activity smokers benefited most from the behavioral program. Measures of attitude toward smoking behavior changed most significantly in the coverant group. The coverant approach appeared to increase motivation to cut down, and might in future studies be effectively employed together with the behavioral method. (Auth. Abs. Mod.)

82–1807. Woeber, K.; Bauermann, E. IV. **Therapie des Rauchens. Typendiagnostik und Behandlung des Rauchers. [4. Therapy of Smoking. Diagnosis of Smoking Type and Treatment of Smokers.]** In: Deutsche Haupstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive and Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 145-150, German.

Each smoker is different from another; thus a diagnosis as precise as possible of a smoker's type of habit must precede withdrawal treatment. The treatment method must be adapted to the particular type of smoker, and after-cure (health education and training) must follow any therapy. These are the fundamental findings of various studies, decade-long observations, and questionnaire surveys of smoking patients. For all practical purposes, the smokers are subdivided into four classes: occasional, regular, psychologically or physically dependent, and multidependent (on alcohol or drugs) smokers. Diagnosis of the smoking type is possible by means of three to four questionnaires. The role of the physician is described in the treatment of each type of smoker, as well as the treatment method to be applied, particularly in the case of classes 2 and 3. The class 4 smoker does not respond positively to any given method of treatment. Quitting smoking by gradually reducing consumption over a period of days or weeks is questionable, and with type 3 (dependent) smokers, not at all possible. Health education and training of ex-smokers appear to be appropriate methods for achieving long-term smoking cessation by stimulating changes in attitude and reinforcing motivation.

See also, **82–1762, 82–1776**

TOBACCO PRODUCT ADDITIVES

82–1808. Boden, R. M. **Flavoring with 1-Ethoxy-1-Ethanol Acetate.** United States Patent No. 4,296,137, October 20, 1981, 13 pp.

The compound presented may be used as a flavorant alone or combined with carbohydrates to generate acetaldehyde. When added to tobacco, the 1-ethoxy-1-ethanol acetate described enhances the sweet, fruity, winey, fresh, and berry characteristics of the smoking composition, both prior to and on smoking, by imparting a freshness to the smoke. This flavorant is more stable than acetaldehyde, and liberates an unexpected, advantageous acetaldehyde nuance in the smoking tobacco. The compound can be obtained in pure form by conventional purification techniques, such as distillation or chromatography. Recommended proportions by weight are between 0.05:1 to 0.50:1 of flavorant to smoking material. The additive may be incorporated in cigarette, cigar, or pipe tobacco, tobacco dust or fines, filter tip materials, seam paste, and other components.

82–1809. Boden, R. M. **Flavoring With 1-N-Butoxy-1-Ethanol Acetate.** United States Patent No. 4,296,138, October 20, 1981, 14 pp.

The compound presented may be used as a flavorant alone or combined with carbohydrates to generate acetaldehyde. When added to tobacco, the 1-N-butoxy-1-ethanol acetate described enhances the sweet, fruity, winey, fresh, and berry characteristics of the smoking composition, both prior to and on smoking, by imparting a freshness to the smoke. This flavorant is more stable than acetaldehyde, and liberates an unexpected advantageous acetaldehyde nuance in the smoking tobacco. Recommended proportions by weight are between 0.05:1 and 0.50:1 of flavorant to smoking material. The additive may be incorporated in cigarette, cigar, or pipe tobacco, filter, or paper wrapper.

82–1810. Mussinan, C. J.; Mookherjee, B. D.; Vock, M. H.; Schmitt, F. L.; Granda, E. J.; Vinals, J. F.; Kiwala, J. **Derivatives of CIS-3-Hexenol and Process for Producing Compositions of Matter Containing CIS-3-Hexenal and Products Produced Thereby and Organoleptic Uses Thereof.** United States Patent No. 4,312,766, January 26, 1982, 23 pp.

CiS-3-hexenyl derivatives useful for their organoleptic properties in smoking tobacco and other products are described. Sweet, fruity, cooling, and green aromas and tastes are provided in connection with augmenting or enhancing the aromas or flavors of smoking tobacco prior to and on smoking in both the mainstream and the sidestream. Satisfactory results are obtained if the proportions by weight of the sum total of cis-3-hexenyl derivatives used to flavor material is between 0.05:1 and 0.5:1. A listing of synthetic materials (including eugenol) and natural oils (including cocoa extract) which may be added along with the cis-3-hexanyl derivatives to the tobacco material, filter, or wrapper, or to a filter which is part of the smoking article is provided.

82–1811. Rowsell, D. G.; Gascoyne, J. M.; Hems, R. **Cyclic Carboxamides Having a Physiological Cooling Effect.** United States Patent No. 4,296,093, October 20, 1981, 8 pp.

The cyclic carboxamides described produce a strong physiological cooling effect similar to that of menthol, but do not have the minty odor of menthol, and are more stable in storage. They may be incorporated directly into tobacco, giving a cooling effect without altering any of the natural tobacco flavors. As little as 0.1 mg is effective. The compositions may be added more advantageously to pipe or cigarette filters, and are suitable also for flavoring lotions, toothpastes, sweets, soaps, soft drinks, toothpicks, and other products.

82–1812. Rowsell, D. G.; Spring, D. J.; Hems, R. **Acyclic Carboxamides Having a Physiological Cooling Effect.** United States Patent No. 4,296,255, October 20, 1981, 11 pp.

The carboxamides described may be incorporated directly into tobacco to give a cool effect when smoking, but without the attendant strong, characteristic odor associated with menthol. These coolants may be even more advantageously utilized in pipe or cigarette filters, by impregnation with an alcoholic solution of the compounds and then dried. The amides may be readily prepared by conventional techniques, such as reacting certain acid chlorides with the appropriate amine in the presence of a hydrogen chloride acceptor. The described compounds are also useful in lotions, toothpastes, sweets, ointments, soaps, toothpicks, shampoos, deodorants, and medicaments.

82–1813. Schweizerische Rundschau fuer Medizin (PRAXIS). **Das Brennende Problem "Raucherwaren." [Fire Problem With "Smoking Products."]** *Schweizerische Rundschau fuer Medizin (PRAXIS)* 71(10): 3, 1982, German.

The problem of rapid-burning and incandescent admixtures in cigarettes is addressed from the viewpoint of increasing fire risk. It is also suspected that these admixtures represent an additional risk for respiratory organs. According to Swiss regulations, a total maximum 25 percent of these admixtures is allowed in cigarettes. The allowed admixtures are: aluminum hydroxide; aluminum oxide; aluminum and silicon heteroxide; aluminum sulfate alum; silica; talc; titanium dioxide; magnesia; potassium nitrate; carbonic, acetic, malic, citric, tartaric, lactic, and formic acids or their potassium, sodium, calcium, and magnesium salts; and ammonium, potassium, calcium, magnesium, and sodium phosphates.

82–1814. Seeman, J. I. **Smoking Compositions.** United States Patent No. 4,312,367, January 26, 1982, 9 pp.

Low-tar and -nicotine delivery smoking compositions containing a quarternary ammonium compound as an additive to tobacco and nontobacco products are described. The additives are characterized by low volatility and low pack aroma and are adapted to impart improved flavoring to the mainstream smoke and improved aroma to the sidestream smoke. This additive also serves as a humectant and antistaling agent during the preparation and storage of the smoking compositions.

82–1815. Simon, E. **Self-Extinguishing Cigarettes.** United States Patent No. 4,303,084, December 1, 1981, 5 pp.

A class of coatings are applied to the paper wrapper that encloses a cigarette's smoking medium to limit in a predictable manner the free burning time of the treated cigarette, or to control the lapsed time prior to self-extinguishment after being lit and left unattended. The coating materials are generally polymeric chlorine-containing film-forming latexes that cure at ambient temperature, forming flexible, water-resistant, flame-retardant deposits. Comparable results are obtained, for equivalent weight percentages of the deposited latex solids, by either precoating the portion of paper wrapper that encloses the charge of tobacco or smoking medium or by posttreatment of the external surface of the assembled cigarette. For cigarettes having paper wrappers coated to contain 19±6 percent by weight of latex solids of this material, a self-extinguishing time of from 1 to 3 min can be expected. This meets a practicable compromise for the smoker between safety and smoking convenience. (Auth. Abs. Mod.)

82–1816. Sprecker, M. A.; Hall, J. B. **Substituted Tricyclodecane Derivatives, Processes for Producing Same and Organoleptic Uses Thereof.** United States Patent No. 4,275,251, June 23, 1981, 63 pp.

Substituted tricyclodecane derivatives, which can act as organoleptic agents in tobacco and a variety of other materials, and processes for preparing such substituted tricyclodecane derivatives and for using the derivatives for their organoleptic properties are described. For chewing tobaccos, as well as foodstuffs, chewing gums, and medicinal products, the derivatives have sweet, cocoa butter-like, herbaceous, green, floral, and anise aroma profiles and sweet, cocoa butter-like, herbaceous, green, floral, and anise flavor profiles. For smoking tobacco flavors, the derivatives have cocoa-like, sweet, floral, and green aroma profiles prior to smoking and sweet, floral, and aromatic aroma and taste nuances on smoking in both the mainstream and the sidestream. When the derivatives are used in a flavoring composition, they can be combined with conventional flavoring materials or adjuvants. (Auth. Abs. Mod.)

82–1817. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Use of 1,3,5,5-Tetramethyl-2-Oxabicyclo(2.2.2)Octane in Augmenting or Enhancing the Aroma or Taste of Foodstuffs.** United States Patent No. 4,269,862, May 26, 1981, 21 pp.

The uses of 1,3,5,5-tetramethyl-2-oxabicyclo(2.2.2)octane as an aroma and flavor enhancer, and its synthesis, is described. When added to tobacco, it imparts sweet, earthy, cooling, and citrus-like notes prior to and on smoking in both the mainstream and sidestream smoke. The compound may be added to the tobacco, a tobacco substitute, filter, wrapper, or other materials comprising the smoking product. The recommended proportion by weight of flavorant to tobacco is 0.05:1 to 0.50:1. This derivative is useful also in perfumes, foodstuffs, chewing gums, toothpaste, and medicinal products.

82–1818. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Use of Cyclic Chemical Compounds for Augmenting or Enhancing the Aroma of Fabric Softener Articles.** United States Patent No. 4,301,018, November 17, 1981, 45 pp.

The organoleptic uses of oxabicyclooctane derivatives and the cyclohexene alkyl and alkenyl carbinols and esters thereof, in tobacco and tobacco substitutes, as well as in a large variety of other materials, are described. The organoleptic agents may increase or enhance the natural tobacco-like properties of smoking tobacco by providing hay, fruity, and herbaceous aroma and taste nuances before and during smoking in mainstream and in sidestream smoke. Other flavoring and aroma additives may also be used. The compounds may be added to smoking tobacco material, the filter or the wrapper or a filter which forms part of the smoking article, and for other materials, including perfumes, perfumed articles, foodstuffs, colognes, chewing gums, medicinal products, and toothpastes.

82–1819. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Cyclohexenemethanols and Lower Alkyl Esters Thereof.** United States Patent No. 4,301,309, November 17, 1981, 47 pp.

Derivatives of 2-oxabicyclo(2.2.2)octanes and their cyclohexenemethanol and lower alkyl ester precursors are described. These derivatives and their esters augment or enhance the natural tobacco-like characteristics of smoking tobacco by imparting thereto hay, fruity, and herbaceous aromas and taste nuances in mainstream and sidestream smoke. Other flavoring and aroma additives may be added to the smoking tobacco material or substitute. The derivatives and/or cyclohexenemethanols may be added to the smoking tobacco material, the filter or the wrapper or to a filter which is part of the smoking article. The tobacco may be shredded, cured, cased, blended or reconstituted tobacco or a tobacco substitute. (Auth. Abs. Mod.)

82–1820. Sprecker, M. A.; Schmitt, F. L.; Vock, M. H.; Vinals, J. F.; Kiwala, J. **Process for Augmenting or Enhancing the Aroma of a Detergent Using 2,4,6-Trimethylcyclohexanemethanol and Derivatives.** United States Patent No. 4,301,022, November 17, 1981, 22 pp.

2,4,6-Trimethylcyclohexanemethanol and derivatives, which have organoleptic properties that impart sweet, floral, green, weedy, and herbaceous aromas prior to and on smoking in the mainstream and in the sidestream while enhancing certain specialty smoking tobacco-like notes, are described. These compounds, and if desired, one or more additional flavorings (including eugenol and cocoa extract) may be added to the smoking tobacco material, filter, or wrapper, or to a filter which is part of a smoking article. Satisfactory results are obtained if the proportion by weight of the additive compound is between 0.05:1 and 0.50:1 of the active ingredients to the smoking tobacco material, or 50 ppm and 1,500 ppm.

82–1821. Thomas, A. F. **Cis-10,10-Dimethyl-Tricyclo(7.1.1.0^{2,7})-Undec-2-en-4-one and Use Thereof as Perfuming and Flavoring Ingredient.** United States Patent No. 4,342,320, August 3, 1982, 4 pp.

The synthesis and organoleptic properties of a new tricyclic carbonyl derivative, defined as cis-10,10-dimethyl-tricyclo(7.1.1.0^{2,7})undec-2-en-4-one, is described. The compound is formed by reacting nopinone with pyrrolidine in the presence of an acidic dehydrating agent. The resulting enamine is then reacted with but-1-en-3-one under the conditions of the Michael reaction. The final step is a reaction with an alkali metal, yielding the tricyclic carbonyl derivative. When added to tobacco, the manufactured cigarettes display an herbal note and stronger natural odor prior to smoking, and a more pronounced sweet, herbal, and coumarine odor upon lighting.

82–1822. Wu, D.L.S.; Swain, J. W. **Reaction Flavors for Smoking Products.** United States Patent No. 4,306,577, December 22, 1981, 6 pp.

Reaction flavors for smoking compositions are disclosed. The flavors are prepared by reacting reducing sugars and selected amino acids in the presence of ammonium hydroxide and optionally in the presence of an aldehyde in an essentially solvent-free system. The prepared flavors may be incorporated in tobacco, reconstituted tobacco, nontobacco substitutes, or mixtures thereof. The reaction flavors can, if desired, be incorporated in cigarette paper or in filters. However, it will generally be more desirable to incorporate the flavors in the smoking material itself. (Auth. Abs. Mod.)

TOBACCO MANUFACTURING AND PROCESSING

82–1823. Bolt, A.J.N.; Chard, B. C. **Smokeable Device.** United States Patent No. 4,340,072, July 20, 1982, 5 pp.

A cigarette-simulating smokeable device is described for controlling objectionable substances such as carbon monoxide, which are released into the mainstream smoke. The loss of inhalant material by pyrolysis and to sidestream during smolder is also minimized. The device has an annular fuel rod of reconstituted tobacco and/or a tobacco substitute in gaseous communication with a mouth-end chamber. The chamber contains a quantity of inhalant material which, when contacted by hot gases during smoking, forms an aerosol for inhalation by the smoker. The inhalant material may comprise nicotine source material or spray dried granules of flavorant whose composition lies within the range 30-60 percent by weight of a solution of flavorant in triacetin or benzyl benzoate encapsulated in 40-70 percent by weight of gum acacia or a modified starch. The inhalant material may further comprise microcapsules formed by the coacervation method which capsules comprise 50-80 percent by weight of flavorant in a mixture of gelatin and gum acacia. (Auth. Abs. Mod.)

82–1824. Cantrell, D. V. **Cigarette Filter.** United States Patent No. 4,343,319, August 10, 1982, 7 pp.

A cigarette filter which lowers tar by combining ventilation and filtration is described. The filter consists of a porous filter rod circumscribed by a smoke-impervious plug wrap. The filter rod has at least one opening in each end, with at least one longitudinal groove embedded into the outer periphery of the rod and the wrapper. The filter rod is wrapped with nonporous plug wrap and overlaid with porous tipping material with the flow-through groove between the plug wrap and the tipping material. A portion of the smoke coming from a tobacco column mixes with ventilating air in the groove and the remaining portion of the tobacco smoke passes through the filter, with little or no contact with ventilating air until it reaches the smoker's mouth. (Auth. Abs. Mod.)

82–1825. de la Burde, R. Z.; Aument, P. E. **Expanding Tobacco.** United States Patent No. 4,340,073, July 20, 1982, 10 pp.

A process for expanding tobacco is provided which employs liquid carbon dioxide as the expansion agent. Expanding tobacco increases the filling power of tobacco so that a smaller amount of tobacco is required to produce a cigarette which has the same firmness but lower tar and nicotine than a cigarette containing nonexpanded tobacco. Tobacco is contacted and thoroughly impregnated with liquid carbon dioxide. The carbon dioxide-impregnated tobacco is maintained at a temperature no lower than -2°C and under conditions of temperature and elevated pressure such that all or substantially all of the carbon dioxide which is in contact with the tobacco is in liquid form. After the impregnation has been completed, any excess liquid carbon dioxide which may be present may be removed from the tobacco. The elevated pressure is reduced to convert the liquid carbon dioxide to solid carbon dioxide within the tobacco structure. The resulting solid carbon dioxide-containing tobacco is subjected to conditions of temperature and pressure, preferably by rapid heating at atmospheric pressure, such that the solid carbon dioxide is vaporized and the tobacco is thereby expanded. Apparatus for carrying out the process is also provided. (Auth. Abs. Mod.)

82–1826. Kehoe, G.; Puglia, W. J.; Witzel, F.; Friello, D. R.; Mackay, D.A.M. **Tobacco-Flavored Chewing Gum.** United States Patent No. 4,317,837, March 2, 1982, 5 pp.

To fulfill a need for a tobacco-like product which is of the smokeless variety and gives the sensation and pleasure generated from chewing tobacco, a tobacco-flavored chewing gum is provided which does not decrease in viscosity upon chewing. It is essentially free of water-solubles, and includes gum base containing high levels of inert fillers, such as calcium carbonate or talc in amounts of at least about 50 percent by weight, softeners such as lecithin, synthetic noncaloric sweeteners such as aspartame, saccharin or cyclamates, and tobacco flavors and/or natural tobacco. The chewing gum includes at least about 10 percent air voids entrapped in the gum base matrix which, upon chewing, become filled with moisture causing a dramatic increase in cud volume such as experienced when chewing tobacco. (Auth. Abs. Mod.)

82–1827. Keritsis, G. D.; Lowitz, D. A. **Method for Employing Tobacco Dust in a Paper-Making Type Preparation of Reconstituted Tobacco and the Smoking Material Produced Thereby.** United States Patent No. 4,341,228, July 27, 1982, 8 pp.

Tobacco dust byproduct material is economically used in a papermaking type process for producing reconstituted tobacco. The tobacco dust is mixed with a bonding material to form a mixture, which is treated to form agglomerated particles. The agglomerated particles are mixed with a tobacco-parts slurry which is formed into a sheet by means of a papermaking process. After drying and shredding, the resultant reconstituted tobacco sheet may be used as a smoking material alone or in combination with other smoking materials such as natural leaf tobacco. (Auth. Abs. Mod.)

82–1828. Luke, J. A. **Production of Tobacco-Smoke Filters.** United States Patent No. 4,292,032, September 29, 1981, 6 pp.

A method for producing rod-shaped tobacco-smoke filter components, each having one or more annular tobacco-smoke barriers, is described. Annular grooves open at their peripheries are formed in rod-shaped bodies of smoke-filtration material; the grooved bodies are conveyed in a direction transverse to their longitudinal axes along an arcuate path, during which time settable barrier material is deposited in the grooves. The bodies are rotated about their axes during the deposition of the barrier material. Depositing the barrier material involves a rotary disc member or members which extends or extend into the groove or grooves of each grooved body and a bath of the said material on which the rotary disc member or members is or are mounted so as to dip into the said material. (Auth. Abs. Mod.)

82–1829. Marritt, C. R.; Williams, R. E. **Manufacture of Filter-Tipped Cigarettes.** United States Patent No. 3,999,559 (Re. 30,865), February 16, 1982 (Reissued), 15 pp.

Filter-tipped cigarettes are produced by dividing a continuous tobacco stream into alternate sections having respective lengths corresponding to a double cigarette filler and a double filter portion. The double filter portion sections are removed from the stream to leave spaced double filler sections which proceed to a position where double filter portions are inserted in the gaps left by the removed sections. The stream of alternating tobacco sections and filter portions is continuously wrapped and subsequently cut at the middles of the tobacco sections and filter portions to produce individual filter cigarettes. The sections of tobacco removed from the continuous stream may be returned to the stream. (Auth. Abs.)

82–1830. Miller, W. L. **Smoking Pipe.** United States Patent No. 4,275,747, June 30, 1981, 4 pp.

A smoking pipe with a small metal air injection pin in its stem is described. An inner condenser portion extending into the smoke-conducting passage of the stem serves as a port for introducing air into the smoke stream; an outer heat-radiating portion between the bowl and mouthpiece opening also serves as a port for allowing air to enter the pipe stem. The pin passageway is perpendicular to the stem passage and terminates about halfway across the passage. Thus, the inner portion not only contacts the smoke, but also the velocity of the smoke, moving directly across the open inner end of the passageway, aspirates outside air into the flow of smoke in proportion to the velocity of the smoke, where it is mixed in the stem as the smoke and air flow to the mouthpiece opening. The desirable effect of cooling the smoke and condensing moisture and tar, resulting in cool, dry, and mild smoking characteristics, is achieved. (Auth. Abs. Mod.)

82–1831. Muto, R. **Cigarette Filter With Oblique Partitions.** United States Patent No. 4,343,320, August 10, 1982, 4 pp.

A filter assembly for removing components such as tar and carbon monoxide from the smoke of cigarettes and the like is described. The assembly comprises an elongated tube with an axial bore, having a plurality of fan-folded interior cross partitions each of oval outline, and extending in a plane oblique to the axis of the bore to form a succession of sealed chambers. The partitions have staggered perforations to allow the filtered substance to travel to the downstream end of the tube in an undulated path. At the upstream end of the tube, the exterior wall of the first sealed chamber has a pattern of intake pin holes. These allow fresh air to cool the smoke and to convert carbon monoxide to carbon dioxide. At the downstream end of the tube, the sealed chamber contains aromatic unburned tobacco for restoring and enriching the flavor of the smoke. The remaining chambers contain cotton batting to filter out undesirable substances such as tar. (Auth. Abs. Mod.)

82–1832. Sanford, R. A. **Cigarette Filter.** United States Patent No. 4,342,322, August 3, 1982, 5 pp.

A cigarette filter for lowering tar predominantly by ventilation instead of filtration is described. The smoke stream is directed to a filter in the center while grooves between the filter and the nonporous wrap simultaneously direct ventilating air along the outer surface. The filter includes a porous filter rod having a hollow tube extending from one end to a preselected distance. The filter rod is circumscribed by a nonporous wrapper, with at least one groove between them for ventilation. Tipping material circumscribes the nonporous wrapper and is provided with flow-through openings in flow communication with the groove. The groove may be oriented to extend to the mouth end of the filter or the tobacco end when connected to a cigarette. (Auth. Abs. Mod.)

82-1833. Sanford, R. A.; Johnson, R. R. **Cigarette Filter.** United States Patent No. 4,338,956, July 13, 1982, 4 pp.

A means of diluting the smoke delivered through a filter is provided by using a porous filter rod circumscribed by a nonporous wrapper. The wrapper is provided with longitudinal grooves, which are in communication with openings in the tipping material. The grooves deliver air to the smoker's mouth, diluting the smoke provided through the filter. Support means are provided for the tipping material to maintain contact between the openings and the wrapper grooves. A filter element made from any fibrous or foamed material may be used with the filter rod described.

82-1834. Sawada, H.; Shinohara, K.; Shiga, M. **Filter for Cigarette Smoke.** United States Patent No. 4,292,984, October 6, 1981, 6 pp.

A cigarette filter composed of a bundle of cellulose acetate filaments is described. The filament bundle has at least two different filament denier sizes randomly distributed in the direction perpendicular to the longitudinal axis of the filter. The cigarette filter has a high tar filtration efficiency and a moderately low pressure drop, and cigarettes with this filter are easy to smoke. The smoke particles are fine and the taste is mellow. (Auth. Abs. Mod.)

82-1835. Tudor, T. T. **Cigarette Material Having Non-Lipsticking Properties.** United States Patent No. 4,340,074, July 20, 1982, 4 pp.

A cigarette tipping material with nonlipsticking properties, having air ventilating perforations for mixing air with the smoke stream at the filter end of a cigarette is described. Preselected areas of the tipping material are covered with the nonlipsticking coating. The perforations which provide ventilation are added in non-coated areas. This eliminates clogging of the perforations and provides consistent mixing of air with the smokestream. A series of synchronized rollers are described for coating, then perforating the tipping material: the tipping material may also be perforated prior to coating.

TOBACCO ECONOMICS

82-1836. Advertising Age. **100 Leading National Advertisers (Total Ad Dollars in Millions: 1980).** *Advertising Age* 52(38): 148 pp., September 10, 1981.

Marketing data for the nation's 100 top advertisers of 1980 are presented including ad expenditures, media mix, sales, profits, and share of market for leading brands. The tobacco industry is represented by Philip Morris Inc., R. J. Reynolds Industries, B.A.T. Industries, American Brands, and Liggett Group. In total ad dollars, Philip Morris Inc. is ranked fourth nationally and R. J. Reynolds Industries is seventh. The six major cigarette companies continue to battle for a bigger share of the low-tar cigarette market that accounts for about 50 percent of the total cigarette market. The ultralow-tar (5 mgs tar and below) category was the scene of the year's most aggressive marketing, with tobacco companies increasing advertising expenditures and marketing new brands. Marketing profiles are provided for each of the 100 advertisers.

82-1837. Ahmad, M. **Pakistan: 1981 FCV Crop Short, High-Priced; 1982's Plagued by Weather Problems.** *Tobacco International* 184(14): 5, 7-10, July 9, 1982.

Antismoking propaganda, inflationary trends in raw material prices, and a short tobacco crop were the three main causes which arrested the normal growth of the Pakistan cigarette industry since January 1981. Antismoking claims about the health hazards of smoking have become a normal feature on Pakistan TV and radio. Because of the high tobacco prices (minimum procurement prices of tobacco increased 38 percent between 1979 and 1982), Pakistan has outpriced herself from the international trade in unmanufactured tobacco. Export of manufactured tobacco, however, has been growing. The three major forms of manufactured tobacco are cigarettes, bidis, and "gazare," a smokeless tobacco. The Pakistan tobacco industry produced 37.3 billion cigarettes in 1981, a 2 percent increase over the previous year. Pakistani smokers, with their rising standards of living, are showing a marked preference for filter cigarettes. This has resulted in a 23 percent increase over the last decade for filter cigarettes which now make up 38 percent of the total market.

82-1838. Humphries, B. **No-Name Products Make a Name for Themselves.** *Tobacco Reporter* 109(8): 38-40, August 1982.

Generic, or no-name, cigarettes produced by Liggett and Myers have shown steady growth for the past 18 months after a startling initial success. They have established a firm position in the 21.1 billion dollar/year U.S. cigarette market with a 1.0 percent share predicted by December 1982 (>200 million dollars). Gary Tobacco Company, established as a Liggett subsidiary to handle the generics, at first produced solely a king-size filter cigarette with tobacco blends closely duplicating the top-selling brands. Gary has expanded its offerings to include king-size menthol, 100 mm filter, and 100 mm menthol. For maximum appeal, the blend created is light, with a tar rating of 14 mg. Plain packaging and no national advertising by Liggett has made possible savings of 1 to 1.20

dollars per carton translating to an annual saving of 73 dollars for a two-pack-a-day smoker. Although no substantiating figures exist, observers believe that many consumers of generics might otherwise have stopped smoking because of rising costs. Liggett is studying the feasibility of expanding the generics into international markets.

82–1839. Maxwell, J. C., Jr. **The Maxwell Report: Cigar Volume Dips in 1981.** *Tobacco Reporter* 109(8): 47, August 1982.

Unit volume information for the 1980 and 1981 little and large cigar market is detailed in tabular form. The industry's unit volume declined modestly in 1981 and a flattening or slight drop is anticipated in 1982. The industry's major trend has been the move to higher-price products. Premium products now cost 50 cents or more and account for 5 percent of units and 25 percent of dollars for the industry.

82–1840. Neuber, D.; Rizio, D. **U.S. Cigarette Companies Bank on the Power of Suggestion.** *Tabak Journal International* 2: 134-135, April 1982.

U.S. cigarette companies spent over 518,883,100 dollars in advertising in 1980, in attempts to establish a certain personality for their brands, and subsequently gain a desirable following. These campaigns have established the tobacco industry as the leading advertiser in three categories: magazine, outdoor, and newspaper. R. J. Reynolds Tobacco Co., with 172,258,800 dollars spent, was the top tobacco advertiser, followed by Philip Morris (161,286,600), BAT (67,426,900), American Brands (47,000,000), Loews (65,407,000), and Grand Metropolitan (503,800). The 10 best selling brands in 1980 were Marlboro, Winston, Salem, Kool, Pall Mall, Benson and Hedges, Camel, Merit, Vantage, and Kent. Advertising budgets for a single brand ranged from 36,977,900 dollars for Salem to 7,800,000 for Pall Mall. A new marketing concept, the "family brand," was introduced in 1981: low-tar or menthol extensions of existing brands were generated, and the same graphic execution and copy were used for the entire family line. Innovative promotions, such as the Kool Jazz Festivals, and the Merit Report, were instituted and carried through 1981. Marlboro, however, maintained its number one position in the United States and in international markets. (Auth. Abs. Mod.)

82–1841. Scott-Malden, J. N. **Brighter Prospects for the U.K. Tobacco Industry?** *Tabak Journal International* 3: 222, 224-226, June 1982.

Cigarette tax increases are credited with producing a 9.2 percent fall in consumption for 1981 in the United Kingdom. Also, the difference in consumption between socioeconomic groups is widening: 57 percent of unskilled workers smoke vs. 21 percent of the professional group, reflecting growing health concerns. The introduction of a specific ad valorem system in place of the old weight-related system made the king-size cigarette proportionately less expensive, transforming it into the most popular segment of the market, rising from 11 to 80 percent of total sales. This led to a price war and erosion of profitability as manufacturers concentrated on grabbing as large a share of this growing market as possible. Thus increasing taxes, declining consumption, and the destructive price war paint a bleak outlook for the U.K. tobacco industry. However, the 10 percent increase in hand-rolling cigarette tobacco, the 43 percent growth of exports between 1976 and 1980, and the departure of Sir George Young from the Department of Health are promising aspects for the industry. The new Health Minister prefers a continuation of the voluntary code, and a ban on advertising or sports sponsorship does not appear imminent. Sensible pricing policies and cutback measures are allowing the companies to cope with the diminishing market.

82–1842. Shelton, A. **Smokeless Sales Continue To Climb.** *Tobacco Reporter* 109(8): 42, 44, August 1982.

Smokeless tobacco products (snuff and chewing tobacco) continue to grow in both use and popularity in the United States. From 1970 to 1979, the sales of such products increased almost 32 percent, from 95 million to 125 million pounds. For 1981, the volume was almost 132 million pounds, up 1.7 percent from 1980. Several factors which may explain the resurgence in popularity of smokeless tobacco products include: (1) increased restrictions on smoking in industries where it is either dangerous and/or prohibited; (2) enhanced public image of smokeless tobacco through advertising and promotion campaigns; (3) the lower price of smokeless products; and (4) improved packaging and the use of milder brands. At present, smokeless tobacco products are not required to have health warnings, nor are they prohibited from television and radio advertising.

82–1843. Tabak Journal International. **U.S. Angered by Japan's Import Laws.** *Tabak Journal International* 3: 208-209, June 1982.

Despite liberalization of Japan's import policy on cigarettes, the marketing of American and other foreign brands in Japan remains a complex and restrictive affair. In response to negotiations with the United States, Japan has recently lessened the price gap between domestic and foreign brands by lowering duties from 90 percent to 35 percent. Also, the number of retailers allowed to carry the imports was raised from 14,000 to 20,000, and the prohibition on media advertising for imports was dropped. Nevertheless, the economic outlook is still considered unpromising. Imported cigarettes had only a 1.4 percent market share in 1981, with 96 percent of that total being American brands. Federal government officials are pushing legislation that would impose similar sanctions on Japanese products entering the United States. (Auth. Abs. Mod.)

82-1844. Tabak Journal International. **Britain Has a Long Record of Bouncing Back After Adversity.** *Tabak Journal International* 3: 220-221, June 1982.

The survival of the British tobacco industry, which has weathered many problems since the Imperial Tobacco Company was founded in 1902 to rebuff attempts of the American Tobacco Company to invade the British market, is discussed. Adverse influences have been ever-mounting levels of taxation, national or international events affecting leaf supplies, and more recently, the change in public opinion toward smoking. The taxation problem involves not only rising tax rates, but also the alignment of the tax formula with that of the European community; removal of the price differential for smaller cigarettes; and a tax formula which makes coupons (which have been included in some cigarette packs as an incentive to sales) uneconomical. Industry optimism, created by 77 years of steady growth, finally was shaken as cigarette sales started declining in 1978, although many manufacturers had already begun to diversify into other areas. The smoking and health controversy, which began in earnest in Britain in 1962, is the greatest current challenge, although taxation is still much more effective than propaganda in reducing tobacco consumption.

82-1845. Tabak Journal International. **U.K. Statistics.** *Tabak Journal International* 3: 258-259, June 1982.

Economic statistics for Great Britain's tobacco industry are presented for the years 1961 through 1980. Listed are the domestic sales for tobacco products, expenditures on tobacco products, tobacco product exports and imports, production of tobacco products, and the import of unmanufactured tobacco. The number of cigarettes sold domestically has decreased in recent years, dropping from a 1973 peak of 137,400 million pieces to 121,500 million pieces in 1980. There have also been overall decreases in the sale of cigars/cigarillos, smoking tobacco (including pipe tobacco), and snuff. In contrast, spending on tobacco products has risen fairly steadily, reaching 4,264 million pounds for cigarettes and 603 million pounds for pipe tobacco, cigars, and snuff in 1980. Exports of cigarettes and cigars/cigarillos have also increased, the 1980 figures showing exports of 36,926 million cigarettes and 61 million cigars or cigarillos. The trends in imports, both for tobacco products and for unmanufactured tobacco, are uneven and less clear, as are the figures for production of tobacco products.

82-1846. Tobacco International. **Leaf Production Increases Planned for Several South African Varieties.** *Tobacco International* 184(11): 42, 44, May 28, 1982.

Despite recent problems, increases in tobacco production are planned in South Africa. At present, South Africa's 1982 tobacco crop is judged to be 5.5 percent larger than the 1981 crop. The 1981 crop experienced difficulties from drought and from chlorine in irrigation water. Cigarette sales displayed a rise of 7.7 percent in 1981 compared with 1980. However, a slowdown in consumption is expected for 1982. Mild cigarettes account for about 12.5 percent of the market, while filter-tipped cigarettes take up approximately 85 percent of total sales. With pressure from the Department of Health, cigarette producers have been putting tar and nicotine content data on cigarette packets since the beginning of 1982. It has been calculated that production will have to be raised by about 22.1 percent over a medium term of 2 or 3 years to meet demand. This applies to all tobacco types but air-cured. For the long term (up to 1988) production of all types will have to be raised by about 35.69 percent to meet demand.

82-1847. Tobacco International. **Zimbabwe: No More 100 Million kg Flue-Cured; Beans, Burley Instead.** *Tobacco International* 184(11): 10, 12, 14, 16, May 28, 1982.

Zimbabwe tobacco production has experienced recent difficulties, with interest growing in the use of Burley. Since Zimbabwe reached independence, attempts have been made to increase tobacco crops; flue-cured production rose from 68 million kilograms (kg) in 1980-1981 to approximately 90 million kg in 1981-1982; Burley rose from 2 million kg to 3 million kg or more over the same period. The 1980-1981 season was excellent, the weather helping to produce high quality crops, but the 1981-1982 season experienced uneven rainfall, resulting in a range of styles and qualities in the crop. Producers also experienced difficulties from a rise in the minimum agricultural wage, inability to mechanize as much as American farmers, and a lack of some agricultural supplies and machinery. Many believe Zimbabwe's flue-cured crop will not exceed 100 million kg again, because as labor costs continue to rise, growers will change from flue-cured tobacco to other crops more suitable for mechanization such as maize and soya beans. However, interest has been shown for Burley tobacco among both peasant farmers and large-scale commercial producers, due to the more stable prices, the consideration that management decisions do not have to be so precise, and the suitability of Burley for small-scale peasant farming. Recent grade, price, and sales information about various Zimbabwe tobaccos is provided.

82-1848. Tobacco International. **Dark Leaf Production May Yet Shrink in Colombia; Virginia Leaf on Rise.** *Tobacco International* 184(13): 54, 56, 58, June 25, 1982.

In Colombia, domestic consumption of dark-leaf tobacco increased 3 percent to 15,950 metric tons of farm sales weight. However, filter light-leaf cigarettes are increasing their market share and now stand at 60 percent of domestic cigarette production. Total per capita consumption for 1981 is estimated at 1,160 cigarettes, compared with 1,150 in 1980. The loose control of cigarette contraband, and high taxes on both domestic and legal cigarettes, are hurting local industry and legal importers. However, both legal and illegal imports are expected to increase 4 to 5 percent yearly as imports comprise one-third of total consumption. Total tobacco production increased 11 percent to 37,400 metric tons in 1981, while hectares planted increased 8 percent to 25,958. Virginia production rose 23 percent over 1980 figures, while Burley tobacco production gained only 1 percent due to lack of price adjustments, heavy rains, and competing rice crops in the irrigated areas. Attempts to improve agricultural practices, thus increasing leaf quality and nicotine content, are being made. On February 17, COLTABACO workers, representing 80 percent of national cigarette production, went on strike with no settlement foreseen, and on March 24, the Ministry of Health launched a national antismoking campaign intended to be permanent.

82–1849. Tobacco International. **Argentine Quality Improves; Agricultural Advancements Planned.** *Tobacco International* 184(13):42, 44, 45, June 25, 1982.

The type of Virginia tobacco grown in Argentina has changed from a low-nicotine, lemon, filler type to a high-nicotine, orange, full-flavored tobacco: dark-leaf tobacco production has virtually ceased. In 1981, 34 million kilos of Virginia tobacco were produced, a crop 20 percent lower than expected due to weather conditions. Measures to further improve leaf quality and production are in progress; barn space is being built, coops for farmers are providing working funds and protecting export markets, and efforts to increase yield, flavor, and nicotine content are increasing. An inflation rate of 140 percent, without wages keeping pace, resulted in an 11 percent drop in cigarette consumption. The use of roll-your-own tobacco has increased by 150 percent.

See also, **82–1850**

LEGISLATION

82–1850. Socolar, M. J. **Tobacco Program's Production Rights and Effects on Competition.** Report by the Comptroller General of the United States, U.S. General Accounting Office, CED-82-70, April 23, 1982, 39 pp.

The Department of Agriculture's tobacco program, through its acreage allotment, marketing quota, and price-support provisions, is affecting the competitiveness of the U.S. tobacco industry in world and domestic markets and the income of those farmers who grow tobacco. The high price of U.S. tobacco and the improved quality and increased production of foreign tobacco have contributed to the decline of the U.S. share of world tobacco trade and domestic use. Insufficient markets for U.S. flue-cured tobacco have resulted in a 32.1 percent reduction in the national flue-cured tobacco quota since 1975. Reductions in individual farm quotas in turn require tobacco producers to lease or rent quotas from others to maintain existing production levels. The Government Accounting Office found that 68 percent of the allotment and quota owners in 32 counties selected for review were not active farmers. Instead, they leased or rented their production and marketing rights to producers at rates ranging from 25 cents to 90 cents a pound. The Government's administrative expenses are estimated at 13.1 million dollars for 1981. Since the program began in the 1930s, the Government has lost approximately 57 million dollars in loan principal, and 591 million dollars from loan repayment practices. (Auth. Abs. Mod.)

82–1851. Tala, E. **Gesetzgeberische Aktionen gegen das Rauchen in Finnland. [Legislative Measures Against Smoking in Finland.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit: Politische Praventive und Therapeutiche Aspekie.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 13-16, German.

With the 1976 Finnish law on smoking-reducing measures, limitations on the tar, nicotine, and carbon monoxide levels in cigarette smoke were established. Labels warning against health damage from smoking were made compulsory on all tobacco products. Selling tobacco products to persons under 16 years and advertising were forbidden. Smoking in public transportation (airplanes, busses, taxicabs), kindergartens and schools, reception and waiting rooms of public offices, showrooms, and sport events open to children was strictly prohibited or limited. A public opinion survey at the time of the law's enactment indicated that 80 percent of the population agreed with most provisions. In addition, a program of antismoking measures, health education, and information for patients and personnel of hospitals was instituted. The absence of regulation of smoking at workplaces and the prices of tobacco products are considered to be the weak points of the law. The cigarette consumption in the general population is shown to have decreased after the law became effective, as well as the percentage of the regular smokers among the 14- to 18-year-old boys and girls. Also, the law is credited with swinging public opinion in favor of "nonsmoking."

82–1852. U.S. House of Representatives, 97th Congress, 2nd Session. **No Net Cost Tobacco Program Act of 1982 (H.R. 6590).** U.S. Congress, January 25, 1982, 20 pp.

The No Net Cost Tobacco Program Act of 1982 (Public Law 97-218) provides for the operation of the tobacco support and production program so as to incur no net cost to taxpayers and limits increases in the support price of tobacco. The Act stipulates that producers of quota tobacco will share equitably in helping to eliminate losses incurred in carrying out the price support program and will contribute to a capital account established by each producer-owned marketing association through which price support advances are made to producers. The capital account is to be used exclusively to achieve a no net cost tobacco program. Provisions of the Act detail: producer contributions to the no net cost tobacco fund, adjustment of price support level of tobacco, penalties related to marketing restrictions, lease and sale of acreage allotments and marketing quotas, marketing assessment to no net cost account, and the agricultural stabilization and conservation county and community committee system.

82–1853. Warner, K. E. **Regional Differences in State Legislation on Cigarette Smoking.** *Texas Business Review* 56(1): 27-29, January-February 1982.

State governments influence cigarette smoking through two types of legislation: excise taxes and the restriction of smoking in public places. Regional differences between the six "tobacco" states and the rest of the country are discussed. Cigarette excise taxation predates the Surgeon General's report, and three tobacco states were among the first to tax cigarettes—based mainly on the revenue potential. However, since 1964, states that do not produce tobacco have increased their excise taxes twice as frequently as have the tobacco states. It is noted that the nonproducing southern states are national leaders in aggressive cigarette excise taxation, along with the northeastern states. While differences between the producing and nonproducing southern states are explainable, regional differences exist outside the tobacco belt, although the four major census regions differ little in tax legislation overall. Most states have passed smoking-restriction laws, and while a majority of southern states are included, their laws are less restrictive. As with excise taxation, the states that are major tobacco producers differ from other southern states in smoking-restriction legislation, but the differences are not as dramatic, and the average restrictiveness of laws in effect is actually higher in the tobacco-producing states than it is in their southern neighbors. The irony in this differential deference toward tobacco-producing states is that the limited antismoking legislation appears to represent a neighborly form of support; yet there is evidence that excise taxation—a proven revenue raiser—is also a much more effective deterrent to smoking than are the laws passed to restrict smoking in public places.

GENERAL

82–1854. Canada, Department of Health and Welfare. **Tar, Nicotine and Carbon Monoxide Yields of 10 Brands of Small Cigars.** Canada, Department of Health and Welfare, News Release 1981-125, November 10, 1981, 2 pp.

Smokers inhaling the smoke of small cigars are exposing themselves to serious risks: the tar, nicotine, and carbon monoxide yields of these cigars are much higher than those of most Canadian cigarettes. Ten brands of small cigars were tested under the same conditions as cigarettes, and the data listed. It is recommended that the Canadian tobacco manufacturers be required to print a health warning and the yields of toxic substances on small cigar packages. Smokers of the cigars are advised to stop inhaling, and preferably to quit smoking.

82–1855. Crowne, J. E.; Shapiro, R. D. **The Nonsmoker as a Preferred Risk.** *National Underwriter* 84(Life & Health Ins. Ed.): 13, 16-17, August 23, 1980.

The State Mutual study conclusively proves that significant differences exist between the mortality of insured smokers and insured nonsmokers. Each company must therefore individually answer a number of questions before determining whether it will use and, if so, how it will adjust the State Mutual experience to arrive at its own smoker/nonsmoker or other preferred risk pricing standards. Critical questions concern the reasons for and the structure of the nonsmoking package, comparison of company structure with that of State Mutual, effect of less honesty on applications, discount request policy for existing insureds who have quit and their effect on mortality rates, supplementary coverage, and handling of reformed smokers and those who use low-tar/nicotine products and their effect on mortality. After clarifying their own plans and procedures, insurance companies must then make adjustments for any material differences between their own and the State Mutual experience. (Auth. Abs. Mod.)

82–1856. Schievelbein, H. **Medizinische und Juristische Aspekte der Praevention des Rauchens. Gezielte Praeventivmassnahmen und ihre Effekte. [Medical and Legal Aspects of Smoking Prevention. Suitable Preventive Measures and Their Effects.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische, Praeventive und Therapeutische Aspekte.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 107-125, German.

Since health risks from nicotine intake are not yet strictly defined, changing the ratio of tar to nicotine from 1:20 to 1:10 mg is recommended, to reduce tar exposure in smokers. British, German, and U.S. epidemiological studies indicate that lower yield cigarettes (having less than 17 mg tar, 1.2 mg nicotine) are less hazardous than high-yield cigarettes (more than 25.8 mg tar, 2.0 mg nicotine). However, at least one British study shows that the smoker inhales nearly as much tar and nicotine from a low-yield as a high-yield cigarette, due to changes in inhalation patterns. Thus raising the tar/nicotine ratio to 1:10, while maintaining nicotine levels in the 0.5 to 1.0 mg range, is recommended. This will, hopefully, reduce smoking-related morbidity and mortality rates by reducing tar exposure.

82–1857. Schmidt, F. 1. **Praevention des Rauchens aus Nationaler und Internationaler Sicht. Das Rauchen als Medizinisches und Politisches Problem. [National and International Aspects of Smoking Prevention. Smoking as a Medical and Political Problem.]** In: Deutsche Hauptstelle Gegen Suchtfahren. *Rauchen Oder Gesundheit. Politische Praeventive und Therapeutische Aspekie.* Hamburg, Neuland-Verlagsgesellschaft mbH, 1982, pp. 7-12. German.

In consideration of the medical aspects of cigarette smoking, advertising of tobacco products in West Germany is deplored and a 10 percent reduction of subsidy for tobacco planters in the European Community is urged. Legal protection of nonsmokers is considered to be particularly urgent because passive smoking is believed to be a genuine health hazard. Also, reforming German law for youth protection is needed to limit accessibility of tobacco products to schoolchildren. Efforts at reduction of cigarette consumption should be shifted to schools, and abolishing school smoking rooms should be one of the first measures. Prohibiting refining of tobacco for cigarettes is suggested: the present artificial refining of tobacco for cigarettes is designed to make the smoker nicotine-dependent. To facilitate the switch to unrefined tobacco, cigarette producers should be authorized to use a mixture of refined and unrefined tobaccos during a transition period. Political leaders are urged to stop smoking.

AUTHOR AND ORGANIZATIONAL INDEX

Abdel-Khalik, A., 82-1735
Abe, H., 82-1571
Abe, T., 82-1594
Acheson, E. D., 82-1636
Adams, E. E., 82-1758
Adena, M. A., 82-1727
Advertising Age., 82-1836
Aeschbacher, H. U., 82-1553
Afford, S. C., 82-1589
Ahmad, M., 82-1837
Akaki, T., 82-1544
Akers, R. L., 82-1770
Albano, W. A., 82-1610, 82-1627
Alexander, J. K., 82-1797
Alster, P., 82-1554
Alvesalo, I., 82-1728
Amankwah, K. S., 82-1716
American Cancer Society., 82-1784
Andersen, R. A., 82-1536
Anderson, J. R., 82-1744
Anderson, M. W., 82-1568
Andreasson, L., 82-1599
Andresen, B. D., 82-1717
Anttonen, T., 82-1729
Aoki, K., 82-1633
Arch, P. J., 82-1680
Armstrong, B., 82-1600
Arnaud, M. J., 82-1595
Arrendale, R. F., 82-1537
Artvinli, M., 82-1649
Arunagiri, R., 82-1591
Asselain, B., 82-1604
Atkins, N., 82-1673
Auerbach, O., 82-1601
Aument, P. E., 82-1825
Autrup, H., 82-1555

Baertl, J. M., 82-1722
Bailit, H. L., 82-1728
Baker, E., 82-1725
Baky, A. A., 82-1647
Balfour, D.J.K., 82-1752
Ballieux, R. E., 82-1642
Bardell, D., 82-1556
Baris, Y. I., 82-1649
Barthel, E., 82-1602
Bauchinger, M., 82-1730
Bauer, L. A., 82-1650
Bauer, S. P., 82-1650
Bauermann, E., 82-1807
Bawol, R. D., 82-1617
Beatty, K., 82-1557
Beck, W., 82-1690
Begin, R., 82-1651
Beilin, L. J., 82-1733
Bellamy, D., 82-1662
Benfield, J. R., 82-1609
Bennett, R., 82-1694
Benwell, M.E.M., 82-1752
Berenson, G. S., 82-1713
Bergia, R., 82-1669
Berkson, D. M., 82-1683, 82-1684
Beveridge, T., 82-1566
Bhooshan, B., 82-1540
Bianchine, J. R., 82-1717
Biener, K., 82-1753
Birch, S., 82-1673
Bircher, J., 82-1595
Bjorlin, G., 82-1599
Black, D., 82-1754
Black, L., 82-1627
Blot, W. J., 82-1603
Blouin, R. A., 82-1650
Boden, R. M., 82-1808, 82-1809
Boeijinga, J. K., 82-1570
Bollag, W., 82-1743
Bollati, A., 82-1669
Bolt, A.J.N., 82-1823
Bond, G., 82-1619
Bone, R., 82-1774
Borgman, R. F., 82-1755
Boswell, D. R., 82-1652
Bottiger, L. E., 82-1596
Boucher, R. C., 82-1666
Boucot, K. R., 82-1645
Boundy, M. G., 82-1676
Bourke, G. J., 82-1616
Boyette, D. M., 82-1534
Bradshaw, E., 82-1632
Breimer, D. D., 82-1570
Brennan, S. O., 82-1652
Brenner, M. H., 82-1677
Brill, R. W., 82-1678
Brille, D., 82-1665
Brine, D. R., 82-1560
†British-American Tobacco Co., Ltd., 82-1828
Brody, J. A., 82-1697
Bromberg, P. A., 82-1666
†Brown and Williamson Tobacco Corp., 82-1824, 82-1832, 82-1833, 82-1835
Brown, L. M., 82-1603
Brugere, J., 82-1604
Brugh, M., 82-1555
Bruzgielewicz, J., 82-1664
Buchholz, E. H., 82-1785
Buiatti, E., 82-1603
Buntin, W. H., 82-1666
Burch, P.R.J., 82-1605, 82-1679
Bureau, M. A., 82-1651
Burenkov, S. P., 82-1786
Burnett, D., 82-1589
Burney, R. E., 82-1558
Burrows, G. D., 82-1581
Burton, H. R., 82-1536
Burton, R. C., 82-1614

Calabro, F., 82-1635
Campbell, C. R., 82-1534
Campbell, J. S., 82-1534
Canada, Department of Health and Welfare., 82-1538, 82-1854
Canner, P. L., 82-1702
Cantin, A., 82-1651
Cantrell, D. V., 82-1824
Carlhoff, H.-W., 82-1756
Carlson, L. A., 82-1596
Carpenter, D., 82-1680
Carr, D. B., 82-1693
Carrell, R. W., 82-1652
Carruth, B. R., 82-1718
Cartwright, R. A., 82-1613
Chadda, V. S., 82-1712
Chait, L. D., 82-1757
Chamberlain, K. G., 82-1581
Chamson, A., 82-1559
Chang, F.-F., 82-1640
Chant, A.D.B., 82-1680
Chaplin, J. F., 82-1534
Chappuis, C., 82-1553
Chard, B. C., 82-1823
Chenoweth, D. E., 82-1654
Chijimatsu, Y., 82-1569
Chopra, N. M., 82-1539
Chortyk, O. T., 82-1537
Chowienczyk, P. J., 82-1672

---

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

Christen, A. G., 82-1758
Chujoh, C., 82-1731
Chung, S. I., 82-1564
Cissik, J. H., 82-1660
Clark, B. R., 82-1567
Clark, T.J.H., 82-1672
Clarke, B. F., 82-1735
Clarke, W. R., 82-1770
Clonfero, E., 82-1653
Clutterbuck, D., 82-1787
Clyne, C.A.C., 82-1680
Coe, R. M., 82-1788
Cohen, A. B., 82-1654, 82-1655
Cohen, B. S., 82-1628
Cohen, J. D., 82-1788
Cohen, J. I., 82-1628
Cohen-Solal, J.-M., 82-1789
Cohen, W. L., 82-1606
Cole, P. J., 82-1657
Colman, R. W., 82-1681
Colombo, A., 82-1669
Colvin, R. B., 82-1614
Colwell, L., 82-1800
Cook, C. E., 82-1560
Cooper, M. A., 82-1607
Cooperstock, R., 82-1759
Cordano, A., 82-1722
Corey, P., 82-1802
Correa, P., 82-1637
Cortese, M. S., 82-1653
Cox, M., 82-1802
Crawford, C. B., 82-1534
Creswell, W. H., Jr., 82-1760
Criqui, M. H., 82-1682
Crouse, E., 82-1788
Crowne, J. E., 82-1855
Cuckle, H. S., 82-1608
Curran, J. G., 82-1551
Curtis, M. B., 82-1585

Dahlen, G., 82-1709
†Daicel Ltd., 82-1834
Dalaker, K., 82-1719
Dalbey, W. E., 82-1738
Dale, B., 82-1732
Daly, L., 82-1616
Daniel, F. B., 82-1590
Daube, M., 82-1790
Davies, B. D., 82-1561
Davies, J. E., 82-1603
Davis, B., 82-1673
Davis, L., 82-1722
Davis, S., 82-1639
De Caro, L., 82-1609
de la Burde, R. Z., 82-1825
De Marini, D. M., 82-1562
de Santis, A., 82-1732
Di Franza, J. R., 82-1628
Di Guiseppi, S. R., 82-1560
Dodge, R., 82-1656
Dowd, D. E., 82-1662
Dresp, J., 82-1730
Dreyer, N. A., 82-1644
Drouet, D., 82-1665
du Bois, R. M., 82-1657
Duncan, L.J.P., 82-1735
Duszynski, K. R., 82-1643
Dyer, A. R., 82-1683, 82-1684

Ebert, R. V., 82-1776
Elmfeldt, D., 82-1715
Elton, R. A., 82-1735
Emley, G. S., 82-1687
Epler, J. L., 82-1567
Ercan, Z. S., 82-1710
Ersoy, A., 82-1710
Estrada, R., 82-1647
Eysenck, H. J., 82-1761

Faber, J., 82-1642
Fagerstrom, K.-O., 82-1762, 82-1804
Fain, P. R., 82-1610, 82-1627
Fatt-Hi, A. S., 82-1740
Faulks, L., 82-1661
Feinleib, M., 82-1697
Fencl, M., 82-1549
Fergusson, D. M., 82-1658
Feyerabend, C., 82-1780
†Firmenich, SA, 82-1821
Fisher, E. B., Jr., 82-1788
Fitscha, P., 82-1566
Flak, E., 82-1664
Flicker, C., 82-1563
Forman, S., 82-1702
Fortson, N. G., 82-1670
Fowler, G., 82-1766
Fraumeni, J. F., Jr., 82-1603
Frecker, R. C., 82-1768
Freeborn, V., 82-1773
Freis, E. D., 82-1685
French, M. A., 82-1581
Frenken, C.W.G.M., 82-1686
Frey, J., 82-1559
Friedlander, E., 82-1644
Friedman, G. D., 82-1617
Friello, D. R., 82-1826
Frost, J. K., 82-1737
Fukuchi, S., 82-1579
Fukuda, M., 82-1571
Fung, W. P., 82-1733
Galanakis, D. K., 82-1564
Gallagher, H. G., 82-1727
Gardner, J. W., 82-1611, 82-1612
Garg, S. K., 82-1565
Garlinska, J., 82-1664
Garner, R. C., 82-1613
Gascoyne, J. M., 82-1811
Gatzy, J. T., 82-1666
Gersh, B. J., 82-1690
Geyer, M. A., 82-1563
Gibbs, L., 82-1673
Gillick, L., 82-1614
Ginns, L. C., 82-1614
Gishen, P., 82-1659
Glasgow, R., 82-1773
Glasunov, I. S., 82-1786
Glover, E. D., 82-1758
Godwin-Austen, R. B., 82-1734
Goldblatt, P. J., 82-1590
Goldenheim, P. D., 82-1614
Golding, J., 82-1763
Goldstein, G., 82-1614
Gordon, T., 82-1767
Grafstrom, R. C., 82-1555.
Granda, E. J., 82-1810
Gray, N., 82-1791
Gray, R. S., 82-1735
Greenberg, S. D., 82-1647
Gregory, J., 82-1736
Griffiths, R. R., 82-1757
Guerin, M. R., 82-1567
Gunasegaram, R., 82-1720
Gunnar, R. M., 82-1696
Gupta, P. K., 82-1737
Guz, A., 82-1779

Haenszel, W., 82-1637
Hagstrom, B. E., 82-1732
Hall, J. B., 82-1816
Hall, S. K., 82-1660
Hall, W. J., 82-1671
Hallgrimsson, J., 82-1607
Hamilton, J. L., 82-1536
Hamilton, P.J.S., 82-1800
Hansky, J., 82-1742
Harington, J. S., 82-1632
Harley, N. H., 82-1628
Harris, C. C., 82-1555
Hartiala, J., 82-1574
Hartshorne, J. M., 82-1597
Hashimoto, S., 82-1579
Haslam, P. L., 82-1657
Hatakeyama, M., 82-1544
Hauf, R., 82-1730
Haugen, A., 82-1555
Haussmann, G., 82-1764

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

Havel, R. J., 82–1699
Hay, J., 82–1728
Hayashi, S., 82–1592
Heckman, C. A., 82–1738
Heilbrun, L. K., 82–1615
Heiss, G., 82–1682
Hems, R., 82–1811, 82–1812
Henry, C. J., 82–1540
Herity, B., 82–1616
Herman, B., 82–1686
Hiatt, R. A., 82–1617
Higashi, E., 82–1580
Higenbottam, T., 82–1618
Higgins, I., 82–1619
Hill, C. R., 82–1628
Hilt, B., 82–1620
Hilton, D. I., 82–1693
Hirashima, T., 82–1622
Hirata, K., 82–1622
Hirota, T., 82–1622
Hirota, Y., 82–1695
Hirsch, J.-M., 82–1739
Hitzenberger, G., 82–1566
Hivert, M., 82–1559
Ho, C.-H., 82–1567
Ho-Yen, D. O., 82–1765
Hodgkinson, P., 82–1792
Hoffmann, D., 82–1628
Holdbrook, M. J., 82–1682
Holmes, P., 82–1661
Homan, K., 82–1701
Homma, H., 82–1569
Horwood, L. J., 82–1658
Hoss, W., 82–1561
Hrabak-Zerjavic, V., 82–1626
Hu, M. X., 82–1782
Huesmert, N., 82–1748
Hugh-Jones, P., 82–1662
Hughes, J. A., 82–1662
Hugli, T. E., 82–1654
Humphries, B., 82–1838
Hunter, N. R., 82–1647
Hurst, G. A., 82–1670
Hurwitz, C., 82–1614
Hurwitz, P., 82–1619
Husum, B., 82–1621
Hutchinson, R. R., 82–1687
Hutchison, D.C.S., 82–1659, 82–1662, 82–1675
Hwang, K. K., 82–1540
Hwang, M. H., 82–1696

Iams, J. D., 82–1717
Ibrahim, A. S., 82–1740
Iizuka, T., 82–1622
†Imperial Group Ltd., 82–1823
†International Flavors and Fragrances Inc., 82–1808, 82–1809, 82–1810, 82–1816, 82–1817, 82–1818, 82–1819, 82–1820
Ioannou, Y. M., 82–1568
Ishihara, Y., 82–1569
Ismail, S., 82–1668
Israel, R. H., 82–1671
Itabashi, M., 82–1622
Itani, S., 82–1580
Iyer, R., 82–1780

Jacobs, J. E., 82–1663
Jaffe, R. A., 82–1693
Jain, N. C., 82–1712
James, H. L., 82–1655
Jamrozik, K., 82–1766
Janoff, A., 82–1564
Jarrett, R. J., 82–1688
Jedrychowski, W., 82–1664
Jeppsson, J.-O., 82–1652
Jochemsen, R., 82–1570
Johnson, D. E., 82–1541
Johnson, R. R., 82–1833
Jones, A. S., 82–1704
Jones, D. R., 82–1678
Jordan, J. W., 82–1629

Kaelber, C. T., 82–1767
Kallianos, A. G., 82–1542
Kallner, A., 82–1709
Kamada, T., 82–1571
Kane, J. P., 82–1699
Kannel, W. B., 82–1689
Karim, S.M.M., 82–1720
Karki, N. T., 82–1723
Kasperbauer, M. J., 82–1536
Kato, H., 82–1622
Kauffmann, F., 82–1665
Kaufman, D. G., 82–1576
Kawano, S., 82–1571
Kazemi, H., 82–1614
Keen, H., 82–1688
Kefalides, A., 82–1705
Kehoe, G., 82–1826
Kennelly, B. M., 82–1690
Keritsis, G. D., 82–1827
Kilburn, K. H., 82–1623
Killen, J., 82–1797
Kinlen, L. J., 82–1608
Kitamura, S., 82–1569
Kiwala, J., 82–1810, 82–1817, 82–1818, 82–1819, 82–1820
Kjuus, H., 82–1624
Klein, L., 82–1640
Klemp, P., 82–1741
Knowles, M. R., 82–1666
Kohn, H. I., 82–1644
Kolendorf, K., 82–1741
Konno, T., 82–1625
Korman, M. G., 82–1742
Kornhuber, H. H., 82–1793
Korsgaard, R., 82–1599
Korte, A., 82–1573
Kosaka, K., 82–1569
Kouri, R. E., 82–1540
Kousaka, Y., 82–1543
Kowalczyk, H., 82–1547
Kozarevic, D., 82–1767
Kozlowski, L. T., 82–1768
Kritzer, M. F., 82–1584
Kulcar, Z., 82–1626
Kumar, M., 82–1668
Kung, P. C., 82–1614
Kuntzleman, C. T., 82–1794
Kunze, M., 82–1795
Kurtz, A. B., 82–1735

Laboda, K., 82–1547
Lam, P.P.S., 82–1588
Lambert, C. A., 82–1769
Lamm, G., 82–1691
Lane, G. K., 82–1690
Langard, S., 82–1620, 82–1624
Lasnitzki, I., 82–1743
Lauer, R. M., 82–1770
Laurell, C.-B., 82–1652
Laurent, P., 82–1564
Lavet, C., 82–1604
Layde, P. M., 82–1692
Lechner, J. F., 82–1555
Leck, I., 82–1633
Lee, P. N., 82–1734
Lefcoe, N. M., 82–1777
Lellouch, J., 82–1665
Lepper, M. H., 82–1683, 82–1684
Leyten, A.C.M., 82–1686
†Life Savers Inc., 82–1826
Lin, J.-P., 82–1561
Lindberg, H. A., 82–1683, 82–1684
Lindsay-Smith, V., 82–1613
Link, L. A., 82–1535
Lionetti, F., 82–1561
Lipsius, S. L., 82–1572
Lislerud, A., 82–1624
Loeb, H. S., 82–1696
Loganath, A., 82–1720
Loscutoff, S. M., 82–1693
Loughlin, J., 82–1644
Lowe, N. J., 82–1749
Lowitz, D. A., 82–1827

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

Luke, J. A., 82-1828
Lynch, H. T., 82-1610, 82-1627
Lynch, J., 82-1627
Lyngdal, P. T., 82-1624
Lyon, J. L., 82-1611, 82-1612

Maccoby, N., 82-1797
Mackay, D.A.M., 82-1826
Madle, S., 82-1573
Madsbad, S., 82-1741
Maeda, K., 82-1544
Mahoney, D. P., 82-1733
Mangan, G. L., 82-1763, 82-1771
Mannisto, J., 82-1574, 82-1575
Marmot, M. G., 82-1734
Marritt, C. R., 82-1829
Martell, E. A., 82-1628
Martin, D. H., 82-1593
Maruotti, R. A., 82-1635
Mashimo, N., 82-1625
Mason, M. K., 82-1629
Mass, M. J., 82-1576
Masse, S., 82-1651
Massey, J., 82-1770
Mastrangelo, G., 82-1653
Masuo, Y., 82-1544, 82-1545
Matarazzo, J. D., 82-1772
Math, M. V., 82-1577
Matthay, R., 82-1630
Mattiasson, I., 82-1599
Matulionis, D. H., 82-1578
Mau, G., 82-1721
Mausner, J. S., 82-1631
Maxwell, J. C., Jr., 82-1839
McCartney, P., 82-1688
McCaul, K. D., 82-1773
McCusker, K., 82-1774, 82-1776
McGee, D., 82-1767
McGlashan, N. D., 82-1632
McLarty, J. W., 82-1670
McLean Ross, A. H., 82-1744
McManus, I. C., 82-1775
McMichael, A. J., 82-1597
McMillan, G.H.G., 82-1667
McNabb, M. E., 82-1776
McRae, D. D., 82-1546
Meadows, W. R., 82-1696
Mebane, I., 82-1682
Mellits, E. D., 82-1722
Melville, G. N., 82-1668
Menard, H. A., 82-1651
Merli, F., 82-1549
Merrett, A. C., 82-1742
Metter, G. E., 82-1640
Miller, L. G., 82-1614
Miller, W. L., 82-1830
Mills, J. L., 82-1668
Mishra, S. N., 82-1712
Mittleman, J., 82-1585
Mizuno, K., 82-1579
Mlynarczyk, W., 82-1547
†Molins Ltd., 82-1829
Mollo, F., 82-1669
Moody, J. P., 82-1765
Mookherjee, B. D., 82-1810
Mooney, A., 82-1677
Moriarty, M., 82-1616
Morimoto, M., 82-1580
Morris, J. N., 82-1754
Morrison, A. S., 82-1633
Mould, A. J., 82-1613
Moyle, J., 82-1805
Mueller, A., 82-1796
Muramatsu, M., 82-1548
Muranaka, H., 82-1580
Mussinan, C. J., 82-1810
Muto, R., 82-1831

Nader, P. R., 82-1797
Nakamura, I., 82-1579
Nakazawa, S., 82-1731
Nash, D. R., 82-1670
NcNabb, E., 82-1774
Nemiroff, M. J., 82-1558
Nettesheim, P., 82-1593
Neuber, D., 82-1840
Nevius, E., 82-1694
Newhouse, M., 82-1634
Ng, A., 82-1603
Ng, K. J., 82-1717
Ngan, L.L.E., 82-1724
Niebuhr, E., 82-1621
Nienstedt, W., 82-1574
Noack, K.-A., 82-1798
Nomura, A., 82-1615
Nordwall, C. W., 82-1603
Norman, T. R., 82-1581
Novotny, M., 82-1549
Nuesch, E., 82-1566

Obata, K., 82-1633
Obe, G., 82-1573
O'Connor, K. P., 82-1806
Oh, M., 82-1619
Ohno, Y., 82-1633
Okada, M., 82-1695
Okuyama, K., 82-1543
Omae, T., 82-1695
Omland, H., 82-1624
O'Neill, H. K., 82-1773
Op de Coul, A.A.W., 82-1686
Ory, H. W., 82-1692
Owen, M. C., 82-1652

Pacha, W., 82-1566
Paige, D. M., 82-1722
Palac, R. T., 82-1696
Palesch, Y. Y., 82-1697
Panchanathan, T. B., 82-1591
Patrick, C. H., 82-1697
Paul, O., 82-1683, 82-1684
Pederson, L. L., 82-1777
Peeler, H. J., 82-1755
Peh, K. L., 82-1720
Pelkonen, O., 82-1723
Perez-Reyes, M., 82-1560
Perry, C., 82-1797
Persijn, J. P., 82-1642
Peters, J. M., 82-1676
Peters, M. A., 82-1724
Petersen, C. C., 82-1698
Peto, R., 82-1582
Pezzuoli, G., 82-1635
Phelps, D. W., 82-1693
†Philip Morris Inc., 82-1814, 82-1822, 82-1825, 82-1827
Phillips, N. R., 82-1699
Pifarre, R., 82-1696
Poe, R. H., 82-1671
Polissar, L., 82-1639
Pope, M. A., 82-1768
Portheine, F., 82-1745
Practitioner., 82-1778
Preisig, R., 82-1595
Presant, C. A., 82-1640
Prevost, R. J., 82-1541
Priola, D. V., 82-1585
Prior, I. M., 82-1726
Puglia, W. J., 82-1826
Puska, P., 82-1701

Quin, G. D., 82-1560

Racic, Z., 82-1767
Rainbow, S., 82-1735
Ramstroem, L. M., 82-1799
Rang, E. H., 82-1636
Rao, A.S.V., 82-1673
Rao, T. K., 82-1567
Ratnam, S. S., 82-1720
Ravenholt, R. T., 82-1628
Ravi Kiran, T. N., 82-1565
Rawbone, R. G., 82-1779
Rees, P. J., 82-1672
Reisine, S., 82-1728
Richards, B., 82-1613

---

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

Rickert, W. S., 82-1768
Rios-Dalenz, J., 82-1637
Rizio, D., 82-1840
Robertie, P., 82-1557
Robinson, J. C., 82-1768
Rodgers, N. T., 82-1576
Rodriguez-Rigau, L. J., 82-1746
Rogers, T. D., 82-1647
Rose, G., 82-1618, 82-1800
Rosenberg, J., 82-1620
Rosenfield, A., 82-1598
Rotenberg, K. S., 82-1583
Rothman, K. J., 82-1644
Roviaro, G., 82-1635
Rowsell, D. G., 82-1811, 82-1812
Ruma, T. A., 82-1610, 82-1627
Rump, B. S., 82-1773
Russell, M.A.H., 82-1780
Ryan, K. C., 82-1662

Sackner, M. A., 82-1673, 82-1781
Sadowski, C., 82-1547
Saeed, T., 82-1549
Saito, T., 82-1622
Salonen, J. T., 82-1700, 82-1701
Samfield, M., 82-1550
Sandwisch, D. W., 82-1590
Sanford, R. A., 82-1832, 82-1833
Saperstein, M. D., 82-1749
Sartori, F., 82-1635
Sato, N., 82-1571
Saunders, A.J.S., 82-1659
Sawada, H., 82-1834
Schenck, K. M., 82-1590
Schenk, J., 82-1801
Schievelbein, H., 82-1856
Schlant, R. C., 82-1702
Schmid, E., 82-1730
Schmidt, F., 82-1857
Schmidt, G. T., 82-1742
Schmitt, F. L., 82-1810, 82-1817, 82-1818, 82-1819, 82-1820
Schoenberger, J. A., 82-1683, 82-1684
Schoenenberger, J. C., 82-1703
Schulte, B.P.M., 82-1686
Schut, H.A.J., 82-1590
Schwab, L. S., 82-1584
Schweizerische Rundschau fuer Medizin (PRAXIS)., 82-1813
Scott-Malden, J. N., 82-1841
Seeman, J. I., 82-1814
Seidman, H., 82-1645
Sekiguchi, T., 82-1625
Selikoff, I. J., 82-1638
Senior, R. M., 82-1557
Seshadri, R., 82-1725
Severson, R. F., 82-1537
Severson, R. K., 82-1639
Shaffer, J. W., 82-1643
Shannon, F. T., 82-1658
Shapiro, R. D., 82-1855
Shekelle, R. B., 82-1683, 82-1684
Shekelle, S., 82-1683, 82-1684
Shelton, A., 82-1842
Shephard, R. J., 82-1747, 82-1802
Shibata, M., 82-1544, 82-1545
Shichiri, M., 82-1571
Shiga, M., 82-1834
Shinohara, K., 82-1834
Shipley, M. J., 82-1618, 82-1800
Shively, J. E., 82-1640
Shonka, M., 82-1627
Silber, W., 82-1641
Silbert, J. E., 82-1674
Simon, E., 82-1815
Simons, L. A., 82-1704
Simpson, W. W., 82-1585
Singhvi, D. R., 82-1712
Sinzinger, H., 82-1705
Skuland, J., 82-1719
Sloan, C. H., 82-1551
Small, W. P., 82-1744
Smith, C., 82-1754
Smith, K. D., 82-1746
Smith, M. A., 82-1744
Smith, T., 82-1706
Smith, T. J., 82-1676
Snook, M. E., 82-1537
Sobel, S., 82-1694
Socolar, M. J., 82-1850
Solomon, D., 82-1797
Sonnenberg, A., 82-1748
Sparrow, D., 82-1674
Spayth, V., 82-1640
Spence, V. A., 82-1765
Spjut, H. J., 82-1647
Sprecker, M. A., 82-1816, 82-1817, 82-1818, 82-1819, 82-1820
Spring, D. J., 82-1812
Staberg, B., 82-1741
Stamler, J., 82-1683, 82-1684, 82-1702
Stamler, R., 82-1683, 82-1684
Stanhope, J. M., 82-1726
Starkey, I. R., 82-1735
Stave, O., 82-1624
Steinberger, E., 82-1746
Stemmermann, G. N., 82-1615
Stepney, R., 82-1586
Stern, G. M., 82-1734
Stich, H. F., 82-1587, 82-1588
Stich, W., 82-1587, 82-1588
Stockley, R. A., 82-1589
Stocksmeier, U., 82-1707
Stoiljkovic, C., 82-1626
Stoner, G. D., 82-1590
Storm, H.-G., 82-1804
Stravynski, A., 82-1806
Strieder, M., 82-1801
Strnad, M., 82-1626
Sturtevant, F. M., 82-1708
Suga, Y., 82-1544
Sugiyama, Y., 82-1569
Sulemani, A. A., 82-1712
Sulochana, G., 82-1591
Sundfor, K., 82-1719
Sutherland, G. R., 82-1725
Sutton, S. R., 82-1780
Swain, J. W., 82-1822

Tabak Journal International., 82-1843, 82-1844, 82-1845
Takeda, K., 82-1544
Takeshita, M., 82-1695
Tala, E., 82-1851
Tamada, T., 82-1580
Tanaka, H., 82-1544
Taylor, B., 82-1658
te Velde, E. R., 82-1642
Telch, M., 82-1797
Tenovuo, J., 82-1729
Terahara, A., 82-1544
Terrill, T. R., 82-1535
Thanh, P., 82-1604
Theorell, T., 82-1709
Thilander, H., 82-1739
Thom, B., 82-1759
Thomas, A. F., 82-1821
Thomas, C. B., 82-1643
Thomas, H. E., Jr., 82-1689
Thomas, R. E., 82-1541
Tillery, J. B., 82-1541
Tobacco International., 82-1846, 82-1847, 82-1848, 82-1849
Tobin, M. J., 82-1659, 82-1675, 82-1781
Toda, N., 82-1592
Todd, C. W., 82-1640
Toki, T., 82-1579
Tomita, H., 82-1545
Topping, D. C., 82-1593
Townsend, P., 82-1754
Townsend, P. J., 82-1657
Travis, J., 82-1557
Trell, E., 82-1599
Trump, B. F., 82-1555
Tudor, T. T., 82-1835

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

AUTHOR AND ORGANIZATIONAL INDEX

Tuimala, R., 82–1723
Tuomilehto, J., 82–1701
Turker, R. K., 82–1710
Turner-Warwick, M., 82–1657
Tuyns, A. J., 82–1782
Tweed, W. A., 82–1679

U.K. Department of Health and Social Security., 82–1711
U.S. House of Representatives, 97th Congress, 2nd Session., 82–1852
Ueda, K., 82–1695
Uotila, P., 82–1574, 82–1575
Urbaniak, S., 82–1735
Ury, H. K., 82–1617
Utell, M. J., 82–1671

Vajpayee, A., 82–1712
Van Der Graaff, M., 82–1570
van Luijk, J. H., 82–1686
Varoli, F., 82–1635
Vaughan, L., 82–1652
Vedin, A., 82–1715
Verhoek, W. G., 82–1633
Verma, M. M., 82–1539
Vesey, C. J., 82–1780
Vigborg, A., 82–1709
Vinals, J. F., 82–1810, 82–1817, 82–1818, 82–1819, 82–1820
Vock, M. H., 82–1810, 82–1817, 82–1818, 82–1819, 82–1820
Voegelin, M., 82–1595
Voors, A. W., 82–1713

Wagner, W. L., 82–1628
Wake, F. R., 82–1783
Wake, R., 82–1803
Walinder, O., 82–1709
Walker, A. M., 82–1644
Walker, W. F., 82–1765
Wallace, R. B., 82–1682
Wang, C.-S., 82–1543
Warner, K. E., 82–1853
Watanabe, H., 82–1622
Watanabe, M., 82–1594
Watanabe, R., 82–1544
Webber, L. S., 82–1713
Weberg, A. D., 82–1716
Webster, J.H.H., 82–1680
Weeks, S. J., 82–1775
Wegman, D. H., 82–1676
Wehner, A. P., 82–1693
Weiss, S. T., 82–1674
Weiss, W., 82–1645
Welch, K., 82–1619
Wennmalm, A., 82–1554
Wheeler, L. A., 82–1749
WHO Advisory Committee on Medical Research., 82–1646
Wiesler, D., 82–1549
Wietholtz, H., 82–1595
Wilhelmsen, L., 82–1714, 82–1715
Wilhelmsson, C., 82–1715
†Wilkinson Sword Ltd., 82–1811, 82–1812
Williams, R. E., 82–1829
Wilson, A.G.E., 82–1568
Winkler, D. G., 82–1647
Winters, T. H., 82–1628
Witzel, F., 82–1826
Woeber, K., 82–1807
World Health Organization., 82–1750
Wu, D.L.S., 82–1822
Wu, S.-C., 82–1558
Wulf, H. C., 82–1621
Wynder, E. L., 82–1628, 82–1648

Yaginuma, K., 82–1579
Yamamoto, T., 82–1552
Yamazaki, M., 82–1544
Yasumatsu, N., 82–1544, 82–1545
Yesner, R., 82–1751
Yoder, E. E., 82–1536

Zannini, P., 82–1635
Zengil, H., 82–1710
Zukel, W. J., 82–1767
Zuniga, T. H., 82–1539

---

*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

SUBJECT INDEX

ABORTION
induced in primigravidae, patients' smoking habits, 82–1719
miscarriage incidence in Maori women, smoking relationship, 82–1726

ABSENTEEISM
company no-smoking ban, effect, 82–1787
welders at naval dockyards, smoking relationship, 82–1667

ACCIDENTS
carbon monoxide poisoning, symptoms and response to treatment of smokers vs. nonsmokers, 82–1558
fire hazard reduction by cigarette paper coatings, 82–1815

ACETYLCHOLINE
cardiac nerves responses in dogs, 82–1585
cholinergic antagonists, central response to nicotine, 82–1584
nicotinic receptor-mediated potassium movements in guinea pig heart, 82–1572

ADENOCARCINOMA
operability, survival rate, smoking relationship compared to other lung cancer types, 82–1635

ADOLESCENTS
antismoking campaigns in French schools, 82–1789
international smoking rates among teenagers, overview, 82–1778
personal and family health behaviors, perceptions, 82–1797
pregnancy outcomes, smoking and diet effects, 82–1718, 82–1722
resting heart rate among biracial youth in Louisiana, smoking relationship, 82–1713
secondary schoolchildren in London, smoking patterns, 82–1779
smoking behavior in Iowa, influencing factors, 82–1770
smoking behavior in Switzerland, motives among 16- to 19-year-old apprentices, 82–1753
smoking behavior prediction factors, 82–1773
smoking prevention programs, 82–1788, 82–1803

ADVERTISING
adolescent smoking, effect, 82–1803
attitudes toward cigarette vs. newspaper advertising profession in West Germany, 82–1801
ban as part of international tobacco control, recommendation, 82–1790
counteractions and nonsmoking support in the media, national conference proceedings, 82–1784
Finland, 1976 smoking control legislation, 82–1851
generic cigarette products, 82–1838
legislative restrictions in Sweden, 82–1799
tobacco industry expenditures in the United States for 1980, 82–1840
top advertisers in the United States, expenditures for 1980, 82–1836

AGE FACTORS
β-receptor blocker pharmacokinetics, influence with smoking, 82–1566
adjusted pulse pressure levels, influence with cigarette smoking, 82–1683
blood pressure, effects on, 82–1682
cardiovascular disease in oral contraceptive users, risk with smoking, 82–1692
hearing loss correlation, duration of smoking comparison, 82–1740
induced abortion in primigravidae, patients' smoking habits, 82–1719
lung cancer patients, younger vs. older, 82–1609
lung neoplasm risks in men over 45 years, 82–1645
mortality risk for oral contraceptive users under age 30, smoking effect, 82–1598
myocardial infarction in young men, risk factors, 82–1690
smoking habit in Calvados, France, 82–1782

AGING
menopause in smokers vs. nonsmokers in Australia, 82–1727

AGRICULTURAL PRACTICES
Argentina, tobacco crop improvement efforts, 82–1849
maleic hydrazide, transfer rate from tobacco to smoke, 82–1539
nicotine level in tobacco leaf and yield/ha, relationship, 82–1534

AIR POLLUTION
cancer from occupational exposure, policies for risk management, 82–1638
indoor air quality, smoking factor, 82–1736, 82–1750
parental smoking and gas cooking, effect on respiratory health of children in Arizona, 82–1656
passive smoking health risks, in-depth review, 82–1747

welding pollutants vs. smoking, respiratory symptom factors, 82–1667

ALCOHOL CONSUMPTION
- Australian male mortality, with dietary, occupational, and smoking factors, 82–1597
- blood pressure, effects on, 82–1682
- esophageal and gastric cancer relationship with smoking, 82–1622
- esophageal cancer, etiological factor with smoking, 82–1632, 82–1641
- head and neck cancers, factor with smoking, 82–1604, 82–1616
- laryngeal cancer incidence in Iceland, relationship with smoking, 82–1607
- miscarriage and stillbirth correlation to smoking and drinking, 82–1726
- problem drinkers and alcoholics, smoking, diet, and coffee drinking correlations, 82–1755
- pulmonary tuberculosis patients in Australia, smoking as predisposing factor, 82–1661
- serum apolipoprotein A-I levels, relationship with cholesterol levels and demographic factors, 82–1699
- sister-chromatid exchange in mothers and infants, relationship with maternal smoking, 82–1725
- Yugoslavian men, smoking patterns relationship, 82–1767

ALCOHOLS
- cis-3-hexenol, derivatives used as tobacco additives for flavor and aroma, 82–1810
- 2, 4, 6-trimethylcyclohexanemethanol and derivatives, additives, 82–1820

ALKALOID CONTENT
- Burley tobacco, aging effects on concentration, 82–1536

ALKALOIDS, TOBACCO
- isolation method and mutagenicity assessment, 82–1567
- pharmacokinetics of nicotine and its metabolites in humans and animals, 82–1583

AMINO ACIDS
- Burley tobacco, aging effects on concentration, 82–1536

AMMONIA
- Burley tobacco, aging effects on concentration, 82–1536
- quarternary ammonium compound, tobacco additive, 82–1814

ANTIBODIES
- erythrocyte antibody complement rosettes in smokers with sarcoidosis vs. cryptogenic fibrosing alveolitis, 82–1657

ANTIGENS
- carcinoembryonic antigen in asbestos workers, smokers vs. nonsmokers, 82–1670
- carcinoembryonic antigen levels in blood of smokers, 82–1745
- carcinoembryonic antigen serum levels in invasive cervical cancer patients, smoking and age as confounding factors, 82–1642
- diabetic retinopathy, HLA antigens and smoking as risk factors, 82–1735
- serum levels in cancer patients vs. controls, smoking effects, 82–1640

ANTISMOKING CAMPAIGNS
- blueprint for action, national antismoking conference proceedings, 82–1784
- cardiovascular risk factor intervention, the North Karelia project, 82–1700
- companies, employee education and financial incentives for cessation, 82–1787
- Finland, 1976 legislation, 82–1851
- France, effectiveness, 82–1789
- Great Britain, doctors' and health visitors' attitudes, 82–1766
- Great Britain, public awareness effect on tobacco industry, 82–1844
- legislative and economic effects in tobacco states vs. other regions, 82–1853
- Pakistan tobacco industry, effect on, 82–1837
- Soviet Union, preventive medicine program, 82–1786
- Sweden, program effectiveness, 82–1799
- West German health insurance industry media program, 82–1785
- West German schools, recommendations, 82–1764
- West Germany, recommendations, 82–1857
- World Health Organization recommendations for tobacco control, 82–1646

ANTITRYPSIN DEFICIENCY
- emphysema, phenotype vs. smoking in etiology, 82–1659
- emphysema relationship, smoking effects, 82–1675

ANTITRYPSIN LEVELS
- α_1-AT abnormalities and smoking in emphysema etiology, 82–1652
- cigarette smoke oxidant content effect, 82–1655
- oxidation effect on trypsin/elastase inhibitory capacity ratio, smoking relationship, 82–1557

ARECA
- betel quid, clastogenic activities of five components, 82–1588

ARECA CHEWING
- oral cancer relationship, 82–1646
- saliva clastogenicity in cell culture, 82–1587

SUBJECT INDEX

ARECOLINE
- clastogenic activities with other betel quid components, 82–1588

ARGENTINA
- tobacco production efforts, 82–1849

ARIZONA
- indoor pollution, parental smoking and gas cooking effect on children, 82–1656

AROMATIC HYDROCARBON CONTENT
- five Polish cigarette brands compared, 82–1547

AROMATIC HYDROCARBONS
- mutagenicity in rat liver and in *Neurospora crassa*, 82–1562

ARSENIC
- lung cancer risk among workers, with smoking, 82–1619

ARTERIAL DISEASES
- myocardial infarction risk factor in young men, with smoking, 82–1690

ARTERIOSCLEROSIS
- collagen production as etiological factor, smoking relationship, 82–1559
- passive smoking effects on blood platelets in etiology, 82–1705
- platelets role in etiology, relationship with smoking and other risk factors, 82–1681

ARYL HYDROCARBON HYDROXYLASE
- human lung microsomes, smoking effect, 82–1594
- inducibility in oral cancer patients, relationship with smoking and oral health, 82–1599
- placental activity vs. lymphocyte inducibility, maternal smoking effect, 82–1723

ASBESTOS
- cancer etiology, synergism with smoking, 82–1634
- cancer mortality among exposed workers, smokers vs. nonsmokers, 82–1620
- control of carcinogenic substances, policy considerations, 82–1638
- lung cancer, etiological factor with smoking, 82–1623
- lung cancer incidence in Duval County, Florida, possible synergistic effect with smoking, 82–1603
- lung function impairment with more vs. less than 20 years exposure, smoking effect, 82–1660
- occupational exposure, respiratory abnormalities in smokers vs. nonsmokers, 82–1670
- respiratory cell changes related to asbestos and smoke, 82–1737

ASTHMA
- Polish chemical workers, occupational vs. smoking factors, 82–1664

ATTITUDES
- adolescents, personal and family health practices, 82–1797
- adolescents, smoking motives of apprentices in Switzerland, 82–1753
- adolescents, smoking prevention program strategy, 82–1788
- adolescents, smoking prevention through coping skills, 82–1803
- antismoking education, opinions of doctors and health visitors in Great Britain, 82–1766
- Canadian schoolchildren in grades 4-6, factors predictive of smoking, 82–1777
- German army recruits, relation to smoking habit, 82–1761
- myths about tobacco consumption, inaccuracies, 82–1758
- passive smoking, opinions of smokers and nonsmokers, 82–1747
- peripheral vascular disease subjects advised to quit smoking, 82–1680
- personality factors in reasons for smoking, 82–1775
- seventh grade students, adolescent smoking predictor factor, 82–1773
- situational vs. generalized smoking intervention method, attitudinal factor, 82–1806
- social approval of marihuana dealing vs. cigarette promotion in West Germany, 82–1801

AUSTRALIA
- cancer incidence and primary/secondary control of risk factors, 82–1600
- male mortality rate by occupational, dietary, alcohol, and smoking factors, 82–1597
- menopausal age in smokers vs. nonsmokers, 82–1727
- pulmonary tuberculosis patients, smoking as predisposing factor, 82–1661

AUSTRIA
- smoking control policy recommendations, 82–1795

BACTERIA
- Ames *Salmonella*/Microsome test for mutagenicity of urine studies, 82–1749
- cigarette smoke condensate mutagenicity in *Salmonella typhimurium*, 82–1562, 82–1567
- cigarette smoke toxicity in vitro, 82–1556

BEHAVIOR, ANIMAL
- maternal nicotine administration in rats, effects on offspring, 82–1724
- nicotine effect on rat behavior, 82–1563
- nicotine effects on blood pressure and aggression in rats and squirrel monkeys, 82–1687

BEHAVIOR, HUMAN
- adolescent smoking, factors in initiation, 82–1770
- adolescent smoking, prevention programs, 82–1803
- behavioral perspective on antismoking campaigns, 82–1783
- initiation and incidence of smoking among Massachusetts students, 82–1769
- myths about tobacco consumption, inaccuracies, 82–1758
- personality factors in reasons for smoking, 82–1775
- placebo chewing gum as nicotine substitute in smoking cessation, effectiveness, 82–1804
- psychologists' role in smoking prevention, 82–1772
- quitting by smoking without inhaling, comment, 82–1805
- reasons for maintaining or quitting smoking, doctor's advice, 82–1759
- situational vs. generalized intervention in smoking cessation, 82–1806

BENZO(*A*)PYRENE
- cocarcinogenicity with benzo(*e*)pyrene, target tissue effects, 82–1593
- DNA adduct formation in lung, liver, and forestomach of mice, 82–1568
- DNA-binding levels in human bladder and bronchus cell cultures, 82–1590
- metabolism by human endometrium in culture, 82–1576
- metabolism in bronchus, esophagus, colon, and duodenum of same individual, 82–1555

BENZO(*E*)PYRENE
- anticarcinogenic retinoid activity in rodent respiratory tissue, 82–1743
- cocarcinogenicity with benzo(*a*)pyrene, target tissue effects, 82–1593

BENZOPYRENES
- polycyclic aromatic hydrocarbons in 5 Polish cigarette brands, 82–1547

BIOASSAY
- ^{14}C caffeine and breath test for demethylation studies, smoking effect, 82–1595
- *Neurospora crassa* vs. *Salmonella typhimurium* in smoke condensate mutagenicity study, 82–1562
- bacterial assessment of cigarette smoke toxicity, 82–1556
- blood samples vs. self-reporting in determining cigarette consumption, 82–1582
- Chinese hamster ovary cells for clastogenicity assessment, 82–1587
- collagen biosynthesis by fibroblast cell culture, tobacco smoke extracts effect, 82–1559
- mutagenicity of cigarette smoke condensate, assessment with *Salmonella typhimurium*, 82–1567

BIRTH WEIGHT
- maternal smoking effect, 82–1718, 82–1721
- nicotine administration effect in rats, 82–1724
- nutritional supplements and smoking effect, 82–1722

BLADDER
- benzo(*a*)pyrene—DNA binding in human cell culture, 82–1590

BLADDER NEOPLASMS
- artificial sweetener use and smoking effects, 82–1633
- mutagenicity of urine of smoking and nonsmoking patients, 82–1613
- nonnutritive sweetener use relationship, smoking effects, 82–1644

BLOOD
- monocytes, activation in sarcoidosis vs. cryptogenic fibrosing alveolitis patients, 82–1657
- oxidation effect on alpha-1-proteinase inhibitors in smokers, 82–1557

BLOOD CHEMICAL ANALYSIS
- plasma nicotine levels from nicotine chewing gum vs. smoking, 82–1776

BLOOD CHOLESTEROL
- acute smoking effects, 82–1591
- breast cancer relationship, with lipids, dietary fat, and smoking, 82–1617
- cardiovascular risk factor intervention, the North Karelia project, 82–1700
- coronary disease risk factor, synergistic relationship with smoking and blood pressure, 82–1714
- coronary heart disease mortality, decline factor, 82–1706
- enzymatic breakdown by C-20, 22-desmolase, cigarette smoke effect, 82–1720
- levels in relatives of ischemic heart disease patients, smoking effect, 82–1712
- levels in smokers vs. nonsmokers, 82–1580
- levels in young adults and subsequent cancer, smoking relationship, 82–1643
- myocardial infarct survival, reduction as factor in long-term prognosis, 82–1702
- progressive aortocoronary vein graft narrowing, risk factor, 82–1696
- reduction, cessation of smoking effects, 82–1703
- serum apolipoprotein A-I levels and demographic factors, relationship, 82–1699
- WHO cardiovascular disease intervention program in Europe, review, 82–1691

BLOOD CIRCULATION
- gastric mucosal blood flow, nicotine effect, 82–1748
- gastric mucosal blood volume, smoking effects, 82–1571

SUBJECT INDEX

BLOOD COAGULATION
arachidonate induced, nicotine effect, 82–1554
fibrin aggregation, inhibition by smoke condensate, 82–1564

BLOOD LIPIDS
acute smoking effects, 82–1591
cerebral stroke in Finland, relationship with smoking, 82–1701
levels in relatives of ischemic heart disease patients, smoking effect, 82–1712
WHO cardiovascular disease intervention program in Europe, review, 82–1691

BLOOD PLATELETS
antiaggregatory prostaglandins sensitivity, passive smoking effect, 82–1705
coronary disease relationship, effect of smoking and other risk factors, 82–1681
monoamine oxidase activity in smokers and nonsmokers, 82–1581
thromboxane synthesis, nicotine effect, 82–1554

BLOOD PRESSURE
age, education, weight, exercise, alcohol, oral contraceptive use, and smoking effects, 82–1682
bradykinin effects on rabbits and guinea pig hearts, nicotine influence, 82–1710
cerebral stroke in Finland, relationship with smoking, 82–1701
coronary disease risk factor, synergistic relationship with smoking and serum cholesterol, 82–1714
daily cigarette consumption correlation, 82–1767
nicotine ingestion effect in rats and squirrel monkeys, 82–1687
reduction, cessation of smoking effects, 82–1703
smoke-exposed vs. sham-exposed rats, effect, 82–1693
systolic vs. diastolic, smoking association, 82–1683, 82–1684
WHO cardiovascular disease intervention program in Europe, review, 82–1691

BLOOD SUGAR
daily cigarette consumption correlation, 82–1767

BLOOD VESSELS
coronary arterial response to nicotine in dogs, effect of various drugs, 82–1592
nicotine effect on bradykinin-caused vasodilation in rabbits and guinea pig hearts, 82–1710
progressive aortocoronary vein graft narrowing, risk factors, 82–1696

BODY WEIGHT
blood pressure, effects on, 82–1682
emphysema patients, smokers vs. ex-smokers, 82–1662
maternal weight gain in pregnant adolescents, smoking effect, 82–1718
reduction, cessation of smoking effects, 82–1703

BOLIVIA
cancer incidence in La Paz, smoking relationship, 82–1637

BRAIN
nicotine administration and withdrawal, effects on stress in rats, 82–1752
nicotine, behavioral responses in rats, 82–1563

BRONCHI
airways narrowing after smoke inhalation, smokers vs. nonsmokers, 82–1672
benzo(*a*)pyrene—DNA binding in human cell culture, 82–1590
benzo(*a*)pyrene metabolism compared with colon, esophagus, and duodenum, 82–1555
transepithelial electric potential difference measurement, cigarette smoke effect, 82–1666

BRONCHIAL DISEASES
mucus gland enlargement, pathology, 82–1669
Polish chemical workers, occupational vs. smoking factors, 82–1664
sarcoidosis vs. cryptogenic fibrosing alveolitis patients, macrophage function differences, 82–1657

BRONCHIAL MUCOSA
mucus gland enlargement in healthy subjects, smokers vs. nonsmokers, 82–1669
swelling response to smoke inhalation in rats, 82–1668

BRONCHIAL NEOPLASMS
atypical bronchial epithelial cells, objective measurement, 82–1647
English furniture workers' incidence, smoking relationship, 82–1636
polonium-210 in etiology, 82–1628

BRONCHITIS
Italian aluminum workers, age, occupation, and smoking factors, 82–1653
Polish chemical workers, occupational vs. smoking factors, 82–1664

CADMIUM
total body burdens for "standard man" determination, 82–1541

CAFFEINE
breath test for demethylation studies, CO_2 exhalation rates in smokers vs. nonsmokers, 82–1595

CALIFORNIA
serum apolipoprotein A-I levels, lipoprotein lipid levels and demographic relationships, 82–1699

CANADA
carbon monoxide and tar levels in 47 cigarette brands, 82–1538
Health Hazard Appraisal fitness program, smoking reduction effects, 82–1802
schoolchildren in grades 4-6, factors predictive of smoking, 82–1777
tar, nicotine, and carbon monoxide yields of 10 small cigar brands, 82–1854

CANNABIS
marihuana smoke components, comparison with tobacco smoke, 82–1549
West Germany, attitudes toward cigarette promotion vs. marihuana dealing, 82–1801

CARBOHYDRATE CONTENT
protein extraction from tobacco, relationship, 82–1550

CARBON DIOXIDE
exhalation rates in smokers vs. nonsmokers, ^{14}C caffeine and breath test determination, 82–1595
generation during and between puffs, 82–1545
minicigarette weight loss per puff and carbon oxides delivery, relationship, 82–1552
tobacco expansion, use in, 82–1825

CARBON MONOXIDE
acute poisoning, symptoms and response to treatment of smokers vs. nonsmokers, 82–1558
blocking of filter vents by smoker, effect on yield, 82–1768
coronary disease, causative role and preventive measures, 82–1711
expired-air concentration measurement, value in determining smoke inhalation depth, 82–1586
minicigarette weight loss per puff and carbon oxides delivery, relationship, 82–1552
passive smoking health risks, in-depth review, 82–1747
plasma levels, smoking reduction effect, 82–1765

CARBON MONOXIDE CONTENT
filter for reduction by mixing with fresh air, 82–1831
levels in 47 varieties of cigarettes in Canada, 82–1538
smoke levels controlled by smoking device, 82–1823
yields of 10 Canadian small cigar brands, 82–1854

CARBOXYHEMOGLOBIN LEVELS
elimination time following acute carbon monoxide poisoning, smokers vs. nonsmokers, 82–1558
low-yield cigarettes, smoking patterns, relationship, 82–1780
nicotine level association during smoking, 82–1580
peripheral vascular disease patients, smoking effects, 82–1680
single measurements vs. self-reporting in determining cigarette consumption, 82–1582
smoking reduction effect, 82–1765

CARCINOGENESIS
atypical bronchial epithelial cells, objective measurement, 82–1647
esophageal cancer, smoking, alcohol consumption, and mineral deficiency factors, 82–1641
oral cancer, smoking, aryl hydrocarbon hydroxylase inducibility, and oral health factors, 82–1599

CARCINOGENICITY REDUCTION
anticarcinogenic retinoid activity in rodent respiratory tissue, 82–1743

CARCINOGENS
benzo(*a*)pyrene—DNA binding levels in human bladder and bronchus cell cultures, 82–1590
benzo(*a*)pyrene, metabolism by human endometrium in culture, 82–1576
cancer etiology and substance management considerations, 82–1631
control and management of carcinogenic substances, review, 82–1638
maleic hydrazide, transfer rate from tobacco to smoke, 82–1539
pathogenesis of lung cancer, smoking relationship, 82–1601
polonium-210, alpha radiation activity in lungs, 82–1628

CARCINOMA, BRONCHOGENIC
atypical bronchial epithelial cells, objective measurement, 82–1647
histological types comparison in lung cancer patients, smoking relationship, 82–1635
survival rates among patients, smoking factor, 82–1629

CARCINOMA, EPIDERMOID
etiological and epidemiological factors, 82–1604
histological types comparison in lung cancer patients, smoking relationship, 82–1635

CARDIOVASCULAR DISEASES
angiotensin-converting enzyme activity, acute smoking effects in etiology, 82–1579
Australian men, occupational, dietary, alcohol, and smoking factors, 82–1597
intervention program of U.S. Air Force, smoking as risk factor, 82–1698
mortality in Great Britain, economic change relationship, 82–1677
mortality in Great Britain, inhalation patterns and tar levels relationship, 82–1618
mortality in the Soviet Union, 82–1786
oral contraceptives with smoking as risk factors, 82–1692
risk factor intervention, the North Karelia project, 82–1700
WHO program in Europe, review, 82–1691

Yugoslavia study, alcohol consumption and smoking habits of men, 82–1767

CARDIOVASCULAR SYSTEM
- Innovar effects on heart function and response to smoke in rabbits, 82–1678
- passive smoking effects, review, 82–1747, 82–1751

CATECHOLAMINES
- cardiac nerves responses to nicotine in dogs, 82–1585
- epinephrine release by smoking, effect on platelets in coronary disease etiology, 82–1681

CELLS
- benzo(*a*)pyrene metabolism in bronchus vs. colon vs. esophagus vs. duodenum, 82–1555
- pulmonary, smoking-induced pathogenic changes to lung cancer, 82–1601

CELLS, EPITHELIAL
- activation of macrophages, sarcoidosis vs. cryptogenic fibrosing alveolitis patients, smoking effect, 82–1657
- atypical bronchial epithelial cells, objective measurement, 82–1647
- cytologic changes from asbestos vs. smoke exposure, 82–1737
- snuff-induced hyperplasia in oral mucosa of rats, 82–1739

CEREBROVASCULAR DISORDERS
- Australian men, occupational, dietary, alcohol, and smoking factors, 82–1597
- cerebral stroke in Finland, smoking relationship, 82–1701
- intervention recommendations, 82–1707
- stroke in the Netherlands, smoking as risk factor, 82–1686
- thrombotic and hemorrhagic strokes, oral contraceptive use and smoking as risk factors, 82–1694, 82–1708

CESSATION OF SMOKING
- anticancer strategy for adults, 82–1648
- avoiding heart attacks, relationship, 82–1711
- behavioral reasons for quitting, doctor's advice, 82–1759
- blood pressure, body weight, and blood serum cholesterol effects, 82–1703
- cardiorespiratory disease in men, effect on, 82–1800
- cardiovascular risk reduction, U.S. Air Force intervention program, 82–1698
- comprehensive program, national conference proceedings, 82–1784
- coronary disease risk screening, effect on, 82–1704
- cost-effective strategies in the United Kingdom, 82–1792
- emphysema patients, effect on lung function, 82–1662
- employees, company financial incentive and educational programs, 82–1787
- health insurance industry campaign in West Germany, 82–1785
- lipoprotein concentration and lipid composition, effect on, 82–1577
- myocardial infarct survival, factor in prognosis, 82–1702, 82–1715
- myocardial infarction intervention and mild hypertension treatment, effect on, 82–1685
- myths and their inaccuracies, 82–1758
- nicotine dependence factor in maintenance, 82–1762
- oral cancer risk effect, 82–1606
- peripheral vascular disease patients, surgeon's advice effect, 82–1680
- placebo chewing gum as nicotine substitute, effectiveness, 82–1804
- primary and secondary prevention of lung and other cancer incidence, 82–1600
- psychologists' role, 82–1772
- situational vs. generalized approach, 82–1806
- smoking control recommendations in Austria, 82–1795
- variation in smoking habits, therapeutic recommendations, 82–1807

CESSATION OF SMOKING, METHODS
- antismoking advice, 10-year trial, 82–1800
- nicotine chewing gum, maintenance factor, 82–1762
- nicotine chewing gum, plasma nicotine levels, 82–1776
- placebo chewing gum as nicotine substitute, effectiveness, 82–1804
- smoking without inhaling, comment, 82–1805

CHEWING TOBACCO
- consumption trends in the United States, 82–1842
- saliva clastogenicity in cell culture, 82–1587

CHILDREN
- Canadian schoolchildren in grades 4-6, factors predictive of smoking, 82–1777
- Feelin' Good coronary disease intervention program for children, 82–1794
- France, antismoking campaigns in schools, 82–1789
- Great Britain, secondary schoolchildren in London, smoking patterns, 82–1779
- respiratory diseases, parental smoking relationship, 82–1658
- respiratory health, parental smoking and gas cooking effect, 82–1656
- resting heart rate among biracial youth in Louisiana, smoking relationship, 82–1713
- West Germany, antismoking education recommendations, 82–1793
- West Germany, smoking habits of 9- and 10-year-olds, 82–1756

CHROMOSOMES
clastogenicity of betel quid components, 82–1588
clastogenicity of saliva from areca and tobacco chewers, 82–1587
sister-chromatid exchange in lymphocytes relationship to breast cancer, smoking association, 82–1621
sister-chromatid exchange in mothers and infants, maternal smoking relationship, 82–1725
sister-chromatid exchanges in smokers exposed to electric and magnetic fields, 82–1730
sister-chromatid exchanges in vitro and in vivo, cigarette smoke condensates effect, 82–1573

CHRONIC BRONCHOPULMONARY DISEASE
cryptogenic fibrosing alveolitis, smoking effect on macrophage function, 82–1657

CIGARETTE PAPER
perforation method, permeability effects on puffing ease and component delivery, 82–1544
self-extinguishing coating, 82–1815
tipping material with ventilatory and nonsticking properties, 82–1835

CIGARETTE SMOKE
arachidonic acid metabolism effect in rat lungs, 82–1574
bacterial toxicity in vitro, 82–1556
chemical composition, review, 82–1542
cholesterol C-20, 22-desmolase activity in human placenta, effects, 82–1720
controlling content, smoking device, 82–1823
filter collection efficiency, spectrophotometric measurement, 82–1551
grooved filter rod for dilution, 82–1833
humidity change response, 82–1543
inhalation depth, self-assessment vs. objective measurement, 82–1586
intake regulation vs. cigarette length, 82–1757
oxidant content, alpha$_1$-antitrypsin activity effect, 82–1655
α_1-proteinase inhibitor, electrophoretic mobility effects, 82–1589
refractive index of particulates, 82–1546

CIGARETTES
burning characteristics during and between puffs, 82–1545
carbon monoxide and tar levels in 47 Canadian brands, 82–1538
filter-tipped cigarette manufacturing process, 82–1829
Pakistan production trends, 82–1837
production and sales statistics for Great Britain, 1961-1980, 82–1845
smoldering process, analysis of temperature, density, and oxygen profiles, 82–1548
ventilation, filtration, perforation and blend type effects on puffing and component delivery, 82–1544
vigilance performance effects of low vs. medium nicotine levels, 82–1771
weight loss per puff and carbon oxides delivery, relationship, 82–1552

CIGARETTES, HIGH-TAR
puffing and inhalation patterns compared with low-tar cigarettes, 82–1781

CIGARETTES, LOW-TAR
initiation of smoking by schoolchildren, relationship, 82–1779
1980 marketing competition in the United States, 82–1836
puffing and inhalation patterns compared with high-tar cigarettes, 82–1781

CIGARETTES, LOW-YIELD
additives to improve flavor, monitoring recommended, 82–1648
generic products marketing success, 82–1838
grooved filter rod for smoke dilution, 82–1833
half-length cigarettes, smokers behavioral response, 82–1757
misuse related to smoking behaviors, 82–1768
nicotine vs. tar and carbon monoxide levels, 82–1762
polonium-210 content, 82–1628
protein extraction relationship, 82–1550
puff and inhalation patterns, 82–1780
tar/nicotine ratio recommendation, 82–1856
unrefined tobacco to lower nicotine content, recommendations, 82–1857

CIGARETTES, NONTOBACCO
oxidant content of smoke, 82–1655
parsley cigarettes with added phencyclidine, 82–1560

CIGARS
Maxwell market report on brands in the United States, 82–1839
production and sales statistics for Great Britain, 1961-1980, 82–1845
tar, nicotine, and carbon monoxide yields of 10 Canadian small cigar brands, 82–1854

COCARCINOGENS
alcohol consumption and smoking as esophageal cancer risk factors, 82–1632
benzo(*e*)pyrene with benzo(*a*)pyrene in mouse tracheal tissues, 82–1593
cancer etiology and management considerations, 82–1631
herpes simplex virus and tobacco in oral cancer, 82–1606
occupational factors and smoking in lung cancer, 82–1623

COFFEE CONSUMPTION
pancreatic cancer, coffee vs. smoking risk, 82–1639
pancreatic cancer mortality correlation, smoking-adjusted, 82–1608
problem drinkers and alcoholics, correlation with smoking and diet, 82–1755
urine mutagenicity, comparison in smokers vs. nonsmokers, 82–1553

COGNITION
vigilance performance, effect of low vs. medium nicotine levels, 82–1771

COLOMBIA
1981 tobacco production and consumption, 82–1848

COLON
benzo(*a*)pyrene metabolism compared with bronchus, esophagus, and duodenum, 82–1555

COLONIC NEOPLASMS
serum antigen levels in patients vs. controls, smoking effects, 82–1640

COMBUSTION TEMPERATURE
smoldering process analysis, 82–1548

CONTRACEPTIVES, ORAL
blood pressure, effects on, 82–1682
cardiovascular disease risk factor with smoking, 82–1692, 82–1694, 82–1708
health risks in smokers vs. nonsmokers, 82–1598
nitrazepam clearance, effect with sex, menstrual cycle, and smoking, 82–1570
serum apolipoprotein A-I levels, relationship with cholesterol levels and demographic factors, 82–1699

COPPER
tap water levels relationship to lung function, smoking factor, 82–1674

CORONARY DISEASE
causal relationship of smoking, diet, and hypertension, pros and cons, 82–1679
Coronary Drug Project, long-term prognosis following myocardial infarct recovery, 82–1702
Feelin' Good intervention program for children, 82–1794
Great Britain, mortality decline since mid-1970s, 82–1706
heart attacks, review of preventive measures, 82–1711
ischemic heart disease, mortality rate reduction and smoking relationship, 82–1697
ischemic heart disease, oral contraceptive use with smoking as risk factors, 82–1694, 82–1708
ischemic heart disease patients, serum lipids and smoking habit in relatives, 82–1712
ischemic heart disease, serum uric acid level relationship, smoking association, 82–1695
ischemic heart diseases, risk factors among Finnish immigrants vs. native Swedes, 82–1709
mortality among diabetics, smoking effects, 82–1688
mortality in men, smoking intervention effect, 82–1800
platelets role in etiology, effect of smoking and other risk factors, 82–1681
risk factor screening program, effectiveness, 82–1704
sudden death, smoking as a risk factor, 82–1689
synergistic relationship of smoking, blood pressure, and serum cholesterol, 82–1714
WHO cardiovascular disease intervention program in Europe, review, 82–1691

COST BENEFITS ANALYSIS
cardiovascular disease intervention program, U.S. Air Force, 82–1698
Finland, dental expenditures relationship to fluoride, oral health and smoking, 82–1728
Great Britain, smoking control and mortality reduction strategies, 82–1792
health costs and workdays lost by smoking employees, 82–1787
lung cancer, early detection and intervention for high-risk individuals, 82–1623
United States, tobacco program cost to government, 82–1850

COTININE
amniotic fluid levels from passive smoking, 82–1717
low-yield cigarettes, smoking patterns and plasma level relationship, 82–1780
plasma level variation in smokers and nonsmokers, consumption effect, 82–1580

COUGH
diagnostic methods compared, 82–1671

DEMOGRAPHIC FACTORS
serum apolipoprotein A-I levels, relationship with lipoprotein lipid levels, 82–1699
urban vs. rural, smoking habits of West German 9- and 10-year-olds, 82–1756
urban vs. rural, smoking incidence in Calvados, France, 82–1782

DENTAL DISEASES
gingivitis, crevicular iodide and thiocyanate relationship with smoking, 82–1729
tooth loss among adults in Finland, smoking relationship, 82–1728

DIABETES
coronary heart disease mortality among borderline diabetics, 82–1688
insulin requirements, smoking effects, 82–1741

progressive aortocoronary vein graft narrowing, risk factor, 82–1696
retinopathy development, HLA antigens and smoking as risk factors, 82–1735

DIET
adolescent pregnancy outcome, effect with smoking, 82–1718, 82–1722
alcohol consumption, correlation with coffee drinking and smoking, 82–1755
artificial sweeteners and smoking, bladder cancer relationship, 82–1633
Australian male mortality, with alcohol, occupational, and smoking factors, 82–1597
breast cancer, dietary fat, serum cholesterol and lipids, and smoking relationship, 82–1617
causal relationship with coronary disease, pros and cons, 82–1679
coronary heart disease in Great Britain, mortality decline factor with smoking, 82–1706
nonnutritive sweetener use and bladder cancer relationship, smoking effects, 82–1644
WHO cardiovascular disease intervention program in Europe, review, 82–1691

DNA
benzo(*a*)pyrene binding levels in human bladder and bronchus cell cultures, 82–1590
benzo(*a*)pyrene:DNA adduct formation in lung, liver, and forestomach of mice, 82–1568

DRUG ABUSE
phencyclidine added to parsley cigarettes, 82–1560

DRUG METABOLISM
β-receptor blocker pharmacokinetics, smoking and age influence, 82–1566
behavioral responses to nicotine, atropine, carbachol, and tubocurarine in the rat, 82–1563
bradykinin effects on rabbits and guinea pig hearts, nicotine influence, 82–1710
coronary arterial response to nicotine in dogs, effect of various drugs, 82–1592
Innovar effects on cardiovascular function and response to smoke in rabbits, 82–1678
insulin absorption in diabetics, smoking effects, 82–1741
nitrazepam clearance, sex, oral contraceptive use, menstrual cycle, and smoking effects, 82–1570
phenylbutazone clearance rate in smokers vs. nonsmokers, 82–1565
ranitidine-treated duodenal ulcer patients, smoking effect on healing, 82–1742
salbutamol, ipratropium bromide, and sodium cromoglycate, effect on airways smoke response, 82–1672
theophylline clearance in obstructive pulmonary disease patients, kidney and heart function effects, 82–1650
thromboxane synthesis, aspirin and nicotine effects, 82–1554

DUST
bronchial cancer in English furniture workers, smoking relationship, 82–1636
exposure effects in Parisian factory workers, smoking relationship, 82–1665
lung cancer incidence in Duval County, Florida, possible synergistic effect with smoking, 82–1603
respiratory symptoms among Polish chemical workers, factor with smoking, 82–1664
talc exposure effect on miners and millers, smoking relationship, 82–1676

EARS
auditory vigilance performance, effect of low vs. medium nicotine, 82–1771
hearing loss and smoking relationship, 82–1740

ECONOMICS
Argentina, Virginia and dark-leaf tobacco production for 1981, 82–1849
Colombia, tobacco production and consumption for 1981, 82–1848
Great Britain, cigarette consumption per capita and economic change relationship, 82–1677
Great Britain, declining tobacco consumption, taxation and public opinion factors, 82–1844
Great Britain, price war, taxation, and diminished consumption effects on tobacco industry, 82–1841
Great Britain, tobacco production and sales statistics for 1961-1980, 82–1845
insurance product packages for nonsmokers, the State Mutual model, 82–1855
Japan, import policies effect on U.S. tobacco industry, 82–1843
Pakistan, tobacco industry trends, 1981 and 1982, 82–1837
South Africa, rise in leaf production planned, 82–1846
state excise taxation and antismoking legislation, effect in tobacco states vs. other regions, 82–1853
tobacco support system program modifications—No Net Cost Tobacco Program Act of 1982, 82–1852
United States, generic cigarette product success, 82–1838
United States, Government tobacco program effects on farmers and market competitiveness, 82–1850
United States, marketing data for 1980, 100 top advertisers, 82–1836
United States, Maxwell market report on cigar brands, 82–1839
United States, smokeless tobacco sales, 82–1842

United States, 1980 tobacco industry advertising expenditures, 82–1840
Zimbabwe, flue-cured and Burley tobacco production, 82–1847

ELECTROENCEPHALOGRAPHY
arousal levels, cigarette smoking effects, 82–1763

EMPHYSEMA
alpha-1-proteinase oxidation, etiological relationship in smokers, 82–1557
antitrypsin abnormalities and smoking in etiology, 82–1652
α_1-antitrypsin and smoking in etiology, 82–1675
antitrypsin phenotype vs. smoking, risk factors, 82–1659
copper in tap water vs. smoking in etiology, 82–1674
elastase releasing factor in pulmonary alveolar macrophages of smokers, 82–1654
lung function, deterioration in smokers vs. ex-smokers, 82–1662
α_1-proteinase inhibitor, electrophoretic mobility effects of cigarette smoke, 82–1589
smoke inhalation effect on rat lungs, 82–1738

ENZYME ACTIVITY
alpha$_1$-antitrypsin, cigarette smoke effect on inhibitory capacity, 82–1655
angiotensin-converting enzyme activity, acute smoking effects, 82–1579
α_1-antitrypsin and smoking in emphysema etiology, 82–1675
aryl hydrocarbon hydroxylase from human lung microsomes, smoking effect, 82–1594
aryl hydrocarbon hydroxylase in lymphocytes vs. placenta, maternal smoking effect, 82–1723
cholesterol C-20, 22-desmolase from human placenta, cigarette smoke effects, 82–1720
human platelet cyclo-oxygenase, nicotine effect, 82–1554
plasma transglutaminase, in vitro smoke effects, 82–1564
platelet monoamine oxidase in smokers and nonsmokers, 82–1581
α_1-proteinase inhibitor, in vitro smoke effects, 82–1589
serum alpha-1-proteinase oxidation, trypsin/elastase inhibitory capacity ratio effect in smokers, 82–1557
serum angiotensin I converting enzyme, smoking effect, 82–1569

ENZYMES
β-D-glucosaminidase, 82–1657
alpha-1-proteinase, elastase, 82–1557
alpha$_1$-antitrypsin, 82–1655
angiotensin-converting enzyme, 82–1579
α_1-antitrypsin, 82–1675
aryl hydrocarbon hydroxylase, 82–1594
cholesterol C-20, 22-desmolase, 82–1720
cyclo-oxygenase, 82–1554
elastase, myeloperoxidase, and lysozyme, 82–1654
monoamine oxidase, 82–1581
plasma transaminase, 82–1564
serum angiotensin I converting enzyme, 82–1569

ESOPHAGEAL NEOPLASMS
epidemiology and etiology, 82–1641
gastric cancer association, incidence in Japan, 82–1622
incidence in Transkei, South Africa, smoking and alcohol consumption as risk factors, 82–1632

ESOPHAGUS
benzo(*a*)pyrene metabolism compared with colon, duodenum, and bronchus, 82–1555

ETHNIC GROUPS
biracial youth in Louisiana, resting heart rate and smoking relationship, 82–1713
black pregnant adolescents, diet and smoking vs. offspring birth weight, 82–1722
Finnish immigrants vs. native Swedes, ischemic heart disease risk factors, 82–1709
Japanese men in Hawaii, cancer incidence and smoking relationship, 82–1615
Maori women, smoking and drinking effects on pregnancy outcome, 82–1726
Mormon men, church involvement and cancer incidence relationship, 82–1611
Mormon women, church involvement and cancer incidence relationship, 82–1612

EUROPE
WHO cardiovascular disease program, review, 82–1691

EX-SMOKERS
population survey in Calvados, France, 82–1782

EYE DISEASES
diabetic retinopathy, HLA antigens and smoking as risk factors, 82–1735

FATTY ACIDS
arachidonic acid metabolism, cigarette smoke ventilation effect in rat lungs, 82–1574
blood concentration, acute smoking effects, 82–1591
blood platelet sensitivity to antiaggregatory prostaglandins, passive smoking effect, 82–1705
6-keto prostaglandin $F_{1\alpha}$ secretion, smoking effects, 82–1733

FEDERAL REPUBLIC OF GERMANY
antismoking measures, recommendations, 82–1857
health insurance industry antismoking campaign, 82–1785
school smoking intervention recommendations, 82–1764, 82–1796, 82–1798

smoking control recommendations and alternatives to cigarettes, 82–1793
smoking habits of 9- and 10-year-old children in Baden-Wurttemberg, 82–1756
social attitudes toward marihuana dealing vs. cigarette promotion vs. newspaper advertising, 82–1801

FERTILITY
nicotine effects in the sea urchin, 82–1732
semen quality of smokers vs. nonsmokers, 82–1746

FERTILIZATION, TOBACCO
nicotine level in tobacco leaf and yield/ha, relationship, 82–1534
nitrogen and potassium interaction, effect on Burley tobacco leaf components, 82–1535

FETAL GROWTH
nicotine administration effect in rats, 82–1724

FETAL MORTALITY
miscarriage and stillbirth correlation to smoking and drinking, 82–1726

FETUS
cotinine in amniotic fluid from maternal passive smoking, 82–1717
maternal smoking effects, review, 82–1721
passive smoking health risks, in-depth review, 82–1747
preputial neural tissue, maternal smoking effects, 82–1716

FILTERS
bacterial toxicity of cigarette smoke, effect, 82–1556
blocking ventilation, effect on cigarette yield, 82–1768
cellulose acetate filaments structure, 82–1834
cigarette manufacturing process, 82–1829
efficiency, spectrophotometric measurement, 82–1551
grooved rod for smoke dilution, 82–1833
production methods, 82–1828
tar reduction and carbon monoxide conversion, 82–1831
ventilation and filtration system for tar reduction, 82–1824, 82–1832
ventilation, filtration, perforation, and blend type effects on puffing ease and component delivery, 82–1544

FINLAND
cardiovascular risk factor intervention, the North Karelia project, 82–1700
cerebral stroke, smoking relationship, 82–1701
dental expenditures, fluoride, oral health, and smoking relationship, 82–1728
1976 smoking control legislation, 82–1851

FIRES
hazard reduction by cigarette paper coatings, 82–1815
Switzerland, rapid-burning cigarette additives, 82–1813

FLORIDA
lung cancer incidence in Duval County, occupation and smoking factors, 82–1603

FRANCE
antismoking campaign effectiveness, 82–1789
head and neck cancers, alcohol and smoking risks, 82–1604
lung function in Parisian factory workers, dust, gas, heat and smoking effects, 82–1665
smoking pattern changes in Calvados, 82–1782

GASTRIC SECRETION
nicotine effects on acid secretion and mucosal blood flow, 82–1748
reduction of mucosal blood flow, smoking and nicotine effect in healing of ulcer in the rat, 82–1731

GASTROINTESTINAL NEOPLASMS
gastric cancer association with esophageal cancer, incidence in Japan, 82–1622

GASTROINTESTINAL SYSTEM
prostacyclin secretion, smoking effect, 82–1733

GERMAN DEMOCRATIC REPUBLIC
lung cancer among pesticide-exposed agricultural workers, 82–1602

GYNECOLOGIC DISEASES
menopausal age in smokers vs. nonsmokers, 82–1727

GYNECOLOGIC NEOPLASMS
cervical cancer, carcinoembryonic antigen serum levels confounded by smoking and age, 82–1642

HAWAII
cancer incidence among Japanese men in Oahu, smoking relationship, 82–1615

HEAD AND NECK NEOPLASMS
France, alcohol and smoking factors, 82–1604
Ireland, sex ratio and etiology, 82–1616
serum antigen levels in patients vs. controls, smoking effects, 82–1640

HEALTH EDUCATION
antismoking attitudes of doctors and health visitors in Great Britain, 82–1766
antismoking program in French schools, 82–1789
behavioral perspective on antismoking campaigns, 82–1783
cigarette smoke inhalation, dangers, 82–1793
comprehensive smoking control program, national conference proceedings, 82–1784

- employees, emphasis of company smoking cessation program, 82-1787
- Feelin' Good coronary disease intervention program for children, 82-1794
- health insurance industry disease prevention campaign in West Germany, 82-1785
- history and current applications, review, 82-1760
- "Know Your Body" child health program, 82-1648
- myocardial infarction and stroke prevention recommendations, 82-1707
- preventive medicine program proposed for the Soviet Union, 82-1786
- role of physician in smoking cessation, therapeutic recommendations, 82-1807
- school smoking intervention recommendations in West Germany, 82-1796, 82-1798
- smoking control in West German schools, recommendations, 82-1764
- smoking prevention efficacy among London schoolchildren, 82-1779
- Surgeon General's report, effect on state tobacco legislation, 82-1853

HEALTH PRACTICES
- adolescent perceptions of personal and family behavior, 82-1797
- alcohol consumption, dietary habits, coffee drinking, and smoking correlations, 82-1755
- disease prevention association, health education effect, 82-1760
- Health Hazard Appraisal fitness program, smoking reduction effects, 82-1802
- health inequality between social classes in Great Britain, smoking factor, 82-1754
- Mormon men, church involvement and cancer incidence relationship, 82-1611
- Mormon women, church involvement and cancer incidence relationship, 82-1612
- secondary schoolchildren in London, health awareness vs. smoking, 82-1779
- sudden coronary death, smoking as a risk factor, 82-1689

HEART
- bradykinin effects in rabbits and guinea pig coronary vessels, nicotine influence, 82-1710
- nicotinic receptors, acetylcholine effects on potassium movement in guinea pig atrial muscle, 82-1572

HEART DISEASES
- ischemic, risk factors among Finnish immigrants vs. native Swedes, 82-1709
- mortality in men, smoking intervention effect, 82-1800
- mortality rate in the United Kingdom, smoking relationship, 82-1792
- mortality rates, men vs. women, 82-1697

HEART FUNCTION
- cardiac nerves responses to nicotine in dogs, 82-1585
- cigarette smoke exposure effect in rats, 82-1693
- Innovar effects on cardiac response to smoke in rabbits, 82-1678

HEART RATE
- arousal levels, cigarette smoking effects, 82-1763
- Innovar effects on heart function and response to smoke in rabbits, 82-1678
- resting rate among biracial youth in Louisiana, smoking relationship, 82-1713
- varying tar/nicotine levels effect in rats, 82-1693

HEMODYNAMICS
- gastric mucosal blood volume, smoking effects, 82-1571

HEREDITY
- antitrypsin phenotype vs. smoking in emphysema incidence, 82-1659
- causal relationship with coronary disease, pros and cons, 82-1679
- genetic antitrypsin abnormalities vs. smoking in emphysema etiology, 82-1652
- lung cancer proband's sex difference, genetic liability, 82-1610
- lung cancer risk in cancer patient relatives, smoking relationship, 82-1627
- relatives of ischemic heart disease patients, serum cholesterol, triglyceride levels, and smoking habit relationships, 82-1712

HISTAMINE
- plasma levels after smoking, 82-1569

HUMECTANTS
- quarternary ammonium compound, tobacco additive, 82-1814

HYPERCHOLESTEREMIA
- ischemic heart disease patients, incidence in smoking vs. nonsmoking relatives, 82-1712

HYPERLIPIDEMIA
- coronary disease risk screening program, factor with smoking, 82-1704
- myocardial infarction risk factor in young men, with smoking, 82-1690

HYPERTENSION
- causal relationship with coronary disease, pros and cons, 82-1679
- coronary disease risk screening program, factor with smoking, 82-1704
- myocardial infarction intervention, mild hypertension treatment recommendations, 82-1685

nicotine effects on blood pressure in rats and squirrel monkeys, 82–1687
progressive aortocoronary vein graft narrowing, risk factor, 82–1696
serum uric acid level relationship, smoking association, 82–1695
systolic vs. "classical," smoking association, 82–1683, 82–1684

ICELAND
laryngeal neoplasm incidence, tobacco and alcohol relationship, 82–1607

IMMUNE SYSTEM
immunoregulatory T-cell alterations, lung cancer and smoking relationship, 82–1614

INFANTS
respiratory diseases, parental smoking relationship, 82–1658

INTESTINES
benzo(*a*)pyrene metabolism in colon, duodenum, esophagus, and bronchi, 82–1555

IOWA
adolescents, factors influencing smoking behavior in Muscatine, 82–1770

IRELAND
head and neck cancer incidence, sex ratio and etiology, 82–1616

ISCHEMIA
mortality in Sweden, risk factors analysis, 82–1596

ITALY
bronchial mucus gland enlargement in healthy subjects, smokers vs. nonsmokers, 82–1669
lung function and bronchitis among aluminum workers, age, occupation, and smoking factors, 82–1653

JAPAN
bladder cancer incidence in Nagoya, artificial sweetener use and smoking relationship, 82–1633
esophageal and gastric cancer association, 82–1622
import policy, effect on U.S. tobacco industry, 82–1843
serum uric acid levels relationship to hypertension and ischemic heart disease, smoking association, 82–1695
stomach cancer risk factors, 82–1625

KETONES
insulin absorption in ketosis-prone diabetics, smoking effects, 82–1741

KIDNEY DISEASES
renal failure effect on theophylline clearance in obstructive pulmonary disease patients, 82–1650

LABELING
health warnings as part of international tobacco control, recommendation, 82–1790
legislation in Sweden, 82–1799
tar and nicotine content on South African cigarette packets, 82–1846
1976 smoking control legislation in Finland, 82–1851

LARYNGEAL NEOPLASMS
asbestos and smoking synergism in etiology, 82–1634
Iceland, incidence and relationship of tobacco and alcohol use, 82–1607
Ireland, incidence and relationship of smoking and alcohol consumption, 82–1616

LEARNING
maternal nicotine administration in rats, offspring developmental effects, 82–1724

LEGISLATION
Finland, 1976 smoking control law, 82–1851
France, 1976 antismoking law, 82–1789
No Net Cost Tobacco Program Act of 1982—P.L. 97-218, 82–1852
state excise taxation and antismoking laws, tobacco states vs. other regions, 82–1853
Sweden, labeling, advertising, and restrictions on public smoking, 82–1799
United States, tobacco price-support program, economic and production effects, 82–1850
West Germany, antismoking recommendations, 82–1857

LEUKOCYTES
antimicrobial peroxidase system function, smoking effect on crevicular thiocyanate and iodide, 82–1729
hydrocortisone administration effects in control vs. sham-treated vs. smoke-exposed mice, 82–1578
noncholinergic nicotine receptor, specific binding, 82–1561

LIP NEOPLASMS
pipe smoking relationship, 82–1606

LIPIDS
blood concentration, acute smoking effects, 82–1591
smoking cessation effects, 82–1577

LIPOPROTEINS
serum apolipoprotein A-I levels, relationship to cholesterol levels and demographic factors, 82–1699
smoking cessation effects, 82–1577

LIVER
benzo(*a*)pyrene:DNA adduct formation in mice, 82–1568
mutagenicity of enriched polycyclic hydrocarbons in the rat, 82–1562

size and daily cigarette consumption correlation, 82–1767

LONGEVITY
bronchial cancer, patients' survival rates, 82–1629
myocardial infarct survivors, prognosis factors, 82–1702, 82–1715

LOUISIANA
resting heart rate among biracial youth, smoking relationship, 82–1713

LUNG DISEASES
hydrocortisone treatment effects in smoke-exposed vs. sham-treated mice, 82–1578

LUNG FUNCTION
airway disease patients, deterioration rate in smokers vs. nonsmokers, 82–1651
airways narrowing after smoke inhalation, smokers vs. nonsmokers, 82–1672
asbestos exposure, effects in smokers vs. nonsmokers, 82–1660, 82–1670
central and peripheral airways response to smoke in humans and rats, 82–1668
copper in tap water and smoking, relationship, 82–1674
decreased values after smoking, 82–1569
dust, gas, heat exposure effects in Parisian factory workers, smoking factor, 82–1665
emphysema patients, deterioration in smokers vs. ex-smokers, 82–1662
Italian aluminum workers, age, occupation, and smoking factors, 82–1653
pulmonary pressor responses to prostaglandins in rat and hamster lungs, smoke inhibition effect, 82–1575
schoolchildren, gas cooking and parental smoking effect, 82–1656
talc miners and millers, occupational exposure and smoking effects, 82–1676

LUNG NEOPLASMS
arsenic exposure and smoking risks, 82–1619
aryl hydrocarbon hydroxylase from human lung microsomes, smoking effect, 82–1594
asbestos-exposed workers' mortality, smokers vs. nonsmokers, 82–1620, 82–1634
atypical bronchial epithelial cells, objective measurement, 82–1647
Australian men, occupational, dietary, alcohol, and smoking factors, 82–1597
cancer patient relatives, risk and smoking relationship, 82–1627
causal role of cigarettes, critique of theory, 82–1605
cessation of smoking in primary and secondary control, 82–1600
cholesterol levels in youth and smoking, relationship to subsequent development, 82–1643
Croatia, "smoker's cancer" incidence, 1969-1975, 82–1626
Duval County, Florida, incidence and relationship to smoking and occupational factors, 82–1603
early detection and intervention strategies for high-risk individuals, 82–1623
etiology, epidemiology, and prevention, 82–1648
etiology, epidemiology, pathology, diagnosis, staging, and treatment, symposium, 82–1630
histological types, smoking relationship, 82–1635
immunoregulatory T-cell alterations, smoking relationship, 82–1614
Japanese men in Hawaii, incidence and smoking relationship, 82–1615
lung cancer proband's sex differences, family cancer risk relationship, 82–1610
men over 45 years, the Philadelphia Pulmonary Neoplasm Research Project, 82–1645
mortality in Great Britain, inhalation patterns and tar levels relationship, 82–1618
mortality in men, smoking intervention effect, 82–1800
mortality rate in the United Kingdom, smoking relationship, 82–1792
occupational exposure and smoking, 82–1624
pathogenesis, smoking relationship, 82–1601
pesticide-exposed agricultural workers in East Germany, incidence, 82–1602
serum antigen levels in patients vs. controls, smoking effects, 82–1640
UICC program on smoking and lung cancer, 82–1791
World Health Organization recommendations for discouraging tobacco consumption, 82–1646
younger vs. older patients, characteristics and survival, 82–1609

LUNGS
anticarcinogenic retinoid activity in rodent respiratory tissue, 82–1743
arachidonic acid metabolism in the rat, cigarette smoke ventilation effect, 82–1574
benzo(*a*)pyrene:DNA adduct formation in mice, 82–1568
cytologic changes from asbestos vs. smoke exposure, 82–1737
lesion induction in rats by smoke inhalation, 82–1738
nicotine absorption, distribution, and metabolism, 82–1583
prostaglandin inactivation in rat and hamster lungs, cigarette smoke effect, 82–1575

LYMPHOCYTES
aryl hydrocarbon hydroxylase, maternal smoking effect on placenta activity, 82–1723
immunoregulatory T-cell alterations, lung cancer and smoking relationship, 82–1614
nicotine, specific binding, 82–1561

sister-chromatid exchange in mothers and infants, maternal smoking relationship, 82–1725
sister-chromatid exchange relationship to breast cancer, smoking association, 82–1621
sister-chromatid exchanges in smokers exposed to electric and magnetic fields, 82–1730
sister-chromatid exchanges in vitro and in vivo, cigarette smoke condensates effect, 82–1573

MACROPHAGES
hydrocortisone administration effects in control vs. sham-treated vs. smoke-exposed mice, 82–1578

MAMMARY NEOPLASMS
serum antigen levels in patients vs. controls, smoking effects, 82–1640
serum cholesterol, lipids, dietary fat, and smoking in etiology, 82–1617
sister-chromatid exchange incidence in lymphocytes, smoking association, 82–1621

MASSACHUSETTS
student smoking and alcohol consumption, 82–1769

MATERNAL SMOKING
adolescents, pregnancy outcome effects, 82–1718, 82–1722
aryl hydrocarbon hydroxylase, placental activity vs. lymphocyte inducibility, 82–1723
fetal effects, review, 82–1721, 82–1747
fetal preputial neural tissue, effects on, 82–1716
lower respiratory illness in children, relationship, 82–1658
Maori women, pregnancy outcome effects, 82–1726
sister-chromatid exchange in mothers and infants, 82–1725

MATHEMATICAL MODELS
precipitator, initiator, and promoter hypotheses in lung cancer, critique, 82–1605

MESOTHELIOMA
asbestos-exposed workers' mortality, smokers vs. nonsmokers, 82–1620
environmental risk factors, smokers vs. nonsmokers, 82–1649

METALS, METAL ISOTOPES
aluminum workers, age, occupation, and smoking effects on lung function, 82–1653
clastogenicity of saliva from areca and tobacco chewers, effect, 82–1587
manganese in betel quid, clastogenic activities, 82–1588
plasma magnesium levels in smokers vs. nonsmokers, 82–1580
rapid-burning cigarette additives used in Switzerland, 82–1813

MORBIDITY
cancer in Australia, primary and secondary control through risk factor modification, 82–1600
cancer in France, alcohol and smoking factors, 82–1604
cancer in La Paz, Bolivia, smoking relationship, 82–1637
cardiorespiratory disease in men, smoking intervention effect, 82–1800
coronary heart disease in Goteborg, Sweden, synergistic relationship of blood pressure, serum cholesterol, and smoking, 82–1714
Croatia, "smoker's cancer" incidence, 1969-1975, 82–1626
lung cancer and coronary heart disease in Britain, inhalation patterns and tar levels relationship, 82–1618
lung cancer in the United States, 82–1623
lung cancer, Philadelphia Pulmonary Neoplasm Research Project, 82–1645
lung cancer risk in cancer patient relatives, smoking relationship, 82–1627
passive smoking, acute and chronic effects, 82–1751
stroke, smoking as a risk factor in the Netherlands, 82–1686
tobacco-related diseases in adults in the United Kingdom, 82–1792

MORTALITY
asbestos-related cancer, smoking relationship, 82–1634
Australian men, occupational, dietary, alcohol, and smoking factors, 82–1597
bronchial cancer, patients' survival rates, 82–1629
cancer among Mormon men, church involvement relationship, 82–1611
cancer among Mormon women, church involvement relationship, 82–1612
cancer, asbestos-exposed smoking vs. nonsmoking workers, 82–1620
cancer in Australia, primary and secondary prevention through risk factor control, 82–1600
cardiorespiratory disease in men, smoking intervention effect, 82–1800
cardiovascular and oncological diseases in the Soviet Union, 82–1786
cardiovascular diseases in Great Britain, economic change relationship, 82–1677
cardiovascular risk factor intervention, the North Karelia project, 82–1700
coronary heart disease among diabetics, smoking effects, 82–1688
coronary heart disease in Goteborg, Sweden, synergistic relationship of smoking, blood pressure, and serum cholesterol, 82–1714
coronary heart disease in Great Britain, decline since mid-1970s, 82–1706
heart disease death rates, men vs. women, 82–1697

hydrocortisone administration, control vs. sham-treated vs. smoke-exposed mice, 82–1578
insured smokers vs. insured nonsmokers, the State Mutual model, 82–1855
lung cancer among agricultural workers in East Germany, smoking relationship, 82–1602
lung cancer among arsenic workers, smoking factor, 82–1619
lung cancer and coronary heart disease in Britain, inhalation patterns and tar levels relationship, 82–1618
lung cancer and heart disease in the United Kingdom, smoking relationship, 82–1792
lung cancer, Philadelphia Pulmonary Neoplasm Research Project, 82–1645
oral contraceptive users, smokers vs. nonsmokers, 82–1598
pancreatic cancer, smoking-adjusted correlation to coffee consumption, 82–1608
postoperative peptic ulcer patients, smoking relationship, 82–1744
Stockholm Prospective Study, risk factors analysis, 82–1596
sudden coronary death, smoking as a risk factor, 82–1689

MOTIVATION
Canadian schoolchildren in grades 4-6, factors predictive of smoking, 82–1777
peripheral vascular disease patients, smoking cessation advice, 82–1680
personality factors in reasons for smoking, 82–1775
placebo chewing gum as nicotine substitute in smoking cessation, effectiveness, 82–1804
reasons for maintaining or quitting smoking, doctor's advice, 82–1759
situational vs. generalized smoking intervention method, attitudinal factor, 82–1806
smoking attitudes among 16- to 19-year-old apprentices in Switzerland, 82–1753

MOUTH MUCOSA
snuff-induced lesions in rats, experimental model, 82–1739

MOUTH NEOPLASMS
areca chewing and tobacco consumption relationship, 82–1646
aryl hydrocarbon hydroxylase, smoking and oral health relationship, 82–1599
herpes simplex virus and tobacco use in etiology, 82–1606
tongue neoplasm incidence in Irish women, smoking and alcohol consumption relationship, 82–1616

MUSCLES
nicotinic receptors, acetylcholine effects on potassium movement in guinea pig atrial muscle, 82–1572

MUTAGENESIS
cigarette smoke condensate fractions in *Neurospora crassa* and *Salmonella typhimurium*, 82–1562
urine analysis in smoking vs. nonsmoking coffee drinkers vs. nondrinkers, 82–1553

MUTAGENS
alkaline subfraction of cigarette smoke condensate, isolation method and assessment, 82–1567
coal tar and ultraviolet light skin treatment effect on urine of smoking vs. nonsmoking psoriatic patients, 82–1749
enriched polycyclic hydrocarbons from cigarette smoke condensate, 82–1562
Transkei home-grown tobacco pyrolosates, levels, 82–1641
urine analysis in smoking vs. nonsmoking coffee drinkers vs. nondrinkers, 82–1553
urine of smoking and nonsmoking bladder cancer patients, 82–1613

MYOCARDIAL INFARCT
angiographic findings and risk factors in young men, 82–1690
intervention recommendations, 82–1707
long-term survival, predictive factors, 82–1702, 82–1715
mild hypertension and smoking risks, treatment recommendations, 82–1685
oral contraceptive use with smoking as risk factors, 82–1598, 82–1694, 82–1708

NASOPHARYNGEAL NEOPLASMS
English furniture workers' incidence, smoking relationship, 82–1636

NEONATE
fetal preputial neural tissue, maternal smoking effects, 82–1716

NEOPLASMS
carcinoembryonic antigen levels in blood of smokers, predictive factor, 82–1745
cholesterol levels in youth, subsequent cancer development relationship with smoking, 82–1643
control of carcinogenic substances, review, 82–1638
environmental risk factors, smokers vs. nonsmokers, 82–1649
epidemiology and carcinogen management considerations, 82–1631
incidence for all sites in La Paz, Bolivia, smoking relationship, 82–1637
Mormon men, church involvement relationship, 82–1611
Mormon women, church involvement relationship, 82–1612
mortality in Sweden, risk factors analysis, 82–1596
mortality in the Soviet Union, 82–1786

- occupational exposure risk factor, smoking relationship, 82–1624
- primary and secondary control of incidence in Australia through risk factor modification, 82–1600
- smoking-associated, familial risk relationships, 82–1627
- smoking-associated vs. nonsmoking-associated, genetic liability, 82–1610
- World Health Organization assessment of global control measures, 82–1646

NERVOUS SYSTEM DISEASES
- Parkinson's disease, smoking relationship, 82–1734

NETHERLANDS
- stroke, smoking as a risk factor in Tilburg, 82–1686

NEURONS
- cardiac nerves responses to nicotine in dogs, 82–1585
- fetal preputial neural tissue, maternal smoking effects, 82–1716
- nicotinic receptor-mediated potassium movements in guinea pig heart, 82–1572

NEUROTICISM
- personality factors in reasons for smoking, 82–1775

NEW ZEALAND
- Maori women, smoking and drinking effects on pregnancy outcome, 82–1726

NICOTINE
- blocking of filter vents by smoker, effect on yield, 82–1768
- blood pressure effects in rats and squirrel monkeys, 82–1687
- cardiac nerves responses in dogs, 82–1585
- coronary disease, causative role and preventive measures, 82–1711
- dependence factor in smoking cessation maintenance, 82–1762
- gastric secretion and mucosal blood flow, effects, 82–1748
- generation during and between puffs, 82–1545
- maternal administration in rats, offspring behavioral and developmental effects, 82–1724
- noncholinergic receptor in human phagocytic leukocytes, 82–1561
- peptic ulcer healing, effect with smoking in the rat, 82–1731
- pharmacokinetics in humans and animals, 82–1583
- plasma level variation in smokers and nonsmokers, consumption effect, 82–1580
- plasma levels among pipe smokers, inhalation patterns relationship, 82–1774
- plasma levels from nicotine chewing gum vs. smoking, 82–1776
- plasma levels, smoking reduction effect, 82–1765
- platelet thromboxane synthesis, effect on in humans, 82–1554
- sperm and egg responses in the sea urchin, 82–1732
- stress in rats, administration and withdrawal effects, 82–1752
- vigilance performance, effect of low vs. medium levels, 82–1771
- yield reduction and smoking pattern relationship, 82–1780

NICOTINE CONTENT
- added as inhalant material by smoking device, 82–1823
- Argentina, tobacco crop improvement efforts, 82–1849
- nitrogen fertilization effects on Burley tobacco, 82–1535
- tar/nicotine ratio recommendation, 82–1856
- tobacco varieties and agricultural practices effect, level and yield/ha, 82–1534
- yields of 10 Canadian small cigar brands, 82–1854

NICOTINE METABOLISM
- characteristics in humans and animals, 82–1583

NICOTINE PHARMACOLOGY
- acetylcholine effect on guinea pig atrial muscle, 82–1572
- behavioral responses in rats, 82–1563
- bradykinin effects on rabbits and guinea pig hearts, influence, 82–1710
- coronary arterial response to nicotine in dogs, effect of various drugs, 82–1592
- prostration—immobilization syndrome, cholinergic antagonists' effect, 82–1584

NICOTINE REDUCTION
- low-yield smoking compositions, quarternary ammonium compounds for flavoring, 82–1814
- protein extraction from tobacco, relationship, 82–1550
- tobacco expansion process, 82–1825

NITRATES
- aging effects on concentration in Burley tobacco, 82–1536

NITROGEN COMPOUNDS
- aging effects on concentration in Burley tobacco, 82–1536
- nicotine levels vs. nitrogen fertilization in Burley tobacco, 82–1535

NITROGEN CONTENT
- nicotine level in tobacco leaf and yield/ha, relationship, 82–1534

NITROSAMINE CONTENT
- protein extraction from tobacco, relationship, 82–1550

NONSMOKERS
- airway disease, site and time course airflow obstruction study, 82–1651
- bronchial airways resistance, smoke inhalation effect, 82–1672
- passive smoking, acute and chronic effects, 82–1751
- preferential insurance packages, the State Mutual model, 82–1855

NORNICOTINE
- aging effects on concentration in Burley tobacco, 82–1536

OBESITY
- coronary disease risk screening program, factor with smoking, 82–1704
- myocardial infarction risk factor in young men, with smoking, 82–1690

OBSTRUCTIVE AIRWAY DISEASES
- lung cancer predisposing factor, with occupation, 82–1603
- site and time course airflow obstruction study, 82–1651
- theophylline clearance, smoking and kidney and heart failure, relationship, 82–1650
- thickened mucous layers, wave frequency effect in wideband acoustic energy studies, 82–1663

OCCUPATIONAL DISEASES
- cancer among asbestos-exposed workers, 82–1620
- cancer, industrial exposure and smoking effect, 82–1624
- cancer risk management, policy considerations, 82–1638
- lung cancer among arsenic workers, risk with smoking, 82–1619
- lung cancer, asbestos or uranium exposure as factors with smoking, 82–1623
- lung cancer incidence in Duval County, Florida, smoking factor, 82–1603
- nasal adenocarcinoma and bronchial cancer in English furniture workers, smoking relationship, 82–1636
- respiratory abnormalities in asbestos workers, smoking effect, 82–1660, 82–1670
- respiratory symptoms among Polish chemical workers, smoking factor, 82–1664

OCCUPATIONS
- agricultural workers, 82–1602
- Air Force personnel, 82–1698
- aluminum workers, 82–1653
- arsenic workers, 82–1619
- asbestos workers, 82–1620, 82–1660, 82–1670
- factory workers in Paris, 82–1665
- furniture workers, 82–1636
- industrial apprentices, 82–1753
- office workers, 82–1736
- physicians, 82–1766
- Polish chemical workers, 82–1664
- switchyard workers, 82–1730
- talc miners and millers, 82–1676
- welders, 82–1667

ORAL HYGIENE
- tooth loss among adults in Finland, factor with smoking, 82–1728

PAKISTAN
- tobacco industry trends, 1981 and 1982, 82–1837

PANCREATIC NEOPLASMS
- coffee consumption and smoking correlations, 82–1608, 82–1639

PARENTAL SMOKING
- adolescent smoking behavior, influence on, 82–1770, 82–1773, 82–1788
- respiratory health of children, relationship, 82–1656, 82–1658
- West German 9- and 10-year-olds' smoking habits, influence on, 82–1756

PARKINSONISM
- smoking relationship, 82–1734

PASSIVE SMOKING
- blood platelet sensitivity to antiaggregatory prostaglandins, effect, 82–1705
- fetus, maternal smoking effects, 82–1721
- health risks, review, 82–1747, 82–1751
- hidden health cost to companies, 82–1787
- indoor air quality, smoking factor, 82–1750
- lung cancer relationship questioned, 82–1648
- office air quality, health effects, 82–1736
- pregnant women, amniotic fluid cotine level relationship, 82–1717
- respiratory health of children, parental smoking relationship, 82–1656, 82–1658

PATENTS
- acyclic carboxamides for cooling flavor effects, 82–1812
- carbon dioxide for tobacco expansion, 82–1825
- cigarette-simulating smokeable device, 82–1823
- cis-3-hexanol derivatives for tobacco flavor and aroma, 82–1810
- cyclic carboxamides as a cooling flavorant, 82–1811
- cyclic chemical compounds, organoleptic uses, 82–1818
- cyclohexenemethanols as organoleptic agents for tobacco, 82–1819
- 1-ethoxy-1-ethanol acetate as flavorant, 82–1808
- filter, cellulose acetate filaments structure, 82–1834
- filter for tar reduction and carbon monoxide conversion, 82–1831

- filter production methods, 82–1828
- filter-tipped cigarette manufacturing process, 82–1829
- filter using ventilation and filtration, 82–1824, 82–1832
- grooved filter rod for smoke dilution, 82–1833
- 1-N-butoxy-1-ethanol as flavorant, 82–1809
- oxabicyclooctane derivative as flavorant, 82–1817
- pipe with cooling capacity, 82–1830
- reaction flavors production, 82–1822
- reconstituted tobacco preparation, tobacco dust use, 82–1827
- self-extinguishing cigarette paper coatings, 82–1815
- smoking compositions containing a quarternary ammonium compound, 82–1814
- substituted tricyclodecane derivatives, additives, 82–1816
- tipping material with ventilatory and nonsticking properties, 82–1835
- tobacco-flavored chewing gum, 82–1826
- tricyclic carbonyl derivative as tobacco flavorant, 82–1821
- 2, 4, 6-trimethylcyclohexanemethanol and derivatives, 82–1820

PATERNAL SMOKING
- lower respiratory illness in children, relationship, 82–1658

PEER GROUPS
- adolescent smoking behavior, influence on, 82–1770, 82–1773, 82–1788
- adolescent smoking, prevention programs, 82–1803
- 9- and 10-year-olds in West Germany, smoking habits relationship, 82–1756

PEPTIC ULCER
- healing rate in duodenal ulcer patients treated with ranitidine, smoking effect, 82–1742
- healing, smoking and nicotine effect in the rat, 82–1731
- mortality among postoperative patients, smoking relationship, 82–1744
- nicotine in etiology, mechanisms, 82–1748
- prostacyclin secretion in etiology, smoking effect, 82–1733
- risk factors and stomach cancer relationship in Japan, 82–1625

PERCEPTION
- adolescents, personal and family health behaviors, 82–1797
- seventh grade students, adolescent smoking predictor factor, 82–1773

PERSONALITY
- German army recruits, relation to smoking habit, 82–1761
- types of smokers and smoking habits, recommendations for cessation therapy, 82–1807

PESTICIDES
- lung cancer among East German agricultural workers, relationship, 82–1602

PHARYNX
- oropharyngeal bacteria, in vitro cigarette smoke toxicity, 82–1556

PHENOLS
- catechol synthesis, 82–1540
- derivatives, glass capillary gas chromatography analysis, 82–1537

PHYSICAL ACTIVITY
- blood pressure, effects on, 82–1682
- myocardial infarct survival, factor in prognosis, 82–1702, 82–1715

PIPE SMOKING
- esophageal cancer relationship with alcohol in Transkei, South Africa, 82–1632
- inhalation patterns, 82–1774
- lip carcinoma relationship, 82–1606

PIPE TOBACCO
- production and sales statistics for Great Britain, 1961-1980, 82–1845

PLACENTA
- aryl hydrocarbon hydroxylase, maternal smoking effect on lymphocyte inducibility, 82–1723
- cholesterol C-20, 22-desmolase activity in humans, cigarette smoke effect, 82–1720

PLASMA
- carcinoembryonic antigen levels as cancer predictor, 82–1745
- clotting time and fibrin aggregation, smoke effects, 82–1564

POLAND
- polycyclic aromatic hydrocarbons in 5 cigarette brands, 82–1547
- respiratory symptoms among chemical workers, smoking factor, 82–1664

POLONIUM-210
- bronchial cancer, etiology, 82–1628

PRECANCEROUS CONDITIONS
- benzo(*a*)pyrene:DNA adduct formation, in forestomach, lung and liver of mice, 82–1568
- carcinoembryonic antigen levels in blood of smokers, 82–1745
- epithelial cytologic changes, smoking and asbestos exposure effect, 82–1737

lesion induction in rat lungs by smoke inhalation, 82–1738
respiratory cellular atypia, objective measurement, 82–1647
smoke-induced pathogenic changes in respiratory epithelial cells, 82–1601

PREGNANCY
cotinine in amniotic fluid from maternal passive smoking, 82–1717
fetal preputial neural tissue, maternal smoking effects, 82–1716
human placental cholesterol C-20, 22-desmolase activity inhibition by cigarette smoke extracts in vitro, 82–1720
maternal weight gain and outcome in adolescents, smoking effect, 82–1718
miscarriage and stillbirth correlation to smoking and drinking, 82–1726
nicotine administration in rats, offspring behavioral and developmental effects, 82–1724

PREVENTION OF SMOKING
adolescent smoking behavior predictor factors, relationship, 82–1773
adolescents, resisting advertising and peer pressure, 82–1788
adolescents, use of peer leaders, 82–1803
antismoking campaign in France, effectiveness, 82–1789
attitudes of Canadian schoolchildren, implications, 82–1777
comprehensive school health education, national conference proceedings, 82–1784
school smoking intervention recommendations in West Germany, 82–1796, 82–1798
World Health Organization recommendations for tobacco control, 82–1646

PROGRAMS AND POLICIES
behavioral perspective on antismoking campaigns, 82–1783
Great Britain, health inequality between social classes, recommendations, 82–1754
health education in behavior change for disease prevention, history and review, 82–1760
nonsmoker's insurance package, the State Mutual model, 82–1855
strategy for international tobacco control, recommendations, 82–1790
UICC program on smoking and lung cancer, 82–1791

PROSTAGLANDINS
prostaglandins E_1 and $F_{2\alpha}$ metabolism in rat and hamster lungs, cigarette smoke effect, 82–1575

PROTEIN CONTENT
extraction processes, less hazardous cigarette relationship, 82–1550

PROTEINS
fibrin aggregation, inhibition by smoke condensate, 82–1564

PSYCHIATRIC DISORDERS
psychoticism, smoking relationship, 82–1775

PULMONARY ALVEOLAR MACROPHAGES
activation in sarcoidosis vs. cryptogenic fibrosing alveolitis patients, smoking effect, 82–1657
antitrypsin and macrophage levels in emphysema etiology, smoking effect, 82–1652
elastase releasing factor in smokers, possible emphysema mechanisms, 82–1654

PULMONARY FIBROSIS
collagen production as an etiological factor, smoking relationship, 82–1559
environmental risk factors, smokers vs. nonsmokers, 82–1649

PULMONARY HEART DISEASE
heart and kidney function effect on theophylline clearance in obstructive pulmonary disease patients, 82–1650

RADIATION
determination in urine of smokers of phencyclidine-treated parsley cigarettes, 82–1560
polonium-210, alpha radiation in bronchial cancer etiology, 82–1628

REDUCTION OF SMOKING
behavioral perspective on antismoking campaigns, 82–1783
blood pressure, body weight, and blood serum cholesterol effects, 82–1703
cardiorespiratory disease in men, effect on, 82–1800
coronary disease risk screening, effect, 82–1704
cost-effective strategies in the United Kingdom, 82–1792
Health Hazard Appraisal fitness program, effects, 82–1802
inhalation behavior change, 82–1765
peripheral vascular disease patients, surgeon's advice effect, 82–1680
psychologists' role, 82–1772
situational vs. generalized approach, 82–1806
smoking control program effect in Sweden, 82–1799
smoking control recommendations in Austria, 82–1795
taxation effects on national consumption in Great Britain, 82–1841

RESPIRATION
- pattern changes in smoke- vs. sham-exposed rats, 82–1693

RESPIRATORY FUNCTION TESTS
- airflow obstruction time course and site determination, smokers vs. nonsmokers, 82–1651
- asbestos exposure and smoking effect, 82–1660
- bronchoscopy comparison in chronic cough diagnosis, 82–1671
- copper in tap water and smoking, relationship, 82–1674
- decreased values after smoking, 82–1569
- density-dependent flow-volume measurement, 82–1673
- emphysema progression analysis in smokers vs. ex-smokers, 82–1662
- talc exposure effects, smoking relationship, 82–1676

RESPIRATORY MUCOSA
- angiotensin-converting enzyme secretion, acute smoking effects, 82–1579

RESPIRATORY SYMPTOMS
- airways narrowing after smoke inhalation, smokers vs. nonsmokers, 82–1672
- Polish chemical workers, occupational vs. smoking factors, 82–1664
- schoolchildren, parental smoking and gas cooking relationship, 82–1656
- secondary schoolchildren in London, smoking relationship, 82–1779
- welders, absenteeism in smokers vs. nonsmokers, 82–1667

RESPIRATORY SYSTEM
- central and peripheral airways response to smoke in humans and rats, 82–1668
- cytologic changes from asbestos vs. smoke exposure, 82–1737
- passive smoking effects, review, 82–1747, 82–1751
- smoke-induced pathogenic changes to lung cancer, 82–1601
- transepithelial electric potential differences in humans, smoking effect, 82–1666
- wideband acoustic energy studies, smokers vs. nonsmokers, 82–1663

RESPIRATORY TRACT DISEASES
- asbestos exposure and smoking relationship, 82–1670
- infants and children, parental smoking relationship, 82–1658
- Switzerland, risk from rapid-burning additives, 82–1813
- welders, absenteeism and smoking relationship, 82–1667

RESPIRATORY TRACT NEOPLASMS
- histological diagnosis in Iceland, classifications, 82–1607

SALIVA
- clastogenicity, areca and tobacco chewers vs. non-chewers, 82–1587

SELENIUM
- lung cancer chemotherapy, potential, 82–1623

SEROTONIN
- plasma levels after smoking, 82–1569

SEX RATIO
- bronchial mucus gland enlargement, smoking relationship, 82–1669
- cancer occurrence, implications for carcinogen management, 82–1631
- cancer risk in relatives of lung cancer patients, 82–1610
- church involvement and cancer incidence relationship in Mormons, 82–1612
- esophageal cancer, alcohol consumption and smoking incidence in Transkei, South Africa, 82–1632
- head and neck cancer in Ireland, tobacco and alcohol consumption relationship, 82–1616
- heart disease mortality rates, 82–1697
- international smoking rates among teenagers, overview, 82–1778
- lung cancer patient by histological type, 82–1635
- lung cancer patients, younger vs. older, 82–1609
- mortality in Sweden, risk factors analysis, 82–1596
- nitrazepam clearance, effect with oral contraceptive use, menstrual cycle, and smoking, 82–1570
- secondary schoolchildren in London, smoking patterns, 82–1779
- serum apolipoprotein A-I levels, relationship with cholesterol levels and demographic factors, 82–1699
- "smoker's cancer" incidence in Croatia, 1969-1975, 82–1626
- smoking and alcohol consumption among students in eastern Massachusetts, 82–1769
- smoking habit in Calvados, France, 82–1782

SKIN
- conductance response, arousal levels vs. cigarette smoking, 82–1763

SKIN DISEASES
- psoriasis, mutagenicity of urine in smokers vs. nonsmokers treated with coal tar and ultraviolet light, 82–1749

SLEEP
- nitrazepam clearance, sex, oral contraceptive use, menstrual cycle, and smoking effects, 82–1570

SMALL AIRWAYS DISEASE
- detection using density-dependent flow-volume measurement, 82–1673

SMOKE INHALATION, ANIMAL
- central and peripheral airways response in rats, 82–1668
- dosimetry and cardiopulmonary function effects in rats, 82–1693
- hydrocortisone-treated mice, pulmonary macrophage and blood leukocyte effects, 82–1578
- Innovar effects on cardiovascular function and response to smoke in rabbits, 82–1678
- lesion induction in rat lungs, 82–1738
- prostaglandin metabolism in air-ventilated isolated lungs of rat and hamster, 82–1575

SMOKE STREAMS
- maleic hydrazide content, transfer rate from tobacco, 82–1539
- passive smoking health risks, in-depth review, 82–1747
- ventilation, filtration, perforation, blend type effects on puffing ease and component delivery, 82–1544

SMOKING ARTICLES
- cigarette holder, smoking behavior effect, 82–1781
- cigarette-simulating smokeable device, 82–1823
- pipe with air injection mechanism, 82–1830

SMOKING CHARACTERISTICS
- cigarette burning characteristics during and between puffs, 82–1545
- cigarette length vs. behavioral responses, 82–1757
- compensatory smoking and nicotine dependency, 82–1762
- inhalation behavior change with reduced smoking, 82–1765
- inhalation depth, self-assessment vs. objective measurements, 82–1586
- inhalation pattern relationship to lung cancer and cardiovascular mortality, 82–1618
- inhalation patterns among pipe smokers, 82–1774
- inhalation patterns, cigarettes vs. pipe or cigar, 82–1793
- inhalation patterns vs. tar/nicotine ratio, 82–1856
- low-yield cigarettes, behavioral misuse, 82–1768
- low-yield cigarettes, puff and inhalation patterns, 82–1780
- minicigarette weight loss per puff and carbon oxides delivery, relationship, 82–1552
- puffing and inhalation patterns with high- vs. low-tar cigarettes, 82–1781
- ventilation, filtration, perforation, blend type effects on puffing ease and component delivery, 82–1544

SMOKING CONTROL PROGRAMS
- Austria, recommendations, 82–1795
- blueprint for action, national antismoking conference proceedings, 82–1784
- cardiorespiratory disease mortality in men, intervention effect, 82–1800
- Feelin' Good coronary disease intervention program for children, 82–1794
- Finland, 1976 legislation, 82–1851
- France, antismoking campaign, 82–1789
- Health Hazard Appraisal fitness program, smoking reduction effects, 82–1802
- strategy for international tobacco control, recommendations, 82–1790
- Sweden, 82–1799
- UICC program on smoking and lung cancer, 82–1791
- West German schools, recommendations, 82–1764
- West Germany, recommendations, 82–1793, 82–1857
- World Health Organization recommendations for tobacco control, 82–1646

SMOKING HABIT
- adolescents and adults in the United States, 82–1772
- adolescents, girls vs. boys ratio, international overview, 82–1778
- adolescents in Muscatine, Iowa, 82–1770
- adolescents, 16- to 19-year-old apprentices in Switzerland, 82–1753
- Air Force personnel, risk factor in cardiovascular disease intervention program, 82–1698
- antismoking activities effect in Sweden, 82–1799
- bronchial cancer patients, survival rate relationship, 82–1629
- children, 9- and 10-year-olds in West Germany, 82–1756
- esophageal cancer with gastric cancer, patients in Japan, 82–1622
- Finnish immigrants vs. native Swedes, 82–1709
- fourth and fifth graders in Canada, 82–1777
- German army recruits, personality factors, 82–1761
- Iceland, laryngeal cancer incidence relationship, 82–1607
- induced abortion patients, 82–1719
- lung cancer patients, younger vs. older, 82–1609
- males in Soviet Union cities, 82–1786
- myths related to tobacco consumption, 82–1758
- peptic ulcer patients, postoperative mortality relationship, 82–1744
- peripheral vascular disease patients, 82–1680
- personality factors in reasons for smoking, 82–1775
- population survey in Calvados, France, 82–1782
- secondary schoolchildren in London, 82–1779
- self-reporting vs. carboxyhemoglobin measurement in determination, 82–1582
- seventh grade students, predictor variables, 82–1773
- students in eastern Massachusetts, aged 10 to 18, 82–1769
- varying types, recommendations for quitting methods, 82–1807

welders, effect on absenteeism, 82–1667
WHO cardiovascular disease intervention program in Europe, review, 82–1691
women, effect on age at menopause, 82–1727
young people and adults in France, 82–1789

SMOKING MACHINES
modifications, actual smoking pattern assessment, 82–1768

SMOKING SURVEYS
Australian men, occupational mortality rates relationship, 82–1597
Canada, schoolchildren in grades 4-6, 82–1777
eastern Massachusetts students, aged 10 to 18, 82–1769
France, population survey in Calvados, 82–1782
Great Britain, doctors and health visitors, 82–1766
Great Britain, schoolchildren in London, 82–1779
Iowa adolescents, factors influencing smoking decisions, 82–1770
problem drinkers and alcoholics, 82–1755
seventh grade students, attitudes and predictor variables, 82–1773
Soviet Union, urban males, 82–1786
Switzerland, motives among 16- to 19-year-old apprentices, 82–1753
Transkei, South Africa, esophageal cancer relationship with alcohol, 82–1632
West Germany, 9- and 10-year-old children, 82–1756

SNUFF
consumption trends in the United States, 82–1842
oral mucosal lesions induced in rats, experimental model, 82–1739
production and sales statistics for Great Britain, 1961-1980, 82–1845

SOCIOECONOMIC STATUS
cigarette consumption patterns in Great Britain, 82–1841
health inequality between social classes in Great Britain, smoking factor, 82–1754

SOUTH AFRICA
esophageal cancer incidence in Transkei, risk factors, 82–1632, 82–1641
rise in leaf production planned, 82–1846

SOUTH CAROLINA
problem drinkers and alcoholics, diet, coffee drinking, and smoking correlations, 82–1755

SPERMATOZOA
nicotine effects in the sea urchin, 82–1732
semen quality in smokers vs. nonsmokers, 82–1746

SPUTUM
α_1-proteinase inhibitor, electrophoretic mobility effects of cigarette smoke, 82–1589

STATISTICAL ANALYSIS
bladder cancer and nonnutritive sweetener use relationship, smoking effects, 82–1644
blood pressure variables, cross-sectional analysis, 82–1683
blood pressure variables, prospective analysis, 82–1684
cadmium and other metals, total burdens for "standard man" determination, 82–1541
Stockholm Prospective Study, mortality risk factors analysis, 82–1596

STOMACH
benzo(*a*)pyrene:DNA adduct formation in mice, 82–1568
gastric mucosal blood volume, smoking effects, 82–1571
healing of ulcers in the rat, smoking and nicotine effect, 82–1731
nicotine effects on acid secretion and mucosal blood flow, 82–1748
prostacyclin secretion, smoking effect, 82–1733

STOMACH NEOPLASMS
esophageal cancer association, incidence in Japan, 82–1622
Japan, high risk associations, 82–1625

STRESS
arousal levels, cigarette smoking effects, 82–1763
coronary disease risk factor with smoking, effect on blood platelets activation, 82–1681
myocardial infarct, factor in prognosis, 82–1715
nicotine administration and withdrawal effects in rats, 82–1752

STUDENTS
elementary and secondary schools in France, antismoking campaign, 82–1789
elementary school, smoking habits of 9- and 10-year-olds in West Germany, 82–1756
middle school, smoking prevention program effectiveness, 82–1788
secondary schoolchildren in London, smoking patterns, 82–1779
seventh graders, adolescent smoking predictor factors, 82–1773
smoking and alcohol consumption in eastern Massachusetts, 82–1769
tenth-graders, personal and family health behaviors, 82–1797

SWEDEN
coronary disease prevention trial in Goteborg, synergistic risk factors, 82–1714

ischemic heart disease risk factors among Finnish immigrants vs. natives, 82–1709
smoking control measures, review, 82–1799
Stockholm Prospective Study, mortality risk factors analysis, 82–1596

SWITZERLAND
fire hazards from rapid-burning cigarette additives, 82–1813
smoking motives among 16- to 19-year-old apprentices, 82–1753

TAR CONTENT
levels in 47 varieties of cigarettes in Canada, 82–1538
tar/nicotine ratio recommendation, 82–1856

TAR REDUCTION
cigarette filter which combines ventilation and filtration, 82–1824, 82–1832
filter assembly, 82–1831
filter with cellulose acetate filaments structure, 82–1834
low-yield smoking compositions, quarternary ammonium compounds for flavoring, 82–1814
pipe with cooling capacity, condensation effect, 82–1830
tobacco expansion process, 82–1825

TARS, CIGARETTE
alkaline subfraction isolation method and mutagenicity assessment, 82–1567
anticarcinogenic retinoid activity in rodent respiratory tissue, 82–1743
blocking of filter vents by smoker, effect on yield, 82–1768
catechol synthesis, 82–1540
dry condensate generation during and between puffs, 82–1545
filter collection efficiency, spectrophotometric measurement, 82–1551
humidity change response, 82–1543
lung cancer and coronary heart disease mortality in Britain, relationship, 82–1618
mutagenicity of various fractions in *Neurospora crassa* and *Salmonella typhimurium* , 82–1562
quantification by glass capillary gas chromatography, 82–1537
refractive index of smoke droplets, 82–1546
sister-chromatid exchange incidence in vitro and in vivo, effect on, 82–1573
yield reduction and smoking pattern relationship, 82–1780

TARS, TOBACCO
cigarette smoke oxidant content relationship, 82–1655
yields of 10 Canadian small cigar brands, 82–1854

TAXATION
Colombian cigarettes, local industrial and legal import effect, 82–1848
Great Britain, tobacco consumption effects, 82–1841, 82–1844
international tobacco control strategy, recommendation, 82–1790
state excise taxes, tobacco states vs. other regions, 82–1853

TEXAS
cadmium and other metals in human tissue, determination for "standard man", 82–1541

THIOCYANATES
crevicular concentrations of thiocyanate and iodide, smoking relationship, 82–1729
plasma levels and platelet monoamine oxidase correlation in female smokers vs. nonsmokers, 82–1581
plasma levels, low-yield cigarettes, smoking characteristics, 82–1780

THROMBOEMBOLISM
oral contraceptive use and smoking as risk factors, 82–1694, 82–1708

TOBACCO ADDITIVES
acyclic carboxamides for cooling flavor effects, 82–1812
carbonyl derivative, organoleptic properties, 82–1821
cis-3-hexenol derivatives for flavor and aroma, 82–1810
cyclic carboxamides as a cooling flavorant, 82–1811
cyclic chemical compounds, for flavor and aroma, 82–1818
cyclohexenemethanols as organoleptic agents, 82–1819
1-ethoxy-1-ethanol acetate as flavorant, 82–1808
monitoring for carcinogenicity recommended, 82–1648
1-N-butoxy-1-ethanol as flavorant, 82–1809
oxabicyclooctane derivative as flavorant, 82–1817
quarternary ammonium compound, organoleptic and humectant, 82–1814
rapid-burning admixtures used in Switzerland, 82–1813
reaction flavors production, 82–1822
substituted tricyclodecane derivatives for flavor and aroma, 82–1816
2, 4, 6-trimethylcyclohexanemethanol and derivatives, 82–1820

TOBACCO AGING
chemical components of Burley, effects on, 82–1536

TOBACCO AROMA
cis-3-hexanol derivatives as additives, 82–1810
cyclic chemical compounds, additives, 82–1818
cyclohexenemethanols, additives, 82–1819
1-ethoxy-1-ethanol acetate, 82–1808

1-N-butoxy-1-ethanol, 82–1809
reaction flavors production, 82–1822
substituted tricyclodecane derivatives, additives, 82–1816
tricyclic carbonyl derivative as additive, 82–1821
2, 4, 6-trimethylcyclohexanemethanol and derivatives, additives, 82–1820

TOBACCO CHEWING
oral cancer, relationship, 82–1606

TOBACCO CONTAMINANTS
maleic hydrazide, transfer rate from tobacco to smoke, 82–1539

TOBACCO CURING
leaf- vs. air-cured Burley, aging effects on chemical components, 82–1536

TOBACCO EXTRACTS
collagen biosynthesis effects, 82–1559
protein fractions, 82–1550

TOBACCO FLAVOR
acyclic carboxamides for cooling, 82–1812
added as inhalant material by smoking device, 82–1823
cis-3-hexanol derivatives as additives, 82–1810
cyclic carboxamides for cooling, 82–1811
cyclic chemical compounds, additives, 82–1818
cyclohexenemethanols, additives, 82–1819
1-ethoxy-1-ethanol acetate, 82–1808
1-N-butoxy-1-ethanol, 82–1809
oxabicyclooctane derivative as additive, 82–1817
reaction flavors production, 82–1822
substituted tricyclodecane derivatives, additives, 82–1816
tricyclic carbonyl derivative as additive, 82–1821
2, 4, 6-trimethylcyclohexanemethanol and derivatives, additives, 82–1820

TOBACCO INDUSTRY
Argentina, production and consumption, 82–1849
Colombia, 1981 production and consumption, 82–1848
fights against smoking control, opposition strategy, 82–1790
Great Britain, increased taxation and diminished consumption effects, 82–1841
Great Britain, production and sales statistics for 1961-1980, 82–1845
Great Britain, taxation and public opinion as factors in decline, 82–1844
Pakistan, economic trends, 1981 and 1982, 82–1837
South Africa, rise in leaf production planned, 82–1846
tobacco support system program modifications—No Net Cost Tobacco Program Act of 1982, 82–1852
United States, 1980 advertising expenditures, 82–1840
United States, effect of Japanese import policies, 82–1843
United States, generic products success, 82–1838
United States, Government tobacco program effects on farmers and market competitiveness, 82–1850
United States, marketing data for 1980, 82–1836
United States, Maxwell market report on cigar brands, 82–1839
United States, smokeless products, consumption trends, 82–1842
Zimbabwe, economic production trends of flue-cured and Burley, 82–1847

TOBACCO LEAF COMPONENTS
aging effects in Burley, 82–1536
catechol synthesis, 82–1540
chemical composition, review, 82–1542
nicotine level and yield, effect of varieties and agricultural practices, 82–1534
nicotine levels vs. nitrogen fertilization in Burley tobacco, 82–1535

TOBACCO PROCESSING
carbon dioxide for expansion, 82–1825
protein extraction processes, 82–1550
reconstituted tobacco preparation, tobacco dust use, 82–1827

TOBACCO PYROLYSIS PRODUCTS
chemical composition, review, 82–1542
maleic hydrazide, transfer rate from tobacco to smoke, 82–1539
marihuana comparison, 82–1549
mono- and dihydroxybenzenes, glass capillary gas chromatography analysis, 82–1537

TOBACCO, RECONSTITUTED
tobacco dust use in preparation, 82–1827

TOBACCO SMOKE
marihuana smoke components comparison, 82–1549

TOBACCO SUBSTITUTES
chewing gum, tobacco-flavored, 82–1826
coca leaves chewing, impact on cancer incidence, 82–1637
nicotine chewing gum in smoking cessation, 82–1762
nicotine chewing gum, plasma nicotine levels, 82–1776
placebo chewing gum, effectiveness in smoking cessation, 82–1804

TOBACCO VARIETIES
air-cured and leaf-cured Burley, aging effects on chemical components, 82–1536
Burley, nitrogen and potassium fertilization effect on leaf components, 82–1535
dark-leaf, Virginia, and Burley production in Colombia, 82–1848

flue-cured vs. Burley, economic trends in Zimbabwe, 82–1847
nicotine level and yield in tobacco leaf, relationship, 82–1534
Virginia and dark-leaf production in Argentina, 82–1849

TRACHEA
transepithelial electric potential difference measurement, cigarette smoke effect, 82–1666

TRACHEAL NEOPLASMS
carcinogenicity of benzo(*e*)pyrene with benzo(*a*)pyrene in the mouse, 82–1593

TRACHEOBRONCHIAL EPITHELIUM
anticarcinogenic retinoid activity in rodent respiratory tissue, 82–1743
transepithelial electric potential differences, measurement in vivo in humans, smoking effect, 82–1666

TRIGLYCERIDES
levels in relatives of ischemic heart disease patients, smoking effect, 82–1712
progressive aortocoronary vein graft narrowing, risk factor, 82–1696
serum apolipoprotein A-I levels, relationship to cholesterol levels and demographic factors, 82–1699

TUBERCULOSIS
predisposing factors among patients in Australia, 82–1661

TURKEY
pleuro-pulmonary disease in Tuzkoy, environmental risk factors with smoking, 82–1649

UNION OF SOVIET SOCIALIST REPUBLICS
preventive medicine program proposed, 82–1786

UNITED KINGDOM
antismoking education, attitudes of doctors and health visitors, 82–1766
bladder cancer incidence in Manchester, artificial sweetener use and smoking relationship, 82–1633
cardiovascular disease mortality, economic change relationship, 82–1677
coronary disease incidence and recommended preventive measures, 82–1711
coronary heart disease mortality among diabetics, smoking effects, 82–1688
coronary heart disease, mortality decline since mid-1970s, 82–1706
furniture workers' cancer incidence, smoking relationship, 82–1636
health inequality between social classes, smoking factor, 82–1754
lung cancer and coronary heart disease vs. inhalation patterns and tar levels, 82–1618
lung cancer etiology, analysis and critique of smoking data, 82–1605
morbidity and mortality, smoking relationship, 82–1792
secondary schoolchildren in London, smoking patterns, 82–1779
taxation policies and public opinion effect on tobacco industry, 82–1844
tobacco industry statistics, 1961-1980, 82–1845
tobacco industry, taxation and diminished consumption effects, 82–1841

UNITED STATES
control and management of carcinogenic substances, review, 82–1638

URANIUM
lung cancer, etiological factor with smoking, 82–1623

URIC ACID
serum levels relationship to hypertension and ischemic heart disease, smoking relationship, 82–1695

URINE
cadmium and other metals content analysis, determination for "standard man", 82–1541
mutagenicity in smoking vs. nonsmoking psoriatic patients treated with coal tar and ultraviolet light, 82–1749
mutagenicity in smoking and nonsmoking bladder cancer patients, 82–1613
mutagenicity, smoking vs. nonsmoking coffee drinkers vs. nondrinkers, 82–1553
nicotine excretion levels in animals and humans, 82–1583

UTERUS
endometrium, benzo(*a*)pyrene metabolism, 82–1576

VASCULAR DISEASES
peripheral arteries, smoking effects, 82–1680

VIRUSES
herpes simplex virus in oral cancer etiology, tobacco use relationship, 82–1606

VITAMIN A
anticarcinogenic retinoid activity in rodent respiratory tissue, 82–1743
lung cancer chemotherapy, potential, 82–1623

VITAMIN C
lung cancer chemotherapy, potential, 82–1623

WITHDRAWAL SYMPTOMS
deprivation stress in nicotine-treated rats, 82–1752
nicotine dependence factor, 82–1762

WOMEN
- cardiovascular disease, oral contraceptives with smoking as risk factors, 82–1692
- cotinine in amniotic fluid from maternal passive smoking, 82–1717
- Croatia, "smoker's cancer" incidence, 1969-1975, 82–1626
- menopausal age in smokers vs. nonsmokers in Australia, 82–1727
- Mormon, church involvement and cancer incidence relationship, 82–1612
- New Zealand Maoris, smoking and drinking effects on pregnancy outcome, 82–1726
- office air quality, health effects, 82–1736
- oral contraceptive use health risks, smokers vs. nonsmokers, 82–1598
- platelet monoamine oxidase and plasma thiocyanate concentrations, correlation in smokers vs. nonsmokers, 82–1581
- sister-chromatid exchange incidence in lymphocytes of breast cancer patients, smoking association, 82–1621
- smoking habit in Calvados, France, 82–1782

YUGOSLAVIA
- alcohol consumption and smoking habits in a cardiovascular disease study, 82–1767
- "smoker's cancer" incidence, 1969-1975, 82–1626

ZIMBABWE
- flue-cured and Burley tobacco production trends, 82–1847

☆ U.S. GOVERNMENT PRINTING OFFICE: 1982-381-166:454

SMOKING AND HEALTH LITERATURE SEARCH REQUEST

Individual requesting search

Title or position

Individual other than requester completing form

Telephone number

Organization

Date of request

Address including zip code

Purpose of search

☐ Research

☐ Paper presentation

☐ Writing article ☐ Lecture

☐ Other ____________

SEARCH REQUIREMENTS Give below a detailed statement of information requirements together with any necessary background data to meet your needs. Be as specific as possible to help formulate search strategy.

If topic has been reviewed or cited in past issues of the *Smoking and Health Bulletin* or *Bibliography on Smoking and Health* give pertinent accession number(s) or citation of articles.

Give below other articles which may serve as a guide on subject area (list full bibliographic citation if known)

List relevant key words or index terms appropriate to search.

If a search has been performed on subject utilizing other data sources, please list sources, results of search and search strategy if known.

SEARCH RESTRICTIONS Check appropriate search out-put desired.

☐ Full range (no restrictions)

☐ Year(s) ______________ only

☐ Citation only (no abstract)

☐ English language only

☐ Other language ______________

____

AGENCY USE ONLY. Do Not Write Below This Line.

Search statement or criteria	Date search received Date search released
	Number of documents retrieved

NOTICE

If you no longer need this publication, check here____, return this sheet to the address below, and your name will be dropped from the mailing list.

If you have a change in name or address, please write the change on this sheet and return it to the address below.

WHEN REQUESTING A CHANGE PLEASE BE SURE YOUR FORMER LISTING ALSO APPEARS ON THIS SHEET.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health
Rockville, Maryland 20857

U.S. MAIL

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396

SPECIAL FOURTH CLASS
BOOK